Open Journal of
Clinical & Medical
Case Reports

Volume 12 (2026)
Issue 5

Case Report
ISSN: 2379-1039

Successful reversal of severe methemoglobinemia caused by intentional sodium nitrite ingestion: A case report and mini-review

Christopher Lai Hipp*; Nishal Brahmbhatt; Robin Inaba

***Corresponding Author: Christopher Lai Hipp**
The Queen's Medical Center, Honolulu, Hawaii, USA.
Email: claihipp@queens.org

Abstract

Severe methemoglobinemia following intentional sodium nitrite ingestion is an escalating public health concern owing to its increasing incidence and substantial mortality risks. We report a case of a 32-year-old woman who presented with profound hypoxia, cyanosis, and persistently low oxygen saturation unresponsive to supplemental oxygen after ingesting sodium nitrite misidentified as "Hawaiian sea salt." Diagnostic challenges included discordant pulse oximetry and arterial blood gas findings, and initial methemoglobin quantification that was unsuccessful due to extremely elevated levels and analytic limitations. Empirically administered intravenous methylene blue resulted in rapid clinical and biochemical improvements without complications. This case underscores the importance of recognizing characteristic signs, such as the occurrence of chocolate-brown blood, and utilizing co-oximetry for accurate diagnosis. Early treatment with methylene blue is critical for reversing life-threatening methemoglobinemia. Given the widespread availability of sodium nitrite and its promotion in online suicide forums, heightened clinical vigilance is essential for timely diagnosis and intervention to improve patient outcomes.

Introduction

Methemoglobinemia is a disorder characterized by the oxidation of iron within hemoglobin from the Ferrous (Fe^{2+}) to the Ferric (Fe^{3+}) state, impairing its ability to bind and transport oxygen and resulting in functional hypoxia despite adequate circulating oxygen levels [1,2]. Clinically, patients often present with cyanosis that is refractory to supplemental oxygen, with dark or chocolate-brown arterial blood, and pulse oximetry values that may plateau near 85%, which can delay recognition and treatment [3]. Although methemoglobinemia has traditionally been associated with exposure to topical anesthetics, nitrates, or certain antimicrobial agents, there has been a notable increase in intentional sodium nitrite ingestion, particularly as a method of self-harm [3,4]. Sodium nitrate is inexpensive, widely accessible online, and is increasingly promoted through online suicide forums, making it an emerging public health threat [5]. Without prompt intervention, severe cases may rapidly progress to profound tissue hypoxia, metabolic acidosis, seizures, cardiovascular collapse, and death. Methylene blue remains the first-line antidote, acting as an electron acceptor to reduce methemoglobinemia to the functional ferrous state; however, timely recognition and administration are critical for survival. Although the number of reported cases of intentional sodium nitrite ingestion has increased, published reports frequently describe poor outcomes. Here, we present a case of severe methemoglobinemia following intentional sodium nitrite ingestion that was successfully reversed with early methylene blue administration, highlighting the key diagnostic and therapeutic considerations that are important for emergency and critical care clinicians as the incidence of this poisoning continues to rise.

Case Presentation

A 32-year-old woman with a medical history of alcohol and polysubstance use, schizoaffective disorder, attention deficit disorder, anorexia nervosa, seizure disorder, and post-traumatic stress disorder presented to the Emergency Department (ED) via Emergency Medical Services (EMS) after calling 911 and reporting an intentional suicide attempt followed by regret. According to the EMS report, the patient stated that she had ingested approximately 5 pounds of what she described as "Hawaiian sea salt," approximately 30 min before their arrival. At the scene, she was found lying on the ground, lethargic, with urinary incontinence and actively vomiting. EMS staff noted a nearly empty bag of white granular material near the patient, which was initially presumed to be sea salt.

