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The Saturation Gap Puzzle: A Diagnostic Challenge in the Emergency Department

Dr. Simran Kumar[1], Dr. Pooja Verma[2]

PGY-3, Emergency Medicine (Corresponding Author)

Assistant Professor, Emergency Medicine

Abstract: ***Background**: Methemoglobinemia is an uncommon disorder that can significantly affect the interpretation of oxygenation parameters. The coexistence of additional causes of hypoxemia may further complicate clinical assessment and decision-making. Recognition of discrepancies between pulse oximetry, arterial blood gas analysis, and the patient's clinical status is essential for accurate diagnosis. **Case Presentation**: A 28-year-old woman presented to the emergency department with headache and severe hypertension. Physical examination revealed central cyanosis with unexpectedly low pulse oximetry readings. Arterial blood gas analysis demonstrated a marked discrepancy between oxygen saturation measurements and the patient's relatively preserved clinical condition. Co-oximetry identified methemoglobinemia, while concurrent chronic kidney disease with pulmonary congestion contributed additional hypoxemic burden. Despite profoundly abnormal oxygenation parameters, the patient showed no evidence of tissue hypoxia. **Conclusion**: Methemoglobinemia can produce significant discrepancies between pulse oximetry and arterial oxygen saturation measurements, particularly when additional causes of hypoxia coexist. Clinical assessment, co-oximetry, and evaluation for tissue hypoxia are essential for accurate interpretation of oxygenation status. Clinicians should exercise caution when relying solely on pulse oximetry in such patients.*

Keywords: Methemoglobinemia; Saturation Gap; Pulse Oximetry; Co-oximetry; Hypoxemia; Cyanosis; Chronic Kidney Disease; Emergency Medicine; Tissue Hypoxia; Diagnostic Challenge

1. Introduction

Methemoglobinemia occurs when the ferrous iron (Fe^{2+}) within hemoglobin is oxidized to the ferric (Fe^{3+}) state, resulting in reduced oxygen-carrying capacity and impaired oxygen delivery to tissues. Both congenital and acquired forms have been described. Pulse oximetry may provide misleading oxygen saturation values in affected patients, and the interpretation of oxygenation becomes even more challenging when other causes of hypoxemia coexist. We present a case of presumed congenital methemoglobinemia complicated by chronic kidney disease and pulmonary congestion, resulting in striking discordance between pulse oximetry, arterial blood gas measurements, and the patient's clinical status.

2. Case Presentation

A 28-year-old woman presented to the emergency department with a three-day history of gradual-onset frontal headache. Her blood pressure was 233/144 mmHg and pulse rate was 95 beats/minute. Cardiovascular and neurological examinations were unremarkable. General examination revealed pallor and central cyanosis (Figure 1). She was mildly tachypneic (respiratory rate 23/minute) but exhibited no overt signs of respiratory distress, and chest auscultation was normal.

Bedside pulse oximetry demonstrated an SpO_2 of 28–30%, which increased to 68–70% with supplemental oxygen via face mask. Point-of-care ultrasound showed occasional bilateral basal B-lines, while echocardiography revealed Grade II diastolic dysfunction without evidence of congenital heart disease. Arterial blood gas analysis on room air demonstrated an SaO_2 of 71%, PaO_2 of 55 mmHg, pH 7.33, lactate 0.5 mmol/L, $PaCO_2$ 38 mmHg, and bicarbonate 19.6 mmol/L. Co-oximetry revealed 11.4% methemoglobin.

Further history revealed previously documented low oxygen saturation readings and a maternal history of similar findings, suggesting a congenital etiology. Computed tomography of the head was normal. Laboratory investigations demonstrated hemoglobin 7.0 g/dL, serum urea 170 mg/dL, and serum creatinine 11.1 mg/dL, consistent with advanced chronic kidney disease. A provisional diagnosis of congenital methemoglobinemia with non-oliguric chronic kidney disease was made. The patient was treated with oxygen therapy, antihypertensives, and diuretics and was advised hospital admission. However, she elected to leave against medical advice.

3. Discussion

Pulse oximetry estimates oxygen saturation using differential light absorption at 660 nm and 940 nm wavelengths. Methemoglobin absorbs light at both wavelengths, producing inaccurate saturation readings and creating a discrepancy between measured SpO_2 and actual oxygenation status. Conversely, arterial oxygen saturation derived from standard blood gas analysis may also be misleading because conventional calculations assume the presence of only oxyhemoglobin and deoxyhemoglobin.

The difference between pulse oximetry-derived saturation and arterial oxygen saturation is termed the 'saturation gap'. A

saturation gap greater than 5% should prompt consideration of dyshemoglobinemias and warrants confirmation using co-oximetry. In our patient, the saturation gap exceeded 50%, highlighting the diagnostic value of this phenomenon.

Although the methemoglobin level was only 11.4%, sufficient to explain the observed cyanosis and headache, it could not fully account for the severity of the hypoxemia demonstrated on arterial blood gas analysis. The coexistence of chronic kidney disease, anemia, and probable pulmonary congestion likely contributed to impaired oxygenation. Notably, the patient remained clinically stable and demonstrated no biochemical evidence of tissue hypoxia, as reflected by a normal serum lactate level.

Methemoglobinemia is a well-recognized cause of oxygen saturation discrepancy owing to its unique optical properties and its effect on pulse oximetry measurements. The presence of concomitant pulmonary edema and chronic kidney disease in our patient created a complex clinical scenario in which neither pulse oximetry nor arterial blood gas values alone accurately reflected the patient's physiological status. Despite markedly abnormal oxygenation parameters, the absence of elevated lactate levels or clinical evidence of end-organ hypoxia suggested preserved tissue oxygen delivery. This case highlights the importance of integrating clinical examination, co-oximetry findings, and markers of tissue perfusion when evaluating patients with unexplained cyanosis and discordant oxygenation measurements.

4. Conclusion

Methemoglobinemia can produce significant discrepancies between pulse oximetry and arterial oxygen saturation measurements, particularly when additional causes of hypoxia coexist. Clinical assessment, co-oximetry, and evaluation for tissue hypoxia are essential for accurate interpretation of oxygenation status. Clinicians should exercise caution when relying solely on pulse oximetry in such patients.

Figure

Figure 1: Central cyanosis observed at presentation