

Anaesthetic Management of a Patient of Methemoglobinemia for Emergency Lower Segment Caserian Section

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Abstract: Pregnancy with methemoglobinemia (metHb) poses several challenges to the anaesthesiologist. MetHb is characterized by iron moiety of Hb in the ferric (Fe³⁺) state. MetHb has decreased oxygen carrying capacity and increased oxygen affinity resulting in cyanosis and tissue hypoxia. Clinical features are proportional to the concentration of the MetHb, which range from asymptomatic state to seizures and cardiac arrhythmia to fatality. We present a case of primigravida with Methemoglobinemia posted for emergency cesarean section. At presentation she had cyanosis, dyspnoea and pain in abdomen. Her methemoglobin levels were 25%. She was treated with Methylene blue before induction. This article addresses the anaesthetic challenges and precautions to be taken for a successful anaesthetic management of a patient with methemoglobinemia.

Keywords: methemoglobinemia (metHb), general anaesthesia, primigravida.

I. Introduction

Methemoglobinemia (or methaemoglobinaemia) is a disorder characterized by the presence of a higher than normal level of methemoglobin (metHb, i.e., ferric [Fe³⁺] rather than ferrous [Fe²⁺] haemoglobin) in the blood. Methemoglobin is a form of hemoglobin that contains ferric [Fe³⁺] iron and has a decreased ability to bind oxygen. However, the ferric iron has an increased affinity for bound oxygen.[1] The binding of oxygen to methemoglobin results in an increased affinity of oxygen to the three other heme sites (that are still ferrous) within the same tetrameric hemoglobin unit. This leads to an overall reduced ability of the red blood cell to release oxygen to tissues, with the associated oxygen-hemoglobin dissociation curve therefore shifted to the left. When methemoglobin concentration is elevated in red blood cells, tissue hypoxia can occur.

II. Case Report

A 23 year old primigravida with height 150 cm, weighing 51 kg with congenital methHb with pregnancy induced hypertension (PIH) married since one and half years, 36 weeks of gestation was posted for emergency cesarean section in view of abruption of placenta and fetal distress. She was a referred case with complaints of cyanosis, dyspnoea and pain in abdomen. Her past history revealed she was diagnosed with metHb when she was 19 years old. She presented with history of cyanosis, dyspnoea and pain in abdomen and was referred to a cardiologist. Her saturation was 85% on pulse oximetry on oxygen. Rest of the examination, general physical and systemic examination and all basic investigations were normal. Cardiologist suspected metHb and was referred to our hospital for further evaluation and management.

At initial presentation her metHb was at 44% (normal levels <1%) Further evaluation revealed reduced NADH cytochrome b5 reductase levels of 15.87 IU/g Hb (normal levels 35.0±5.00 IU/g Hb) Her hemoglobin molecule and G6PD activity were normal. She was started on vitamin C medications. She had two or three episodes of the same symptoms since her diagnosis for which she had taken treatment (details not known).

At 36 weeks of gestation she presented to emergency with cyanosis, dyspnoea and pain in abdomen. She was posted for emergency cesarean section in view of abruption of placenta and fetal distress. She had PIH for which she was on Methyl dopa 200 mg QID and Nifedipine 5 mg QID. Her methemoglobin levels at 35 weeks of gestation were 25%. She had no other significant medical or surgical history.

1. Obstetric History: Patient is G1P1 with G1 being Present pregnancy

2. On Examination: Patient was conscious and oriented to time place and person.

PR-72/min, BP-125/75 mm of Hg,

She had pallor. Pulse oximetry was showing saturation of 94% on air and 98 on oxygen.

ECG was normal.

3.Systemic Examination:

3.1 Cardiovascular system: S1S2 Heard, no any murmur,

3.2 Respiratory System: Air entry bilaterally equal and clear,

3.3 Spine Examination: No any obvious deformity.

4.Airway Examination :

Mouth opening : >3 fingers,
MPC II,
neck movements :Adequate,
Thyro-mental distance :~6 cm.

5.Investigations

Laboratory tests revealed

Hb-13.9 gm%,

TLC-8400/ mm³,

Platelet-3 lacs/mm³,

Creatinine-0.7mg%,

SGOT/SGPT-19/5 IU,

PT/INR-9.6/0.97.

Arterial blood gases revealed

pH-7.32,

PCO₂-23,

PO₂-482,

HCO₃-11.9,

SpO₂-100% on oxygen through Hudson's mask.