On arrival at the ED, the patient was lethargic, diaphoretic, and markedly pale with gray skin discoloration. Her initial Oxygen Saturation (SpO_2) was 80% on room air, and she was immediately placed on 15 L of oxygen via a non-rebreather facemask without improvement. The patient reported associated nausea and vomiting and admitted to concurrent alcohol use, but denied other ingestions. Her vital signs were notable, with a systolic blood pressure of 90 mmHg. Based on her physical presentation, acute anemia was suspected. Subsequent laboratory evaluation revealed a serum sodium level of 155 mmol/L and a point-of-care hemoglobin level of 13.9 g/dL, which was later confirmed by laboratory-reported hemoglobin concentrations. This finding was inconsistent with the initial suspicion of severe anemia. Arterial blood gas analysis revealed a pH of 7.17, partial Pressure of Oxygen (PaO_2) >400 mmHg, and calculated oxygen saturation of 100%. Despite high-flow oxygen therapy via a non-rebreather mask, the patient's SpO_2 remained between 79% and 85%. Manual ventilation via a bag-valve device failed to improve oxygenation, and the patient was subsequently intubated for progressive hypoxic respiratory failure. Postintubation chest radiography revealed no evidence of pulmonary edema, pneumonitis, or pneumothorax. Electrocardiography revealed normal QRS duration and QTc values.

Given the marked cyanotic discoloration, profound refractory hypoxia despite 100% FiO_2, and the discordance between pulse oximetry and arterial oxygen tension, methemoglobinemia was suspected. Initial laboratory attempts to quantify the methemoglobin concentration resulted in instrument errors in three different samples, which the laboratory attributed to extremely elevated methemoglobin levels that exceeded the analytical range of the device. Following consultation with a clinical pharmacist, empirical treatment with methylene blue was initiated intravenously at a dose of 1 mg/kg. Ascorbic acid was considered an adjunctive therapy but was unavailable. Following the initial methylene blue dose, the patient's SpO_2 increased to approximately 85% but remained below the expected levels, and a second dose of 1 mg/kg was administered after transfer to the intensive Care Unit (ICU). Subsequent measurement approximately 3 h after the second dose revealed a methemoglobin level of 0.7%. The patient did not require vasopressor support. Her initially elevated lactate level (9.2 mmol/L) and metabolic acidosis were corrected within approximately 8 h of treatment initiation. The patient was successfully extubated on the following day. The hypernatremia was gradually reversed with intravenous administration of D5W. Rebound methemoglobinemia, hemolysis, or acute kidney injury were not observed. Following medical stabilization, the patient was transferred to an inpatient psychiatric unit and discharged. The total hospital length of stay was 8 days.

Shortly after ICU admission, a review of outpatient psychiatric records by our colleagues revealed prior documentation that the patient had stated she had researched suicide methods involving the ingestion of cured salts. This, along with the near-empty bag located at the scene, suggested that the ingested substance was sodium nitrite and that the reference to "Hawaiian sea salt" was an initial miscommunication during the pre-hospital handoff.

Discussion

Epidemiology and public health context

The first case of intentional sodium nitrite/nitrate ingestion for suicide was reported in 2010; however, its incidence has increased sharply in recent years. Hickey et al. reported a case series from Ontario, Canada, showing that most cases occurred in the last 2 years of their 1980–2020 review, highlighting a new and growing trend [4]. Khan et al. analyzed U.S. data from 2018 to 2020 and found 260 sodium nitrite/nitrate suicide deaths, with a marked increase in 2020, and a typical victim profile of young white males with depression [5]. McCann et al. reported rising cases and high mortality rates in the U.S. poison center data since 2017, with most exposures occurring at home and a mortality rate of 30% [6]. Bloom et al. confirmed this trend, noting that clusters of cases were often linked to information obtained online [7].

Multiple scholarly articles have directly implicated online forums and Internet resources for the marked increase in the intentional ingestion of sodium nitrite for self-harm. A special report from the Centers for Disease Control and Prevention found that online content and forums influenced changes in suicide methods, including sodium nitrite ingestion, and the popularity of Google searches for these methods has increased since 2019 [8]. Some authors have specifically noted that online forums are used to share the procurement methods and preparation details for sodium nitrite poisoning [5]. Some sites described "suicide kits" assembled with instructions from pro-suicide forums, often with recommendations to co-ingest antiemetics and antidiarrheal agents to improve their absorption [7]. A 2023 case report of an adolescent suicide using sodium nitrite ordered online emphasized the ease of access and the role of web-based instructions [9]. Mörch et al. provided a broader context, showing that pro-suicide forums on both the surface and dark web offer detailed information on suicide methods (including poisoning) and are associated with increased public health risks [10]. Statistical associations have been demonstrated between increased Google searches for pro-suicide websites and subsequent increases in suicide rates due to poisoning, particularly among youths and young adults [11].

