Methemoglobinemia in an Elderly Patient with Multiple Comorbidities: A Diagnostic and Management Challenge

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Case Report

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ABSTRACT:

Methemoglobinemia, a rare yet serious disorder impairing oxygen transport, can lead to significant hypoxia and requires urgent intervention. This case report presents an elderly patient with multiple comorbidities, including diabetes, hypertension, ischemic heart disease, end-stage renal disease (ESRD), and hypothyroidism who developed acquired methemoglobinemia. Discrepancies between arterial blood gas (ABG) and pulse oximetry, alongside dark brown arterial blood, led to the diagnosis of methemoglobinemia. Treatment involved ascorbic acid and exchange transfusion, tailored due unavailability of methylene blue in Bangladesh. The patient's condition was further complicated by diabetic ketoacidosis and septic shock that led to his demise. This case underscores the diagnostic challenges and the need for a multidisciplinary approach in managing methemoglobinemia in patients with complex comorbidities. It emphasizes the importance of clinical vigilance, appropriate diagnostic tools, and tailored treatment strategies to improve patient outcomes.

Key words: Methemoglobinemia, Oxygen Transport, End-Stage Renal Disease (ESRD), Ascorbic Acid, Exchange Transfusion.

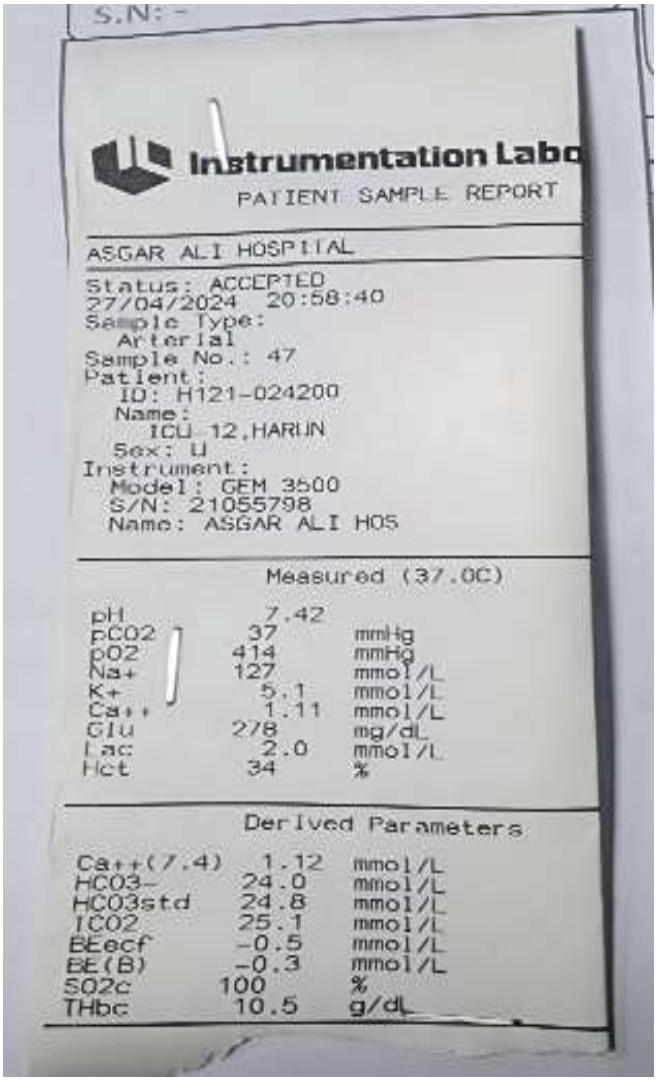
INTRODUCTION:

Methemoglobinemia, while uncommon, presents with a significant clinical challenge, particularly in patients with complex medical histories. It is a rare blood disorder. This blood disorder, characterized by elevated methemoglobin levels, compromises oxygen transport and can lead to severe hypoxia.[1] This condition can be congenital or acquired, with the later often resulting from exposure to certain drugs or chemicals.[2] While uncommon, its potential for severe complications necessitates swift recognition and treatment. We present a case of acquired methemoglobinemia in an elderly patient with multiple comorbidities, highlighting the importance of clinical suspicion, prompt diagnosis, and multidisciplinary management.

