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CASE REPORT

Toxicology

Survival after self-poisoning with sodium nitrite: A case report

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Abstract

Sodium nitrite ingestion poses a considerable public health threat. The incidence of sodium nitrite self-poisoning in the United States has been trending upward since 2017. Our case report describes an intentional sodium nitrite ingestion with favorable outcomes. We highlight the proper treatment of this ingestion with intravenous methylene blue. Sodium nitrite is an oxidizing agent that is commonly found in processed meats, fish, and cheeses as a preservative, antimicrobial, and food coloring agent. It is an odorless, white crystalline powder that has been confused for table salt or granulated sugar. It has become more readily available in large quantities online. Unfortunately, online forums exist that detail how to dose sodium nitrite for suicide. Furthermore, it has been recently discussed in popular news streams after a celebrity died of an overdose. Sodium nitrite toxicity is capable of causing severe methemoglobinemia with high mortality. Prompt identification is crucial. We discuss the important implications in regard to media coverage, imitative suicide, and accessibility of sodium nitrite.

KEYWORDS
ingestion, methemoglobinemia, poisoning, sodium nitrite, toxicology

1 | INTRODUCTION

Sodium nitrite ($NaNO_2$) oxidizes heme iron from the ferrous (Fe^{2+}) state to the ferric (Fe^{3+}) state. This changes the redox state of heme, causing the removal of an electron.[1–5] When oxidation occurs within the heme moiety of hemoglobin, methemoglobin is formed. This impairs the delivery and exchange of oxygen to tissues as methemoglobin is incapable of carrying oxygen or carbon dioxide.[6,7] Excess methemoglobin can lead to impaired aerobic respiration, cyanosis, metabolic acidosis, and death.[8] As a result of methemoglobinemia, the skin can be cyanotic even at levels at which the patient would be otherwise asymptomatic.[8,9]

As a chemical asphyxiant, sodium nitrite affects vital organs, especially those with high oxygen dependence, such as the brain and heart. Clinical manifestations of sodium nitrite poisoning include peripheral cyanosis, headache, skin flushing, altered skin color, orthostatic hypotension with reflex tachycardia, hypoxia, altered mental status, nausea, vomiting, diarrhea, loss of consciousness, dysrhythmias, and death.[5]

2 | CASE

A 23-year-old male with a medical history significant for previous suicide attempts, polysubstance use disorder in remission from alcohol and benzodiazepine use, nicotine dependence, current marijuana use, bipolar disorder, post-traumatic stress disorder, and irritable bowel syndrome presented to the emergency department via emergency medical services (EMS) after intentional overdose. Patient reported ingesting 2 teaspoons of sodium nitrite he obtained from Walmart.com 6 hours before presentation in an attempt to end his life.

The patient reported losing consciousness after ingestion and waking up in his own vomit and feces. He felt confused and called EMS. In the emergency department (ED), the patient acknowledged nausea,

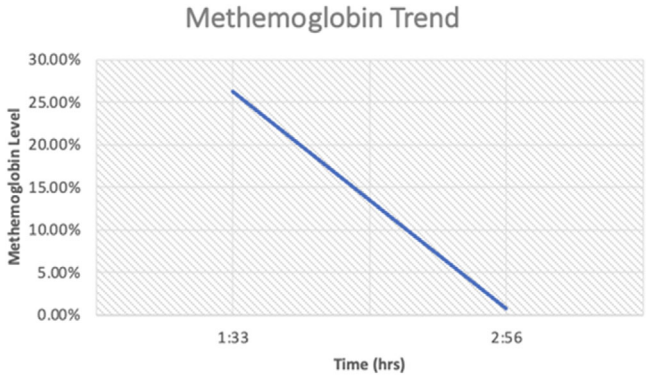

FIGURE 1 Methemoglobin levels measured versus time. Intravenous methylene blue was administered at 02:01 am, demonstrating the rapid efficacy of the antidote

vomiting, and bowel incontinence. He complained of severe diffuse abdominal pain that was 8/10 in severity. He had used marijuana that day. He denied chest pain, shortness of breath, coingestion with other prescription drugs, over-the-counter drugs, street drugs, and ethanol. He had no auditory hallucinations, current suicidal ideation, or current homicidal ideation.

The patient presented to the ED appearing ill, ashen, discolored and in distress. He was afebrile, tachycardic to 108, with a respiratory rate at 22, and blood pressure in the 110s systolic. His oxygen saturations were 84% on 6 L nasal cannula. On examination, his skin was ashen gray and he had perioral cyanosis. Cranial nerves were intact, and his cardiopulmonary examination demonstrated mild tachycardia. There were no pulmonary abnormalities. He had diffuse tenderness on his abdomen without rebound, rigidity, or guarding. He had soiled himself and was actively retching.