Pharmacology and pathophysiology

Sodium nitrite is a yellowish-white, crystalline, odorless, inorganic substance that often appears similar to table salt [12]. It has various clinical and industrial applications. Its primary clinical use is for the treatment of cyanide poisoning [13]. In the food industry, sodium nitrite is used as a preservative to inhibit bacterial growth and exploit its interaction with myoglobin to enhance the appearance, color, and taste of certain meat-containing products (giving the compound its common name "curing salts") [14]. Sodium nitrite is highly water-soluble, allowing large amounts of the compound to easily dissolve in a liquid medium, facilitating rapid ingestion. After ingestion, sodium nitrite has a bioavailability of 98% and minimal first-pass metabolism in the liver [15]. These pharmacokinetic aspects of sodium nitrite are reflected in the rapid onset of symptoms and clinical deterioration often observed in patients who ingest large amounts of sodium nitrite. The reported lethal dose of sodium nitrite after intentional ingestion is highly variable, with a broad range of doses reported between 0.7 and 6 g of nitrite [16]. With ease of access to sodium nitrite-containing compounds and a lack of regulation, patients interested in self-harm can easily ingest toxic or lethal doses.

After the oral ingestion of sodium nitrite, the acidic environment of the stomach facilitates the chemical transformation of a portion of the salt into Nitric Oxide (NO) and other nitrogen compounds, while the rest enters the systemic circulation unchanged [17]. Once in systemic circulation, nitrite interacts with hemoglobin molecules found within red blood cells, causing the bound Ferrous Iron (Fe^{2+}) of oxyhemoglobin to oxidize into the bound Ferric Iron (Fe^{3+}) of methemoglobin [3,4,18]. The oxidized form of Fe cannot bind to oxygen molecules. Additionally, the presence of one or more Fe^{3+} ions within the hemoglobin tetramer causes the remaining Fe^{2+} groups to have a higher affinity for their bound oxygen molecules, thus preventing their subsequent release at the target sites. Taken together, these changes result in effective anemia due to the decreased transport of oxygen by methemoglobin, causing a shift in the oxygen dissociation curve to the left [2-4]. Blood containing methemoglobin is classically described as "chocolate brown" due to the iron molecules in their Fe^{3+} state, which alters the optical properties of blood. The degree of discoloration correlates with the methemoglobin concentration, with higher levels producing a more pronounced brown color, which is often observed during both venous and arterial blood collections [19-21]. In addition to methemoglobinemia, sodium nitrite has been reported to cause hypotension (via nitric oxide) and hemolysis [22].

Clinical presentation and diagnostic challenges

Patients who ingest sodium nitrite most often present with central cyanosis, which is most evident in the oral mucosa and lips, along with shortness of breath, fatigue, and sometimes altered mental status. Cyanosis due to methemoglobin typically occurs when blood levels exceed 10–15% of the total hemoglobin level and is a key diagnostic clue [3,19,20]. The diagnosis of methemoglobinemia requires careful interpretation of clinical and laboratory findings because the condition often presents as cyanosis that is not corrected with supplemental oxygen. Oxygen-refractory cyanosis occurs due to the inability of methemoglobin to perform normal oxygen transport and exchange, thereby reducing functional hemoglobin levels despite adequate dissolved oxygen in the plasma [23,34].

Pulse oximetry can be misleading in patients with methemoglobinemia. In standard pulse oximetry, light absorption is measured at wavelengths of 660 and 940 nm, which provides the oxyhemoglobin-to-deoxyhemoglobin ratio. This is then translated into an estimated percentage of blood-oxygen saturation, where a ratio of 1 corresponds to an approximate blood-oxygen saturation of 85%. This relationship is based on the Beer-Lambert Law, which accounts for the different molar extinction coefficients of oxy- and deoxyhemoglobin at these wavelengths, as well as the optical path through the tissues [25-27]. Methemoglobin absorbs both wavelengths of light nearly equally, which confounds pulse oximeter calculations that rely only on oxyhemoglobin and deoxyhemoglobin [28-30].