CASE REPORT:

A 71-year-old man with a history of diabetes mellitus, hypertension, ischemic heart disease, end-stage renal disease on maintenance haemodialysis, and hypothyroidism, was admitted in a Tertiary Care Hospital, Dhaka on April 27, 2024. The patient presented with acute onset shortness of breath for 30 minutes following routine haemodialysis. On presentation, the patient was conscious but severely hypoxic, with an SpO_2 of 60% despite high-flow oxygen. Initial management included escalating respiratory support from high flow nasal cannula to non-invasive ventilation (NIV). Due to persistent hypoxemia, the patient was intubated and started on vasopressors to maintain blood pressure. Initial investigations revealed normal cardiac markers and D-Dimer levels. Echocardiography showed no significant abnormalities. A striking discrepancy was noted between the arterial blood gas (ABG), oxygen saturation (SaO_2 100%, PaO_2 316mmHg) and the pulse oximetry reading (SpO_2 73%) despite 100% FiO_2 (Fig-1). The arterial blood appeared unusually dark brown despite high PaO2 (Fig-2) raising suspicion for methemoglobinemia.

Diagnosis was confirmed based on clinical presentation and laboratory findings. Treatment included ascorbic acid administration and two sessions of small-volume exchange transfusion (Fig-3). Due to unavailability in Bangladesh, Methylene blue was not in treatment option. A multidisciplinary team managed the patient's complex clinical course. The patient showed gradual improvement in the SaO_2 and SpO_2 discrepancy. Since admission a serial ABG was done which revealed improvement of the patient gradually. Later the patient developed high anion gap metabolic acidosis (HAGMA) with high lactic acid levels and significantly elevated blood glucose (>25mmol/L), leading to a diagnosis of diabetic ketoacidosis (DKA). Management consisting DKA protocol was commenced, resulting in correction of HAGMA. Concurrently, the patient developed septic shock, necessitating the initiation of empirical broad-spectrum antimicrobials. Other measures according to sepsis guidelines was also taken. Despite initial improvement, the patient's condition further deteriorated, and he ultimately succumbed to his multiple comorbidities and complications.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Instrumentation Labo

PATIENT SAMPLE REPORT

ASGAR ALI HOSPITAL

Status: ACCEPTED
27/04/2024 20:58:40
Sample Type:
Arterial
Sample No.: 47
Patient:
ID: H121-024200
Name:
ICU 12,HARUN
Sex: U
Instrument:
Model: GEM 3500
S/N: 21055798
Name: ASGAR ALI HOS

Measured (37.0C)

pH	7.42	
pCO2	37	mmHg
pO2	414	mmHg
Na+	127	mmol/L
K+	5.1	mmol/L
Ca++	1.11	mmol/L
Glu	278	mg/dL
Lac	2.0	mmol/L
Hct	34	%

Derived Parameters

Ca++(7.4)	1.12	mmol/L
HCO3-	24.0	mmol/L
HCO3std	24.8	mmol/L
tCO2	25.1	mmol/L
BEecf	-0.5	mmol/L
BE(B)	-0.3	mmol/L
SO2c	100	%
THbc	10.5	g/dL

Fig-1: ABG after admission

Fig-2: Dark brown arterial blood

Fig-3: Exchange transfusion

DISCUSSION:

