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Case Report

Navigating Methemoglobinemia: A Sodium Nitrite Poisoning Case Report and Therapeutic Considerations

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Abstract

Incidences of sodium nitrite poisoning have frequently increased in the last few years with the wide, easy availability of the drug and the increase in suicidal tendencies in the population. Sodium nitrite is widely used as a food preservative and in treating cyanide toxicity. It is a powerful oxidizing agent, which oxidizes hemoglobin to methemoglobin, reducing the oxygen-carrying capacity of erythrocytes and causing tissue hypoxia. Patients with sodium nitrite toxicity present to emergency with hypotension, cyanosis, hypoxia, altered consciousness, dysrhythmia, and cardiac arrest. We report a case of a suicide survivor who presented with an intentional fatal overdose of sodium nitrite in an 18-year-old female by drinking approximately one tablespoon of sodium nitrite in a suicidal attempt. Upon arrival at the emergency department (ED), the patient was hypotensive, cyanotic, and cyanosis non-responsive to oxygen therapy. Hospital professionals should consider sodium nitrite toxicity in patients with a suspected overdose who present with a cyanotic appearance and pulse oximetry that remains around 85% despite oxygen and dark brown blood seen on venipuncture. Prompt diagnosis is crucial to start early treatment.

Keywords: sodium nitrite, poisoning, cyanosis, suicide

Abbreviations: ABG: arterial blood gas

1. Introduction

Of all the suicidal methods, poisoning ranks third among other methods, with an incidence of 28.6%, according to the National Institute of Health survey. Sodium nitrite self-poisoning incidence has increased over a few years, following the easy availability of the drug and substantial upsurge in suicide intoxication in recent years.

2. Case Report

An 18-year-old female presented to our hospital emergency with an alleged history of intentional ingestion of white crystalline powder 5–6 hours back. She presented with abdomen pain, vomiting, and breathlessness. On examination, the patient was drowsy with bluish discoloration of lips, nails, and tongue. Vitals revealed a heart rate of 142 per minute, blood pressure of 102/60 mmHg, and SpO2 was 78% on room air. Immediately, oxygen therapy was started with non rebreathing mask (with reservoir); however, the cyanosis remained unresponsive. The patient was shifted to the ICU, intravenous access was obtained, and a sample of blood was taken for arterial blood gas (ABG) analysis and other routine investigations, which was dark brown in color on venipuncture. Ryle's tube was inserted, and gastric lavage was done with charcoal. ABG with CO-oximetry revealed metabolic acidosis with a methemoglobin level of 65.7%. Injection methylene blue 100 mg was administered intravenously in 100 mL normal saline over 5–10 minutes and 50 mg after one hour. Injection of sodium bicarbonate 50 mEq was given intravenously slowly. Injection ascorbic acid 1gm intravenously was given over 1 hour. Within 1 hour of methylene blue administration, her symptoms improved significantly, and methemoglobin levels decreased to 15.6%. Diagnosis of sodium nitrite poisoning was made, and methemoglobin levels were assessed at regular intervals. Injection of methylene blue 50 mg in 100 mL normal saline was repeated twice in 48 hours whenever levels > 20%. Methemoglobin levels decreased to 1.6% after 48 hours. On detailed history, the patient was suffering from some psychiatric illness for which she was on irregular treatment. She obtained sodium nitrite from the chemical lab she was

working in. She was discharged without any complications after a neuropsychiatric evaluation and advice to follow up with a psychiatrist.

3. Discussion

Nitrites induce toxicity by producing methemoglobin from the oxidation of ferrous iron (Fe^{2+}) to ferric iron (Fe^{3+}) in hemoglobin. Methemoglobin has a characteristic chocolate brown color appearance and is often recognized as an abnormal color during blood sampling. Unlike normal hemoglobin, methemoglobin does not bind to oxygen, resulting in functional anemia, diminished oxygen delivery to tissues, and the development of lactic acidosis. The oxidizing properties of sodium nitrite can also cause hemolysis, which further impairs oxygen delivery. Also, nitrites act as potent vasodilators in the peripheral vasculature, causing vasodilatory shock [1].

The lethal dose of sodium nitrite in adults has a broad range between 0.7 and 6 g of nitrite component [2]. However, based on the therapeutic dose of sodium nitrite used in cyanide poisoning in a typical adult, the lethal dose is approximately 2.6 g. This accounts for the fact that even a teaspoonful of sodium nitrite has the potential to be lethal.

The first-line antidote therapy for acute toxic methemoglobinemia with methemoglobin levels of > 30% is methylene blue. Methylene blue is also used in patients symptomatic with methemoglobin levels between 20% and 30%, especially those with high-risk factors such as anemia or pulmonary or cardiac comorbidities. It is administered at a dose of 1–2 mg/kg body weight intravenous over 5 minutes. Most cases show rapid clinical improvement with methylene blue and reduction of methemoglobin levels to < 10% within 10–60 minutes. It may be repeated within 1 hour if the methemoglobin level remains high (e.g., > 20%) and/or has an increasing trend. Rapid diagnosis and early intervention with methylene blue infusion can prevent a fatal outcome with an initial methemoglobin level as high as 92.5% [3].

However, administration of > 2–3 doses (> 7 mg/kg) is generally avoided because of the possible risk of hemolysis. Alternative treatments are taken into consideration which include RBC or exchange transfusions to replace dysfunctional hemoglobin and hyperbaric oxygen therapy [4].

Sodium nitrite poisoning causes methemoglobinemia, and therefore, ascorbic acid has been proposed as a potential treatment for this condition due to its ability to reduce methemoglobin back to normal hemoglobin. Ascorbic acid acts as an antioxidant and can convert the oxidized form of hemoglobin (methemoglobin) back to its normal, functional state (reduced hemoglobin). By doing so, it helps to restore the blood's oxygen-carrying capacity. However, it is important to note that the use of ascorbic acid in sodium nitrite poisoning is still a subject of debate and research. The appropriate dosage and timing of administration are not well-established, and there may be other factors to consider based on the severity of the poisoning and the individual's overall health condition [5].

4. Conclusion

Methylene blue is the first-line antidote used in the treatment of sodium nitrite poisoning. It acts as a cofactor for NADPH MetHb reductase in a dose of 1–2 mg/kg intravenous over 5 minutes. However, N-acetyl cysteine, exchange transfusion, and hyperbaric oxygen therapy have also been used in the treatment of sodium nitrite poisoning.

Methemoglobinemia is a rare but life-threatening condition. Rapid diagnosis and early intervention are key to preventing fatal outcomes. Controlling and monitoring the sale of sodium nitrite can be highly difficult due to its wide application in food industries as food preservatives and local laboratory supplies in relatively high quantities. A clinical suspicion of sodium nitrite intoxication should be warranted in cases of unexplained cyanosis or methemoglobinemia for better patient outcomes.

Conflicts of Interest

The authors declare no conflict of interest.

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