In contrast, the Arterial Blood Gas (ABG) PaO_2 is generally normal or elevated because PaO_2 reflects dissolved oxygen rather than hemoglobin function [31-33]. Consequently, the presence of low Oxygen Saturation (SpO_2) along with normal or elevated PaO_2 should prompt the evaluation of potential hemoglobin alterations such as methemoglobinemia. However, neither ABG nor standard pulse oximetry can be used to quantify methemoglobinemia. In such instances, co-oximetry serves as the gold standard for diagnosis, because its multi-wavelength detection apparatus can directly quantify the levels of oxyhemoglobin, deoxyhemoglobin, carboxyhemoglobin, and methemoglobin [30,33,34]. To ensure a definitive diagnosis, co-oximetry should be performed promptly when methemoglobinemia is suspected. Co-oximetry measurements of methemoglobin are inherently constrained by the linearity limitations of the instrument's detection range, which can significantly affect the accuracy of the readings at extreme methemoglobin concentrations. Typically, co-oximeters are calibrated to measure physiologically and moderately elevated methemoglobin levels in clinical settings. However, when confronted with exceptionally high methemoglobin concentrations, such as those resulting from severe sodium nitrite poisoning, the reliability of these measurements reduces [35]. This is primarily because the absorbance signals generated at high methemoglobin levels potentially exceed the linear dynamic range of device sensors, leading to signal saturation or nonlinear responses. Consequently, the instrument may underestimate the true methemoglobin concentration, produce plateau readings that do not reflect further increases in methemoglobinemia, or fail to yield a value [36]. These limitations pose a critical challenge in cases of acute poisoning, where precise quantification is essential for accurate diagnosis and timely intervention. Furthermore, the inability to accurately analyze samples with extremely elevated methemoglobin levels can hinder the effective monitoring of treatment efficacy and patient recovery, underscoring the need for cautious interpretation of co-oximetry results in such cases.

Characteristic bedside findings can further support the diagnosis of methemoglobinemia in these patients. Blood with a "chocolate-brown" or slate-colored appearance that does not brighten when exposed to air is a classic vital clue. In addition to methemoglobinemia, sodium nitrite can cause hypotension (via nitric oxide) and hemolysis [22]. The rapid detection of elevated methemoglobin levels is crucial for timely and effective treatment. Symptoms and clinical presentations are associated with the percentage of methemoglobin in the blood compared with that of normal oxyhemoglobin. In patients with methemoglobin levels between 20% and 50%, the symptoms include dizziness, syncope, dyspnea, exercise intolerance, fatigue, headache, and weakness. When the levels rise above 50%, cardiac dysrhythmia, central nervous system depression, metabolic acidosis, tachypnea, seizures, and coma are often observed [37]. Blood levels of >70% can rapidly become fatal without prompt intervention [38,39].

Management strategies

The primary treatment for clinically significant methemoglobinemia is methylene blue, which acts as an artificial electron carrier for the nicotinamide adenine dinucleotide phosphate-dependent methemoglobin reductase pathway, restoring Ferric (Fe^{3+}) hemoglobin to its functional Ferrous (Fe^{2+}) state [37,40]. Early recognition and prompt administration of methylene blue are critical for preventing tissue hypoxia and end-organ injury, particularly in high-dose exposures, which are often observed in intentional sodium nitrite ingestion [20,23]. The initial dose of methylene blue is 1−2 mg/kg infused intravenously over 3−5 min. Responses are typically observed rapidly, with a full clinical response expected within 30−60 min. If symptoms persist or methemoglobin levels remain elevated, an additional dose of 1 mg/kg IV may be administered. The cumulative daily dose should not exceed 7 mg/kg because higher doses can paradoxically induce methemoglobinemia and hemolysis [3,41]. Co-oximetry testing should be repeated to confirm the therapeutic response and ensure the absence of rebound methemoglobinemia [32].

