International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Katyal M. Int J Reprod Contracept Obstet Gynecol. 2020 Jun;9(6):2602-2604
www.ijrcog.org

pISSN 2320-1770 | eISSN 2320-1789

DOI: http://dx.doi.org/10.18203/2320-1770.ijrcog20202356

Case Report

Anaesthetic management of a young primigravida with 8 months amenorrhea, rheumatic heart disease, hypothyroidism and met-hemoglobinimea schedule for cesarean section

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Received: 16 March 2020
Accepted: 22 April 2020

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ABSTRACT

A young patient, primigravida with rheumatic heart disease, hypothyroidism and met-hemoglobinemia had central and peripheral cyanosis was scheduled for caesarean section under general anesthesia in two days. Author managed met-hemoglobinemia with ascorbic acid keeping methylene blue as standby. Haemoglobin saturation was 89% at room air and 92% with supplemental oxygen. Initial met-haemoglobin levels were 31.54% (normal values <1%). After optimizing thyroid and RHD status (moderate MR and mild MS), she was given tablet ascorbic acid 2 gram thrice a day with methylene blue as standby. Conventional balanced general anesthesia technique was used and she was followed up in postoperative period with ICU care. A healthy live female baby was extracted and mother had stable vital parameters. Postoperative treatment with ascorbic acid was continued. Repeated methaemoglobin levels showed a drop from preoperative value of 31.54% to 11.39% by 3rd postoperative day and 8.05% at the end of 1st week. At the time of discharge, she did not have any cyanosis. Ascorbic acid is a good alternative drug with limited experience in met-hemoglobinemia. Author present a case of a met-hemoglobinemia treated with ascorbic acid successfully to emphasize the use of ascorbic acid as an alternative method without any adverse effects.

Keywords: Ascorbic acid, Cesarean section, Met-hemoglobinemia

INTRODUCTION

Met-hemoglobinemia refers to the oxidation of ferrous iron (Fe++) to ferric iron (Fe+++) within the haemoglobin molecule.[1] It reduces the oxygen carrying capacity of blood by two mechanisms. First, it is unable to carry oxygen molecules. Second, the presence of meth-haemoglobin shifts the oxygen dissociation curve to the left. This shift increases the affinity of remaining haemoglobin molecules to oxygen.[2] Meth-haemoglobin may arise from a variety of etiologies including genetic, dietary, idiopathic, and toxicologic sources. Normal level of meth-haemoglobin in blood is <1% of total haemoglobin.[1] It presents as generalized cyanosis. Symptoms depending on the level of meth-haemoglobin in blood vary from anxiety, headache, confusion, seizures, coma and death at level > 70%.

When patients have significantly elevated methaemoglobin levels (>20%), the pulse oximeter falsely indicates high levels of oxygen saturation. Arterial blood gas analysis may also be initially deceptive, because the partial pressure of O_2, as a measure of dissolved oxygen, is normal.

Thus, extrapolation of this figure to predict the expected oxygen saturation will provide a falsely elevated result. The best definitive diagnostic test is multiple wavelength co-oximetry, an in vitro spectrophotometric method that is capable of differentiating between oxy-, deoxy-, met- and carboxyhaemoglobin.[3] The treatment of choice for severe meth-hemoglobinemia is methylene blue. Methylene blue is provided as a 1% solution (10 mg/mL). The dose is 1 to 2 mg/kg (0.2 mL/kg of a 1% solution) infused intravenously over 3 to 5 minutes.[4] The dose may be

repeated at 1 mg/kg if MHb does not resolve within 30 minutes.

CASE REPORT

A 23 years old primigravida with known history of rheumatic heart disease and hypothyroidism (on treatment) and 8 months amenorrhea presented to the preanesthetic clinic for elective LSCS after two days. On examination both peripheral and central cyanosis (Figure 1) was noticed without dyspnoea. Room air oxygen saturation was 89% which increased to 92% only with 100% oxygen. We suspected methemoglobinemia and advised co-oximetry test. Methemoglobin level came to be 31.54% (normal <1%). TSH was 4.34 microIU on 75 mcg thyroxine. She had moderate MR with mild MS. Biochemical investigations were within normal range (Table 1). Intra cardiac shunts were ruled out by pulmonary angiography and 2D-ECHO.

Since, author did not want to expose the pregnant patient and the fetus to IV methylene blue for the risks of anaphylaxis we prescribed her tablet vitamin C 2 gms 8th hourly and kept methylene blue as standby. Also, this case review did not elicit any literature wherein methylene blue was used in a pregnant patient.

Table 1: Biochemical investigations.