In the ED, he was switched to a non-rebreather mask. Minnesota Poison Control was contacted, and the patient was promptly treated with 1 mg/kg intravenous bolus of methylene blue over 5 minutes. Toxicology labs were obtained along with venous blood gas (VBG) with co-oximetry and methemoglobin levels. He remained stable in the ED, then was transferred to the intensive care unit (ICU) for further evaluation and care. Initial methemoglobin level was found to be 26.3% (0.0-1.5%). VBG demonstrated pH of 7.39, PCO_2 to 33 mmHg with PO_2 of 38 mmHg. Complete blood count demonstrated platelet count of 329 and leukocytosis to 23.3 with strong left shift of 18.75. Basic metabolic panel demonstrated a mild decrease in bicarbonate at 19 (22-29 mmol/L) and increased anion gap 16 (7-15). Urine drug screen was positive for tetrahydrocannabinol and amphetamines. He denied use of amphetamines, which may be a false positive from bupropion.[10]

He was cared for in the ICU overnight on non-rebreather, then high-flow nasal cannula, and ultimately transitioned to room air. From a clinical perspective, the patient improved drastically after timely methylene blue treatment with progressively decreasing oxygen requirements. His repeat methemoglobin level trended down to 0.7% within 90 minutes of intravenous methylene blue administration (Figure 1). The patient was transferred to the general medicine floor the next day. He was discharged after 2 nights total in the hospital for further psychiatric care. The patient continues to do well at the time of this report without identifiable long-term sequela.

FIGURE 2 Sodium nitrite purchased online[21]

3 | DISCUSSION

The identification of sodium nitrite ingestion and antidote administration are time sensitive. A thorough history from the patient, family, friends, or EMS can delineate the type of ingestion. Finding a container with the product may also be helpful, if available. Sodium nitrite may be sold in a bottle or package (Figure 2). It may also be labeled as a "curing salt".[11,12] Without corroborating information, a patient with cyanosis and low oxygen levels without respiratory disease should raise suspicion for sodium nitrite poisoning. A Poison Control resource should be contacted as soon as ingestion is suspected. Diagnostic tests include electrolytes, arterial blood gas or oximetry, methemoglobin concentration, glucose, and ECG. Patients should be placed on supplemental oxygen and labs should be obtained as soon as possible. A hallmark finding for sodium nitrite poisoning is unexpectedly high saturation in the setting of high levels of methemoglobin. Our patient had oxygen saturations of 86% despite methemoglobin levels of 26%. This is because pulse oximeter saturation overestimates the fractional oxygen

saturation in the setting of methemoglobinemia.[13] It is not necessary to wait for methemoglobin levels to come back if clinical suspicion is high.[14,15]

Sodium nitrite causes methemoglobinemia and hypotension. The recommended treatment of sodium nitrite and resultant methemoglobinemia is intravenous methylene blue. The dose is typically 1–2 mg/kg intravenously over 5 minutes,[8] with a maximum dose of 7 mg/kg, even in cases of severe methemoglobinemia. Larger doses may paradoxically induce methemoglobinemia.[15,16] It is an inhibitor of nitric oxide synthase and guanylate cyclase.[17] This chemical causes a reduction of methemoglobin to functional hemoglobin, allowing oxygen delivery to tissues. Improvement may be seen within minutes of administration. Our patient demonstrated rapid clinical improvement evidenced by a decrease in oxygen requirements, change in skin color, improvement in mental status, and drop in methemoglobin levels less than 2 hours after treatment.[15,16]

Sodium nitrite ingestions may continue to become more common as a result of its ease of access. The California Poison Control System (CPCS) was consulted on 5 patients who intentionally ingested sodium nitrite between May and November of 2019. All cases acquired the product from online vendors. There were no cases reported to CPCS in the 5 years prior.[2] Our case occurred only 4 days after Matthew Mindler, a child actor in the movie "My Idiot Brother," was reported to have ingested sodium nitrite. The case was featured on multiple news streams including People, USA Today, NBC News, and CBS. It is reported that the child star was able to find instructions on how to ingest a lethal dose of sodium nitrite via online forums,[4] and purchased the chemical on Amazon.[5] Similarly, our patient reported purchasing the chemical on Walmart's website. The occurrence of our ingestion in close proximity to a celebrity ingestion may be a coincidence, but it leads to further questions surrounding the ethics of reporting novel and dangerous methods of suicide. The ease at which vulnerable populations may obtain access to information or products that can cause self-harm should be considered. In 1 meta-analysis, the risk of suicide increased by 13% after the media reported death of a celebrity by suicide. The effect was larger in the same method used by the celebrity.[18] There are data to suggest that guidelines for reporting do have an impact on imitative suicide.[19,20] Further implications include regulations surrounding access to toxic chemicals and online forums that detail how to ingest lethal doses.

In conclusion, sodium nitrite is a toxic chemical that can cause severe methemoglobinemia and even death. The ease of access to the chemical, increasing information on overdose online, popularization in mainstream media, or a combination of these factors may have contributed to our patient's decision to use the chemical as a means of suicide. Prompt identification and delivery of intravenous methylene blue is crucial in order to maximize the chance of a favorable outcome. Understanding the social and ethical factors that contribute to the access of this chemical are points of future discussion.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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