Alternative therapies for methemoglobinemia include exchange transfusion, hyperbaric oxygen therapy, and ascorbic acid, each of which has specific limitations compared with methylene blue. Exchange transfusion effectively removes methemoglobin-containing red blood cells, replacing them with normal hemoglobin, and has been used successfully in cases where methylene blue is contraindicated or ineffective [42,43]. Hyperbaric oxygen therapy, applied as a monotherapy or adjunctive treatment, can improve outcomes but may have delayed effects on methemoglobin reduction and is limited by availability and impracticality in emergency settings [42-44]. Ascorbic acid is a slower-acting alternative, particularly for sodium nitrite poisoning, often requiring multiple doses, and is typically used alongside other treatments when methylene blue cannot be administered (patients with Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency, etc) [45-47].

Other agents, such as riboflavin, have demonstrated partial efficacy in vitro but lack substantial clinical evidence, whereas N-acetylcysteine has been shown to be ineffective for sodium nitrite-induced methemoglobinemia [3,48]. In severe refractory cases involving hypoxemia and cardiovascular collapse, venovenous extracorporeal membrane oxygenation may provide temporary tissue oxygenation support until definitive therapy can be administered [49]. These alternatives are especially relevant when methylene blue is contraindicated, such as in patients with G6PD deficiency or when it is unavailable.

Conclusions

The increasing incidence of intentional ingestion of sodium nitrite for self-harm is a significant and increasing public health concern. This case highlights the critical importance of early recognition of methemoglobinemia, particularly in patients presenting with refractory hypoxia, cyanosis, discordant pulse oximetry, and arterial blood gas results. The widespread availability of sodium nitrite and its promotion in online forums have contributed to increased exposure and severe poisoning cases. Rapid diagnosis supported by clinical suspicion and co-oximetry, combined with the prompt administration of methylene blue, can effectively reverse severe methemoglobinemia and prevent fatal complications. Emergency and critical care clinicians should maintain a high index of suspicion for sodium nitrite poisoning in an appropriate clinical context to ensure timely intervention, improve patient survival, and reduce morbidity.

References

1. Mansouri A, Lurie AA. Methemoglobinemia. Am J Hematol. 1993; 42: 7-12.

2. Loscalzo J, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL. Harrison's Principles of Internal Medicine. McGraw Hill. 2022.

3. Cefalu JN, Joshi TV, Spalitta MJ, Kadi CJ, Diaz JH, Eskander JP, et al. Methemoglobinemia in the Operating Room and Intensive Care Unit: Early Recognition, Pathophysiology, and Management. Adv Ther. 2020;37: 1714-1723.

4. Hickey TBM, MacNeil JA, Hansmeyer C, Pickup MJ. Fatal methemoglobinemia: A case series highlighting a new trend in intentional sodium nitrite or sodium nitrate ingestion as a method of suicide. Forensic Sci Int. 2021: 326.

5. Khan H, Barber C, Azrael D. Suicide by sodium nitrite poisoning: Findings from the National Violent Death Reporting System, 2018-2020. Suicide Life Threat Behav. 2024; 54: 310-316.

6. McCann SD, Tweet MS, Wahl MS. Rising incidence and high mortality in intentional sodium nitrite exposures reported to US poison centers. Clin Toxicol (Phila). 2021; 59: 1264-1269.

7. Bloom J, Sharpe A, Nulman S, Monday K, Marraffa JM, Stayton C, et al. Comparing Confirmed Sodium Nitrite Suicide Deaths With Poison Center Surveillance Estimates. JAMA Netw Open. 2024; 7: e2434192.

8. Mack KA, Kaczkowski W, Sumner S, Law R, Wolkin A. Special Report from the CDC: Suicide rates, sodium nitrite-related suicides, and online content, United States. J Safety Res. 2024; 89: 361-368.

9. Loiseau M, Matheux A, Sabini S, Cavard S, Advenier AS, Pasquet A, et al. Suicide of an adolescent girl with sodium nitrite ordered on the internet. J Forensic Sci. 2023; 68: 2200-2204.

10. Mörch CM, Côté LP, Corthésy-Blondin L, Plourde-Léveillé L, Dargis L, Mishara BL. The Darknet and suicide. J Affect Disord. 2018; 241: 127-132.

11. Kelsall NC, Gimbrone C, Olfson M, Gould MS, Shaman J, Keyes K. Association Between Prosuicide Website Searches Through Google and Suicide Death in the United States From 2010 to 2021: Lagged Time-Series Analysis. J Med Internet Res. 2024: 26.

