

Acute Sodium Nitrite Poisoning and Methemoglobinemia Following Suicidal Attempt: A Case Report and Literature Review Identificativo

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Left: Patient initially with 64% methemoglobin, peripheral oxygen saturation 84%, and PaO2 of 188 mm. Note the PaO2-saturation gap (normally a PaO2 of 188mm should produce an oxygen saturation of 100%).

Right: Improvement following treatment with 60 mg methylene blue and 500 mg ascorbic acid.

ABSTRACT

Sodium nitrite poisoning is a rare but potentially life-threatening condition, often resulting from intentional ingestion in suicide attempts. This case report describes a 26-year-old male who presented with unconsciousness, cyanosis, and respiratory distress following the ingestion of sodium nitrite in a suicidal attempt. Early recognition and treatment with methylene blue are critical for reversing the toxic effects of sodium nitrite and preventing life-threatening complications. This report emphasizes the need for emergency physicians to be aware of non-traditional toxins in poisoning cases and to promptly initiate appropriate treatment to improve patient outcomes.

CASE REPORT

A 26-year-old male was found unconscious in his car near a cemetery, surrounded by vomit. On the passenger seat, a white granular substance, a kitchen scale, and a glass of water were discovered, raising suspicion of poisoning. On EMS arrival, he exhibited bradypnea (10 breaths/min), cyanosis, pallor, and a low Glasgow Coma Scale score (GCS 7), but no signs of trauma. His oxygen saturation (SpO₂) was critically low at 75%, heart rate 110 bpm, blood pressure 120/85 mmHg, and body temperature 33.2°C.

Initial treatment for suspected opioid overdose included oxygen therapy and two doses of naloxone, with no clinical improvement. Due to persistent hypoxia, he was intubated and mechanically ventilated, yet SpO₂ remained at 80%.

Upon hospital arrival, arterial blood gas analysis showed metabolic acidosis (pH 7.23), elevated lactate (7.8 mmol/L), a PaO₂ of 605 mmHg, and a non-measurable methemoglobin level.

Following consultation with Poison Control, the patient received **methylene blue** (1 mg/kg IV), which led to a progressive improvement in oxygen saturation and clinical status. He was admitted to ICU, continued on supportive care, and extubated successfully within 48 hours. He later admitted to intentionally ingesting sodium nitrite purchased online as a food preservative. He recovered fully without long-term complications and was discharged after further observation.

TOXICOLOGY UNFOLDED

Sodium nitrite (NaNO₂), a **chemical commonly used as a food preservative** is known for its role in maintaining color and preventing bacterial growth. However, it also poses significant toxic risks, particularly when ingested in large quantities, **often seen in suicide attempts or self-harm**.

Sodium nitrite toxicity is rooted in its ability to convert hemoglobin from its normal ferrous state (Fe²⁺) to a ferric state (Fe³⁺), forming methemoglobin. Methemoglobin cannot bind oxygen, nor can it release it to tissues effectively, thus disrupting oxygen transport and leading to hypoxia.

The clinical features of methemoglobinemia are directly related to the level of methemoglobin in the blood.

Clinical presentation can range from asymptomatic methemoglobinemia to symptoms as cyanosis (particularly around the lips and extremities), dyspnea, headache, dizziness, confusion, tachycardia, fatigue, lethargy, respiratory distress, arrhythmias, and, if left untreated, coma or death.

The treatment involves the use of methylene blue, which facilitates the conversion of methemoglobin back to functional hemoglobin, by donating electrons from NADPH. The recommended dose of methylene blue is 1-2 mg/kg IV. Cut-off for treatment is symptomatic methemoglobinemia with concentrations under 30% or methemoglobinemia above 30%. If possible, it is strongly suggested consultation with Poison Control.

IF YOU HEAR HOOFBEATS, THINK ALSO ZEBRA

Methemoglobinemia often causes the pulse oximeter to show an oxygen saturation of around 82–86%, even when the arterial oxygen pressure (PaO₂) is very high. This leads to a situation of "refractory hypoxemia" - oxygen saturation remains low despite 100% FiO₂ being administered.



This creates a noticeable discrepancy between the PaO₂ (in our case of 605 mmHg) and the pulse oximetry reading. **This mismatch is called the PaO₂ - oxygen saturation gap**, a key sign of a hemoglobin abnormality, most commonly methemoglobinemia.

TAKE HOME MESSAGE

The early recognition of methemoglobinemia and the prompt administration of methylene blue were pivotal in reversing this life-threatening condition. Timely intervention with methylene blue can be life-saving, underscoring the need for a high index of suspicion and rapid response in suspected cases of methemoglobinemia.

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