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Case Study

Severe Methemoglobinemia Following Sodium Chlorate Ingestion: A Rare Case Report and Review of Management

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Abstract

Sodium chlorate is a highly effective oxidizing agent primarily used as a non-selective herbicide for weed control and eradication. While its popularity has decreased in some areas due to safety concerns, it continues to be available in agricultural settings, where its low cost and potency make it a viable option for herbicide application. However, its accessibility also poses risks, as it can be used for both accidental and intentional poisoning. Though sodium chlorate toxicity is rare, it is associated with severe health risks and can result in high morbidity and mortality. The toxic effects stem from its systemic oxidative properties, which can damage various organs. One of the most dangerous consequences of poisoning is acute hemolytic anemia, where red blood cells are destroyed at an accelerated rate. Renal failure may also occur due to the kidney's inability to cope with the oxidative stress caused by sodium chlorate. Additionally, methemoglobinemia is a serious complication that can arise. In this condition, the hemoglobin in red blood cells is oxidized to methemoglobin, which is incapable of carrying oxygen, leading to impaired oxygen delivery to tissues and potentially causing life-threatening consequences. Thus, while sodium chlorate remains useful in certain agricultural practices, its risks should not be underestimated.

Keywords: *Acute Hemolytic Anemia; Methemoglobinemia; Oxidizing Agent; Renal Failure; Sodium Chlorate*

Introduction

Methemoglobinemia is a condition characterized by elevated levels of methemoglobin in the blood. Under normal circumstances, hemoglobin contains iron in the ferrous (Fe^{2+}) state, which allows it to reversibly bind oxygen. However, exposure to oxidizing agents like sodium chlorate causes the conversion of ferrous iron to the ferric (Fe^{3+}) state, leading to the formation of methemoglobin. Unlike normal hemoglobin, methemoglobin cannot bind oxygen, resulting in functional anemia and tissue hypoxia despite adequate oxygen levels and blood circulation.

Clinical symptoms of methemoglobinemia can range from cyanosis and shortness of breath to neurological complications such as seizures, arrhythmias, and, in severe cases, death. The primary treatment for methemoglobinemia is methylene blue, an agent that reduces methemoglobin back to its functional ferrous state. However, in cases of sodium chlorate poisoning, the effectiveness of methylene blue is time-sensitive and complicated by ongoing red blood cell destruction and renal impairment.

Due to the rarity of sodium chlorate toxicity, particularly in cases involving methemoglobinemia, there is limited clinical experience and awareness of how to manage these cases. We report a case involving

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a 53-year-old woman who ingested sodium chlorate in a suicide attempt and developed severe methemoglobinemia. This case highlights the challenges of early diagnosis, the importance of prompt antidotal treatment, and the critical care needed for patients with this uncommon and severe toxidrome.

Case Description

A 53-year-old woman with a known medical history of Major Depressive Disorder presented to the Emergency Department (ED) approximately ten hours after intentionally ingesting half a cup of sodium chlorate, a potent weed killer, in a suicide attempt. Upon her arrival, she reported experiencing severe acute abdominal pain, persistent vomiting, and profuse, watery diarrhea. These gastrointestinal symptoms were quickly followed by escalating respiratory distress, widespread generalized cyanosis (a bluish discoloration of the skin), and dark-colored urine, prompting an immediate and thorough assessment along with the rapid initiation of critical care.

Initial vital signs were concerning: a heart rate of 110 beats per minute, blood pressure of 132/78 mmHg, respiratory rate of 28 breaths per minute, and an oxygen saturation of only 83% on room air. The patient was swiftly placed on a high-flow oxygen mask to assist with oxygenation, but despite this intervention, her oxygen saturation remained dangerously low. She continued to exhibit pronounced cyanosis of the lips and extremities. Arterial blood gas analysis revealed a paradoxically elevated partial pressure of oxygen (PaO_2), despite the low peripheral oxygen saturation, which is a key diagnostic indicator of methemoglobinemia. In addition, point-of-care testing confirmed a dangerously high methemoglobin level exceeding 30%, solidifying the diagnosis of severe methemoglobinemia, which is consistent with her exposure to sodium chlorate (Figure 1).