Lab variables	Results
Haemoglobin	10.9 gms/dl
Leucocyte count	6500 cells/µl
Platelet count	2 lakh/µl
pCO_2	36 mmHg
pO_2	140 mmHg
HCO_3	22 mEq/l
pH	7.41

Infective endocarditis prophylaxis was given half hour before surgery. The anesthetic technique used was general anesthesia with controlled ventilation. Patient was pre-oxygenated with 100% oxygen for 3 minutes. Pre-medication included inj Glycopyrrolate 0.2 mg + inj midazolam 1 mg IV patient was induced with inj. fentanyl 125 mcg IV + 6% Sevoflurane and intubation facilitated with inj. vecuronium 4 mg using 7.5 mm cuffed endotracheal tube. Bilateral air entry was equal and cuff inflated. Maintenance was using oxygen and Sevoflurane with controlled mode ventilation. Peri-operative routine monitoring was done. Vital parameters throughout the procedure were stable. A healthy live female baby was extracted following which patient was given inj. Oxytocin 10 IU diluted in 500 ml normal saline as infusion. Uterus contracted well and the extubation was uneventful following adequate reversal. Post-operative treatment with ascorbic acid was continued and the patient was observed in intensive care unit for a day. Repeated methemoglobin levels showed a drop from pre-operative value of 31.54% to 11.39% by 3rd post-operative day and 8.05% at the end of 1st week. At the time of discharge, she did not have any cyanosis (Figure 2).

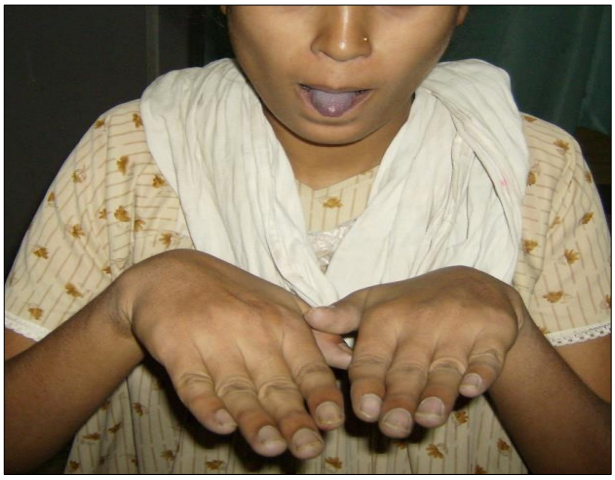

Figure 1: Central and peripheral cyanosis.

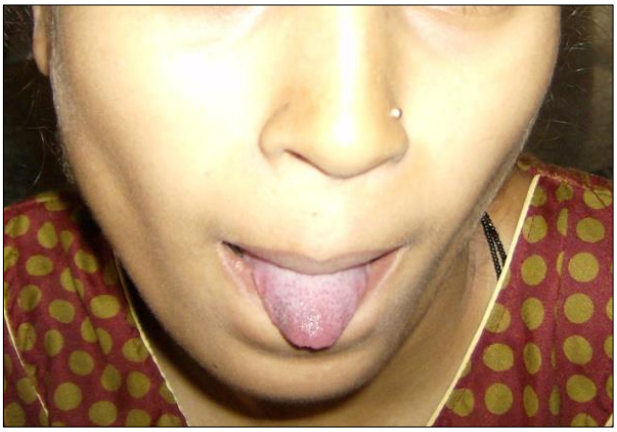

Figure 2: Cyanosis improved after vitamin C.

DISCUSSION

Met-hemoglobinemia was considered in our patient as she had cyanosis with oxygen saturation 89% at room air which did not increase more than 92% with 100% oxygen. Also, 2D-ECHO and pulmonary angiography showed no intra cardiac shunts. Her baseline blood met-hemoglobin levels as tested by co-oximetry were 31.54%. There are reports of ascorbic acid used in treatment of met-hemoglobinemia.[5] Author used ascorbic acid keeping methylene blue as stand by as author did not want to expose the fetus and the mother to the drug because of possible adverse effect of the drug (anaphylaxis) and we could not elicit any literature wherein a pregnant woman has been given methylene blue. Also, she showed improvement in cyanosis and oxygen saturation after the first dose of ascorbic acid. Vitamin C scavenges free radicals and protects cells from oxidative damage. Recycling of α-tocopherol by ascorbate has been demonstrated in cellular organelles and erythrocyte membranes. It also acts as a co-factor for NADP reductase

required for glutathione metabolism. Furthermore, it can directly reduce met-haemoglobin.[6]

CONCLUSION

Methemoglobinemia although rare should be considered in all cyanotic patients who remain unresponsive to oxygen therapy. Met-hemoglobinemia patients with asymptomatic cyanosis can be treated effectively with oral ascorbic acid instead of methylene blue without any adverse effects.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Katyal M. Anaesthetic management of a young primigravida with 8 months amenorrhea, rheumatic heart disease, hypothyroidism and met-hemoglobinimea schedule for cesarean section. Int J Reprod Contracept Obstet Gynecol 2020;9:2602-4.