This case highlights the complex interplay of multiple severe comorbidities and the challenges in managing methemoglobinemia in a critically ill patient. Methemoglobinemia is characterized by the oxidation of ferrous iron (Fe^{2+}) to ferric iron (Fe^{3+}) in haemoglobin, rendering it unable to bind oxygen effectively.[3] The condition can be life-threatening, especially in patients with underlying cardiovascular or pulmonary diseases, as in this case. The diagnostic process in this case exemplifies the typical challenges associated with methemoglobinemia. The discrepancy between arterial blood gas and pulse oximetry readings, along with the characteristic dark brown blood colour, served as crucial diagnostic indicators. This "saturation gap" is well-documented in literature as a hallmark of methemoglobinemia. Skold et al. (2011) emphasize that pulse oximetry can be misleading in methemoglobinemia, often reporting falsely low oxygen saturations.[4] Nitzan et al. (2005) further elaborate on the limitations of standard pulse oximetry in such cases, recommending co-oximetry for accurate diagnosis.[5] These findings underscore the importance of maintaining a high index of suspicion and utilizing appropriate diagnostic tools when faced with unexplained hypoxemia. In patients with multiple comorbidities like Mr. Rashid, identifying the exact cause of acquired methemoglobinemia can be challenging. Mansouri and Lurie (1993) discuss various etiologies, including medications, environmental toxins, and underlying health conditions.[6] In this case, the patient's end-stage renal disease (ESRD) and recent haemodialysis session may have played a role.

Ash-Bernal et al. (2004) reported cases of methemoglobinemia associated with renal failure and haemodialysis.[2] Moreover, the oxidative stress associated with chronic diseases like diabetes and cardiovascular disease, both present in our patient, may contribute to methemoglobin formation, as suggested by Umbreit (2007).[7] This complex interplay of factors highlights the need for a comprehensive approach to identifying potential causes in patients with multiple comorbidities. The management of methemoglobinemia in patients with multiple comorbidities requires careful consideration. While methylene blue is a common antidote, it was not used in this case. This decision aligns with recommendations by Ash-Bernal et al. (2004), who cautioned against its use in patients with severe renal impairment.[2] The choice of ascorbic acid and exchange transfusion in this case reflects a tailored approach considering the patient's ESRD. Wright et al. (1999) discuss the efficacy of ascorbic acid in treating methemoglobinemia, particularly in cases where methylene blue is contraindicated.[3] This tailored approach underscores the importance of considering a patient's entire clinical picture when selecting treatment options.

The patient's history of ESRD, diabetes, and cardiovascular disease raises important questions about their potential role in predisposing to methemoglobinemia. Chávez-Iñiguez et al. (2023) reported cases of methemoglobinemia in haemodialysis patients, suggesting a possible link between renal failure and increased risk.[8] Furthermore, the oxidative stress associated with diabetes and cardiovascular disease may contribute to methemoglobin formation, as discussed by Umbreit (2007).[7] These observations highlight the need for further research into the relationship between chronic diseases and methemoglobinemia risk. The use of exchange transfusion in this case was an appropriate alternative therapy, especially given the patient's multiple comorbidities. Exchange transfusion helps remove methemoglobin-containing red blood cells and replaces them with normal red blood cells, improving oxygen-carrying capacity. There is a need for more comprehensive studies on the prevalence and risk factors of methemoglobinemia in patients with chronic diseases, particularly ESRD.[9] Additionally, research into optimized treatment protocols for methemoglobinemia in patients with multiple comorbidities could improve outcomes in complex cases like this one. Such research could lead to more targeted and effective management strategies, ultimately improving patient outcomes.

CONCLUSION:

In conclusion, this case illustrates the complexities involved in diagnosing and managing methemoglobinemia in patients with multiple comorbidities. It emphasizes the importance of clinical vigilance, appropriate diagnostic techniques, and a tailored, multidisciplinary approach to treatment. This case also highlights gaps in our understanding, how chronic diseases may predispose to or complicate methemoglobinemia, suggesting avenues for future research to improve patient care in similar complex scenarios. As our understanding of these complex interactions grows, we can hope to develop.

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