III. Anaesthetic Management

A written informed consent for anaesthesia was taken including explanation regarding general anaesthesia. General anaesthesia was planned in view of fetal distress. Patient was taken inside operation theatre and monitors ECG, pulse –oximetry, NIBP attached. Then 18 G venous access was secured on both the upper limbs. The blood from inserted IV cannula collected for VBG sampling which appeared brown in colour. Inj. Ondansetron 0.1mg/kg given i.v. along with Inj. Ranitidine 50mg i.v. slowly. Fetal heart rate on operation table was 100/min. Co-loading with crystalloids (Ringers Lactate) started along with preparation for rapid sequence induction for general anaesthesia. Patient was given Methylene blue at 1 mg/kg body weight i.e 50 mg in 500 ml normal saline before induction (after consulting with the haematologist). Supine position with left lateral tilt was given. Rapid sequence induction was done with Inj. Thiopentone 250mg and Inj. succinylcholine 75mg and intubation done with 6.5 cuffed endotracheal tube and anaesthesia was maintained with Oxygen and Air with Sevoflurane. Caesarean section was performed with an incision over lower uterine segment. Her peripheral cyanosis which was prominent at her finger tips disappeared and pulse oximeter reading increased from 92% to 97%, 20 minutes into the caesarean section.

A male child which cried immediately after birth with birth weight 2.63 kg and APGAR scores of 9 and 10 at 5 and 10 minutes respectively were noted. Baby had no pallor or cyanosis and other general and systemic examinations were normal.

Inj. Pitocin (20U in 500ml of Ringer Lactate) was started immediately after the delivery of the baby. Patient remained hemodynamically stable throughout the procedure. A long acting muscle relaxant viz. Atracurium 25mg was given to the patient after the action of succinylcholine wore off.

Blood loss: 700 ml.

Urine output: 250ml.

I.V. Fluids: 3 pint RL (1500ml).

Post-operatively patient's neuromuscular blockade was reversed with inj. Neostigmine 2.5mg and inj. Glycopyrrolate 0.4mg and patient was extubated.

Patient was conscious, oriented and vitally stable.

Postoperatively patients methemoglobin levels were found to be reduced to 7% with no peripheral cyanosis and pulse oximeter reading of 95% on air.

Baby's methemoglobin levels were 2%.

IV. Discussion

Methemoglobinemia (congenital or acquired) occurs when red blood cells (RBCs) contain methemoglobin at levels higher than 1%. Methemoglobin results from the presence of iron in the ferric form instead of the usual ferrous form. This results in a decreased availability of oxygen to the tissues. Symptoms are proportional to the methemoglobin level. Skin color changes and blood color changes appear as the MethHb levels increase to 15% . Neurologic and Cardiac symptoms begin to appear as a consequence of hypoxia when the level cross 15% . Levels higher than 70% are usually fatal.(1)

Methemoglobinemia may result from congenital deficiencies of enzymes that normally convert methemoglobin to hemoglobin like the enzyme diaphorase(NADH methemoglobin reductase) and pyruvate kinase, alterations in the hemoglobin molecule itself or, most commonly, from the ingestion of medications or toxins that oxidize the ferrous iron of hemoglobin. Classical drug causes of methemoglobinaemia include antibiotics like trimethoprim, sulfonamides and dapsone, local anaesthetics like prilocaine and benzocaine and aniline dyes, metoclopramide, chlorates and bromates. Ingestion of compounds containing nitrates can also cause methemoglobinemia. (2,3)

Several issues must be considered when anesthetizing patients with methemoglobinemia, including the potential for decreased O₂ delivery, which may be exacerbated by intraoperative blood loss and anemia, interference with normal intraoperative monitoring devices especially the pulse oximeter and the potential for medications to cause or exacerbate methemoglobinemia.(4) Prompt recognition of the condition and initiation of treatment, as indicated (especially in acquired methemoglobinemia), are critical in the management of methemoglobinemia. However, at the same time, it may be essential to carry out extensive investigations to rule out cardiac and pulmonary causes as these also present with cyanosis. Methylene blue is the primary emergency treatment for documented symptomatic methemoglobinemia. It is given in a dose of 1-2 mg/kg (up to a total of 50 mg in adults, adolescents, and older children) as a 1% solution in IV saline over 3-5 minutes. Ascorbic acid and riboflavin can also be used. Hyperbaric oxygen therapy and exchange transfusion therapy can be used if methylene blue fails to act.(5)

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