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# Anaesthetic Management of a Pregnant Patient with Methaemoglobinaemia Undergoing Emergency Caesarean Section

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**Abstract**

Methaemoglobinaemia is a rare disorder where haemoglobin is oxidized to methaemoglobin, which cannot carry oxygen efficiently. In pregnancy, unrecognized methaemoglobinaemia may pose risks to both mother and foetus. Congenital methaemoglobinaemia poses unique anaesthetic challenges, particularly in pregnant patients undergoing surgical interventions. We present the successful anaesthetic management of a young female at 32 weeks gestation who was diagnosed intraoperatively with congenital methaemoglobinaemia during an emergency lower segment caesarean section for preeclampsia with placental abruption.

**Keywords:** Methaemoglobinaemia; Regional anaesthesia; Spinal anaesthesia; Oxygen desaturation; Co-oximetry; Maternal-foetal outcomes.

## Introduction

Methaemoglobinaemia is a rare haematological condition characterized by the presence of methaemoglobin (MetHb), an oxidized form of haemoglobin in which iron exists in the ferric ($Fe^{3+}$) state rather than the normal ferrous ($Fe^{2+}$) state. This altered form of haemoglobin is unable to effectively bind and transport oxygen, leading to impaired oxygen delivery despite normal arterial oxygen tension and haemoglobin concentration. Clinically, patients may present with cyanosis that does not improve with oxygen supplementation, and in severe cases, symptoms of tissue hypoxia such as headache, fatigue, dyspnoea, and altered mental status may occur [1,2].

Methaemoglobinaemia may be acquired, most commonly due to exposure to oxidizing agents such as nitrates, local anaesthetics (e.g., benzocaine, prilocaine), sulfonamides, or certain antibiotics. Alternatively, it may be congenital, resulting from inherited defects in enzymes responsible for reducing methaemoglobin to haemoglobin, most notably cytochrome b5 reductase (also known as NADH-dependent methaemoglobin reductase) [3,4]. Congenital methaemoglobinaemia is further

classified into Type I (limited to erythrocytes) and Type II (generalized, with neurological involvement), with the former typically presenting as lifelong cyanosis without systemic symptoms [5].

During pregnancy, physiological changes in plasma volume, oxygen consumption, and respiratory function increase maternal and foetal vulnerability to hypoxia. Additionally, foetal haemoglobin is more susceptible to oxidative stress, although the foetal NADH-dependent reductase system is generally sufficient to maintain MetHb at safe levels unless a congenital enzyme deficiency is present [6,7]. In such cases, both maternal and foetal outcomes may be jeopardized if the condition goes unrecognized.

Anaesthetic management of patients with methaemoglobinaemia presents specific challenges. Pulse oximetry becomes unreliable due to the unique light absorption spectrum of MetHb, typically resulting in $SpO_2$ readings around 85% regardless of the actual oxygenation status. Arterial blood gas (ABG) analysis with co-oximetry is required for accurate measurement of MetHb and oxygenation parameters [8]. Avoidance of oxidative stress, careful drug selection, and appropriate oxygen delivery strategies are vital. Regional anaesthesia, when feasible, is often preferred over general anaesthesia in stable patients to minimize oxidative drug exposure and airway manipulation.

This report presents the anaesthetic management of a parturient with undiagnosed congenital methaemoglobinemia who underwent emergency lower segment caesarean section (LSCS) under spinal anaesthesia. The case highlights the importance of clinical vigilance, timely diagnosis, and safe anaesthetic planning in patients with this rare condition.

## Case Description

A young female, at 32 weeks of gestation, presented with complaints of mild vaginal bleeding and abdominal pain of one-day duration. She had no significant past medical history and was only on routine antenatal iron and calcium supplements. Antenatal course until then was uneventful. Her previous pregnancy was uneventful.

On evaluation in the labour room, her BP was elevated (160/100 mmHg), and oxygen saturation ($SpO_2$) was noted to be 88% on room air. Notably, she showed no clinical signs of respiratory distress. She was diagnosed with preeclampsia with suspected placental abruption and was posted for emergency LSCS.

She received a loading dose of 4 grams of magnesium sulphate intravenously and was shifted to the Operating Room with oxygen via face mask at 6 litres per minute. On arrival, her vitals were stable: HR 88 bpm, RR 20/min, BP 140/90 mmHg, and $SpO_2$ 91% on 6 L/min oxygen. Clinical examination revealed peripheral cyanosis involving fingers and toes, with warm peripheries and bilateral pedal oedema. Chest auscultation revealed normal vesicular breath sounds, with no added sounds. Administration of 100% oxygen marginally improved $SpO_2$ to 93%. Foetal Heart Rate was 140 beats per minute.

Upon further history taking, a sibling was reported to have similar finger discoloration, raising suspicion of congenital methaemoglobinaemia.

## Anaesthetic Management

In this patient, multiple factors influenced the choice of anaesthetic technique, including her pregnancy, the urgency of the Caesarean section, and the suspicion of methaemoglobinaemia based on unexplained cyanosis and low pulse oximetry readings unresponsive to supplemental oxygen.

**Preoperative Assessment and Planning**

On arrival in the operating room, the patient's vital signs were stable, but her peripheral cyanosis and $SpO_2$ of 91% on supplemental oxygen were concerning. There were no signs of respiratory distress, and auscultation of the lungs was normal, which made intrinsic pulmonary pathology less likely. The differential diagnosis at this stage included methaemoglobinaemia and dyshaemoglobinaemias, with further suspicion supported by a family history of similar bluish discoloration of the fingers in a sibling.