12. Padovano M, Aromatario M, D'Errico S, Concato M, Manetti F, David MC, et al. Sodium Nitrite Intoxication and Death: Summarizing Evidence to Facilitate Diagnosis. Int J Environ Res Public Health. 2022: 19.

13. Leavesley HB, Li L, Mukhopadhyay S, Borowitz JL, Isom GE. Nitrite-mediated antagonism of cyanide inhibition of cytochrome c oxidase in dopamine neurons. Toxicol Sci. 2010; 115: 569-576.

14. Sindelar JJ, Milkowski AL. Human safety controversies surrounding nitrate and nitrite in the diet. Nitric Oxide. 2012; 26: 259-266.

15. Hunault CC, van Velzen AG, Sips AJAM, Schothorst RC, Meulenbelt J. Bioavailability of sodium nitrite from an aqueous solution in healthy adults. Toxicol Lett. 2009; 190: 48-53.

16. Bockman O, Grandil T, Alonzo M. Poisons Information Monograph G016: Nitrates and Nitrites. IPCS INCHEM. 1996.

17. Castiglione N, Rinaldo S, Giardina G, Stelitano V, Cutruzzolà F. Nitrite and nitrite reductases: from molecular mechanisms to significance in human health and disease. Antioxid Redox Signal. 2012; 17: 684-716.

18. Desrosiers NA, Chow BLC. Impact of methemoglobin on carboxyhemoglobin saturation measurement in fatal sodium nitrate and sodium nitrite cases. J Anal Toxicol. 2023; 47: 750-752.

19. Chowdhary S, Bukoye B, Bhansali AM, Carbo AR, Adra M, Barnett S, et al. Risk of Topical Anesthetic-Induced Methemoglobinemia: A 10-Year Retrospective Case-Control Study. JAMA Intern Med. 2013; 173: 771-776.

20. Skold A, Cosco DL, Klein R. Methemoglobinemia: pathogenesis, diagnosis, and management. South Med J. 2011; 104: 757-761.

21. Yu G, Li Y, Cui S, Jian T, Kan B, Jian X. Two cases of methaemoglobinaemia and haemolysis due to poisoning after skin absorption of 4-chloro-1-nitrobenzene. Clin Toxicol (Phila). 2022; 60: 970-973.

22. Pinheiro S, Carvalho F, Carmo H. Self-poisoning by sodium nitrite ingestion: Investigating toxicological mechanisms in vitro. Toxicol Lett. 2025; 409: 152-162.

23. Ash-Bernal R, Wise R, Wright SM. Acquired methemoglobinemia: a retrospective series of 138 cases at 2 teaching hospitals. Medicine (Baltimore). 2004; 83: 265-273.

24. Batton R, Villard S, Popoff B. Methemoglobinemia. Rev Med Interne. 2024; 45: 479-487.

25. Blaney G, Sassaroli A, Fantini S. Critical analysis of the relationship between arterial saturation and the ratio-of-ratios used in pulse oximetry. J Biomed Opt. 2024; 29.

26. Pulse oximetry: theory and applications for noninvasive monitoring. PubMed.

27. Mannheimer PD. The light-tissue interaction of pulse oximetry. Anesth Analg. 2007; 105.

28. Sinex JE. Pulse oximetry: principles and limitations. Am J Emerg Med. 1999; 17: 59-66.

29. Absorption spectra of human fetal and adult oxyhemoglobin, deoxyhemoglobin, carboxyhemoglobin, and methemoglobin. PubMed.

30. Haymond S, Cariappa R, Eby CS, Scott MG. Laboratory assessment of oxygenation in methemoglobinemia. Clin Chem. 2005; 51: 434-444.

31. Barker SJ, Tremper KK, Hyatt J. Effects of methemoglobinemia on pulse oximetry and mixed venous oximetry. Anesthesiology. 1989; 70: 112-117.

32. Wright RO, Lewander WJ, Woolf AD. Methemoglobinemia: Etiology, pharmacology, and clinical management. Ann Emerg Med. 1999; 34: 646-656.

33. Tintinalli JE, Stapczynski JS, Ma OJ, Yealy DM, Meckler GD, Cline DM. Tintinalli's Emergency Medicine: A Comprehensive Study Guide. McGraw-Hill Education. 2020.