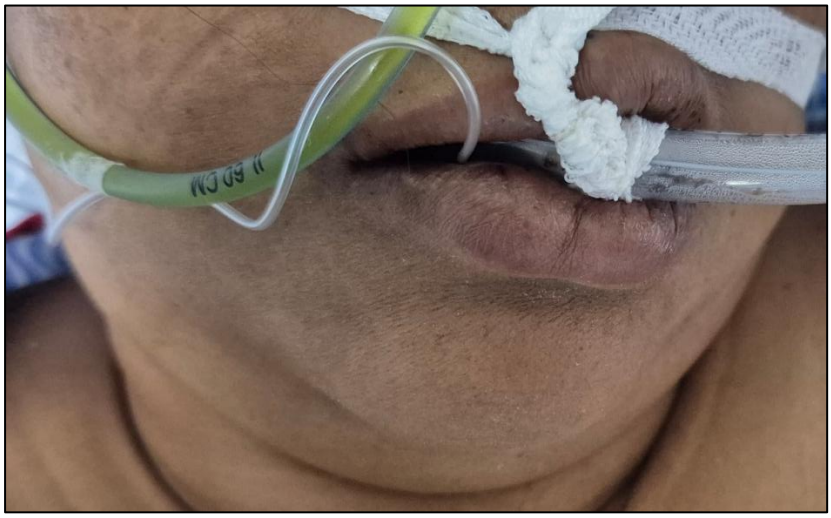

Figure 1: Central Cyanosis Over Lips

Dark-colored urine was noted upon catheterization, consistent with hemoglobinuria, indicating early intravascular hemolysis. Additional laboratory results revealed that her G6PD levels were within the normal range. Renal function was normal upon presentation but showed progressively increasing creatinine levels over the next 24 hours (Figure 2).

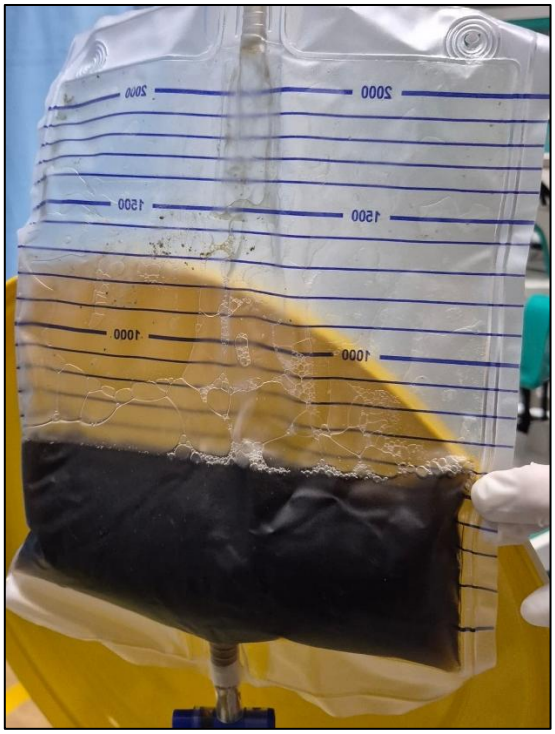

Figure 2: Dark Colored Urine

The patient was treated emergently with intravenous methylene blue at a dose of 1 mg/kg. This dose was repeated two more times over a 4-hour period in the ED due to persistent cyanosis and elevated methemoglobin levels. Her methemoglobin levels gradually decreased to 25%, but her respiratory status continued to worsen. After 1 hour on a high-flow nasal cannula, she became obtunded, with a Glasgow Coma Scale of 8, and required intubation for airway protection (Figure 3).

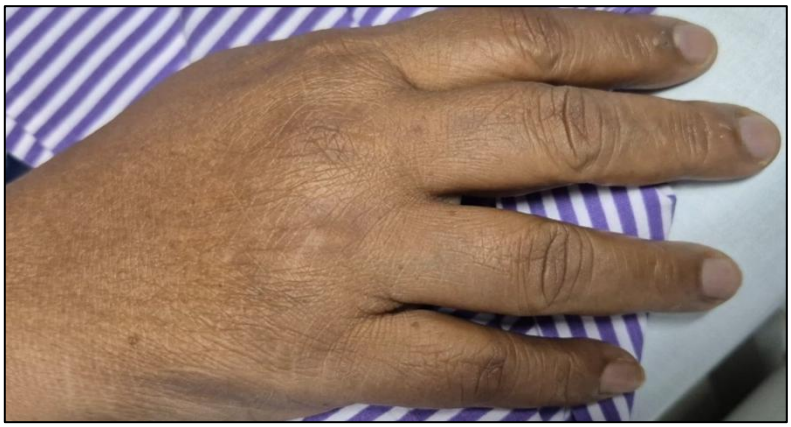

Figure 3: Cyanosis Over Peripheries

She was transferred to the intensive care unit (ICU) for continued monitoring and supportive care, including mechanical ventilation, intravenous fluids, and repeat methylene blue dosing. After receiving intravenous methylene blue at a dose of 7 mg/kg, her methemoglobin level was further reduced to 10%. However, her renal function declined over the next 48 hours, requiring the initiation of renal replacement therapy via dialysis. The Nephrology, Psychiatry, Medical, and Surgical teams were consulted, and she was managed collaboratively using a multidisciplinary approach.