Given the non-progressive desaturation and preserved oxygenation on ABG, the team prioritized a regional technique over general anaesthesia to avoid the use of oxidizing agents and to minimize foetal drug exposure. A high-risk consent was obtained, clearly explaining the possibility of rare congenital blood disorders, risk of intra-operative blood loss and risk of further worsening with administration of anaesthetic agents.

**Intraoperative Course**

The patient was coloaded with 500 mL of Ringer's lactate to reduce the risk of post-spinal hypotension. Under strict asepsis, spinal anaesthesia was administered at the L3-L4 interspace using 2.2 mL of 0.5% hyperbaric bupivacaine without adjuvants. Of note, the blood withdrawn during needle placement appeared chocolate-brown, which was a critical visual clue supporting the diagnosis of methaemoglobinaemia. Sensory block to T4 was achieved within 5 minutes. The patient remained hemodynamically stable throughout the surgery, with a mean arterial pressure maintained above 70 mmHg. Prophylactic phenylephrine boluses (50-100 mcg IV) were administered intermittently to counteract mild hypotension.

$SpO_2$ levels remained between 91-93% throughout the procedure despite administration of 100% oxygen via facemask. Capnography and electrocardiography remained normal. Estimated blood loss was approximately 800 mL, and urine output was adequate. No sedatives, opioids, or vasodilators were administered intraoperatively to avoid exacerbation of methaemoglobinaemia or foetal depression.

A male neonate weighing 1.4 kg was delivered with Apgar scores of 8 and 9 at 1 and 5 minutes, respectively. No resuscitation was required, and the baby was transferred to the NICU for preterm care. No signs of neonatal cyanosis were noted.

**Postoperative Management**

Postoperatively, the patient was monitored in the intensive care unit. Pain management was achieved with intravenous paracetamol (1g every 6 hours). She remained hemodynamically stable with no evidence of neurologic deficits, worsening cyanosis, or lactic acidosis.

A confirmatory arterial blood gas analysis with co-oximetry revealed a methaemoglobin level of 34%, consistent with moderate methaemoglobinaemia. As the patient remained asymptomatic with no hypoxia or organ dysfunction, conservative management was chosen. She was started on oral vitamin C (ascorbic acid 500 mg twice daily) and riboflavin supplementation to promote endogenous reduction of methaemoglobin.

Genetic counselling and testing were advised for confirmation of congenital cytochrome b5 reductase deficiency, the most common cause of hereditary methaemoglobinaemia.

Follow-up at two weeks showed a decreasing trend in methaemoglobin levels upto 13% and

continued clinical stability.

## Discussion

Methaemoglobinaemia is a rare but potentially serious condition resulting from the oxidation of haemoglobin's iron molecule from $Fe^{2+}$ to $Fe^{3+}$, forming methaemoglobin (MetHb), which cannot bind oxygen. This impairs oxygen delivery despite normal arterial oxygen tension, resulting in a functional anaemia. Clinically, it presents with cyanosis, low $SpO_2$ on pulse oximetry, and a lack of response to oxygen therapy. When severe, it can lead to hypoxic symptoms such as headache, fatigue, dyspnoea, arrhythmias, and seizures [1,9].

The diagnosis is often delayed because standard pulse oximeters cannot accurately detect methaemoglobin. They typically show saturation readings around 85%, regardless of the true oxygenation status. In our patient, cyanosis was limited to extremities, and she was otherwise asymptomatic with no signs of respiratory distress, suggesting a functional, rather than hypoxic, cause of desaturation [10]. Definitive diagnosis requires co-oximetry, which uses multiple light wavelengths to directly measure methaemoglobin levels. This was performed postoperatively in our case, confirming the diagnosis [11].

Anaesthetic implications are significant. Many common anaesthetic agents, such as benzocaine, prilocaine, nitrites, and nitroprusside, can induce or worsen methaemoglobinaemia [9]. General anaesthesia involves the risk of such drug exposure and may also impair oxygenation due to airway manipulation and sedative effects. In contrast, regional anaesthesia minimizes these risks and preserves patient consciousness for early detection of symptoms [12].

Our patient underwent spinal anaesthesia with bupivacaine, which is considered safe. Spinal anaesthesia preserves spontaneous ventilation and maintains stable cardiovascular status. It gives rapid onset and adequate anaesthesia suitable for emergency Caesarean section. It allowed us to avoid polypharmacy. No sedatives or other oxidizing agents were administered. Intraoperative monitoring and oxygen supplementation ensured maternal and foetal stability throughout the procedure.

Treatment of methaemoglobinaemia depends on severity. In mild or asymptomatic cases (MetHb <20%), conservative management is often sufficient. In symptomatic or severe cases, methylene blue is the treatment of choice, acting as an electron donor to reduce MetHb back to functional haemoglobin via the NADPH-methaemoglobin reductase pathway. However, it is contraindicated in patients with G6PD deficiency [13,14].

In pregnancy, foetal MetHb levels should also be considered, though foetal reductase systems are usually sufficient unless there is a congenital enzyme deficiency. Our patient's neonate was clinically stable at birth and did not require any intervention.

This case reinforces the importance of clinical suspicion in the presence of low $SpO_2$ with normal respiratory examination and highlights the role of regional anaesthesia in managing such cases safely. It also emphasizes the importance of postoperative monitoring and appropriate use of diagnostic tools like co-oximetry to confirm the diagnosis.

## Conclusion

Methaemoglobinaemia, though rare, should be considered in pregnant patients presenting with unexplained low oxygen saturation, especially when accompanied by peripheral cyanosis and minimal respiratory symptoms. Early recognition, avoidance of oxidizing agents, and safe use of regional anaesthesia are key to successful management. Co-oximetry is essential for diagnosis, and most mild cases can be managed conservatively. With appropriate perioperative planning, excellent

maternal and foetal outcomes can be achieved.

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