34. Saeui C, Charlton N, Brady WJ. Biochemical issues in emergency medicine: diagnostic and therapeutic considerations of selected toxic presentations. Am J Emerg Med. 2012; 30: 231-235.

35. Hulse E, Shihana F, Buckley NA. Methemoglobin measurements are underestimated by the Radical 7 co-oximeter: experience from a series of moderate to severe propanil poisonings. Clin Toxicol (Phila). 2016; 54: 826-828.

36. Rausch-Madison S, Mohsenifar Z. Methodologic problems encountered with cooximetry in methemoglobinemia. Am J Med Sci. 1997; 314: 203-206.

37. Hoffman RS, Howland MA, Lewin NA, Nelson LS, Goldfrank LR, Smith SW. Goldfrank's Toxicologic Emergencies. McGraw-Hill. 2019.

38. Cruz M, Dela Glick J, Merker SH, Vearrier D. Survival after severe methemoglobinemia secondary to sodium nitrate ingestion. Toxicol Commun. 2018; 2: 21-23.

39. Mun SH, Park GJ, Lee JH, Kim YM, Chai HS, Kim SC. Two cases of fatal methemoglobinemia caused by self-poisoning with sodium nitrite: A case report. Medicine (Baltimore). 2022; 101: E28810.

40. Pushparajah Mak RS, Liebelt EL. Methylene Blue: An Antidote for Methemoglobinemia and beyond. Pediatr Emerg Care. 2021; 37: 474-477.

41. Qutob RA. Methylene blue in the intensive care unit: A comprehensive review article. Anaesthesia Pain Intensive Care. 2025; 29: 407-417.

42. Cao D, Arens AM, Chow SL, Easter SR, Hoffman RS, Lagina AT, et al. Part 10: Adult and Pediatric Special Circumstances of Resuscitation: 2025 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2025; 152: S578-S672.

43. Lavonas EJ, Akpunonu PD, Arens AM, Babu KM, Cao D, Hoffman RS, et al. 2023 American Heart Association Focused Update on the Management of Patients With Cardiac Arrest or Life-Threatening Toxicity Due to Poisoning. Circulation. 2023; 148: E149-E184.

44. Cho Y, Park SW, Han SK, Kim HB, Yeom SR. A Case of Methemoglobinemia Successfully Treated with Hyperbaric Oxygenation Monotherapy. J Emerg Med. 2017; 53: 685-687.

45. Dhibar DP, Sahu KK, Jain S, Kumari S, Varma SC. Methemoglobinemia in a Case of Paint Thinner Intoxication, Treated Successfully with Vitamin C. J Emerg Med. 2018; 54: 221-224.

46. Rino PB, Scolnik D, Fustiñana A, Mitelpunkt A, Glatstein M. Ascorbic acid for the treatment of methemoglobinemia: the experience of a large tertiary care pediatric hospital. Am J Ther. 2014; 21: 240-243.

47. Maio E, Volpi C, Abdalmahmoud M, Dafalla YMA, Hamed AM, Abdalla SHA, et al. Sodium Nitrite-Induced Methemoglobinemia in A Conflict Area: A Case Series from Sudan and Implications for Humanitarian Response. J Emerg Med. 2025; 77: 93-97.

48. Dötsch J, Demirakça S, Kratz M, Repp R, Knerr I, Rascher W. Comparison of methylene blue, riboflavin, and N-acetylcysteine for the reduction of nitric oxide-induced methemoglobinemia. Crit Care Med. 2000; 28: 958-961.

49. Lien YH, Lin YC, Chen RJ. A case report of acquired methemoglobinemia rescued by veno-venous extracorporeal membrane oxygenation. Medicine (United States). 2021; 100: E25522.

Manuscript Information: Received: May 09, 2026; Accepted: June 11, 2026; Published: June 30, 2026

Authors Information: Christopher Lai Hipp, Pharm D*; Nishal Brahmbhatt, MD; Robin Inaba, MD
The Queen's Medical Center, Honolulu, Hawaii, USA.

Citation: Lai Hipp C, Brahmbhatt N, Inaba R. Successful reversal of severe methemoglobinemia caused by intentional sodium nitrite ingestion: A case report and mini-review. Open J Clin Med Case Rep. 2026; 2422.