The patient remains in intensive care, requiring mechanical ventilation and intermittent dialysis due to ongoing renal impairment. Her Glasgow Coma Scale (GCS) has shown poor recovery, prompting the need for ventilation through a tracheostomy tube. A CT scan of her brain reveals no signs of intracranial bleeding. She is also being treated for a ventilator-associated infection. Despite nearly a month of

intensive care, her recovery has been limited. As a result, a multidisciplinary meeting with her family was arranged to discuss their expectations and the patient's prognosis.

Discussion

This case illustrates the classic yet rare presentation of methemoglobinemia secondary to sodium chlorate ingestion. Sodium chlorate is a potent oxidant known to cause direct oxidative damage to erythrocytes, leading to methemoglobinemia and hemolysis (Obodozie et al., 2024). The toxicity of chlorate salts is dose-dependent, with even small amounts (5–10 g) potentially being lethal in adults due to their systemic effects.

The pathophysiology of methemoglobinemia involves the oxidation of hemoglobin's iron component from the ferrous (Fe^{2+}) to the ferric (Fe^{3+}) state. This oxidized hemoglobin, methemoglobin, cannot carry oxygen, resulting in functional anemia. Additionally, the presence of methemoglobin alters the oxygen-hemoglobin dissociation curve, impairing the release of oxygen to tissues, which worsens hypoxia (Samuel, 2023).

The clinical features of methemoglobinemia are largely dependent on the proportion of hemoglobin that has been oxidized (Samuel, 2023):

- 10–20%: Cyanosis, often without symptoms
- 20–50%: Dyspnea, headache, fatigue, dizziness
- 50%: Altered mental status, arrhythmias, seizures
- 70%: Potentially fatal due to profound hypoxia

In this case, the patient presented with cyanosis, respiratory distress, and mental status changes when her methemoglobin level exceeded 30%. These symptoms prompted immediate consideration of methemoglobinemia in the differential diagnosis. A key diagnostic clue was the discrepancy between the high partial pressure of oxygen (PaO_2) and low oxygen saturation on pulse oximetry—a "saturation gap" typical in methemoglobinemia, which should prompt confirmatory co-oximetry testing.

The cornerstone of treatment is intravenous methylene blue, which acts as a co-factor in the enzymatic reduction of methemoglobin back to hemoglobin. Methylene blue is reduced to leukomethylene blue via NADPH-dependent methemoglobin reductase, and leukomethylene blue then reduces ferric iron to the ferrous form, restoring oxygen-carrying capacity. The standard dose is 1–2 mg/kg intravenously over 5 minutes, with additional doses as needed (Iolascon et al., 2021).

However, the efficacy of methylene blue in sodium chlorate poisoning may be limited. Sodium chlorate not only causes methemoglobinemia but also induces direct oxidative hemolysis. As red blood cells are destroyed, the ability to reduce methylene blue is diminished due to decreased NADPH availability and reduced erythrocyte viability. Furthermore, renal toxicity—likely resulting from hemoglobin precipitation and oxidative injury—complicates clearance and recovery (Iolascon et al., 2021). While methylene blue remains a key treatment, it did not eliminate the need for renal replacement therapy (dialysis), which may assist in removing the offending agent from the circulatory system, even in the absence of renal impairment.

In this patient, early methylene blue administration likely prevented further increases in methemoglobin levels but did not prevent progression to respiratory failure and acute kidney injury. Nevertheless, aggressive supportive care, timely ICU transfer, and repeated methylene blue dosing were crucial for achieving clinical recovery.

There is limited literature on chlorate-induced methemoglobinemia, especially sodium chlorate toxicity. Most case reports are over a decade old. One of the few reports documented a similar case, where a patient developed life-threatening methemoglobinemia and hemolysis following sodium chlorate ingestion and required intensive care, including dialysis. This underscores the importance of early recognition, consultation with toxicology experts, and comprehensive supportive measures.

Conclusion

Sodium chlorate poisoning is a rare but potentially fatal condition characterized by severe oxidative stress, methemoglobinemia, hemolysis, and renal failure. Emergency physicians and critical care providers should be vigilant for this toxidrome, especially in patients presenting with unexplained cyanosis, hypoxia unresponsive to oxygen therapy, and a saturation gap.

Methemoglobinemia should be suspected when oxygen saturation fails to improve despite high PaO_2 levels, and confirmation requires co-oximetry. Early administration of methylene blue is crucial, although its effectiveness may be limited in cases with concurrent hemolysis and renal dysfunction. Aggressive supportive care, including airway protection, fluid management, and renal support, is essential for survival.

This case emphasizes the need for heightened clinical suspicion, prompt antidotal treatment, and interdisciplinary management in patients with suspected oxidant poisoning. Further research is needed to establish evidence-based protocols for the treatment of chlorate toxicity and to explore adjunctive therapies beyond methylene blue.

Conflict of Interest

The author(s) declare that there is no conflict of interest regarding the publication of this article.

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