



doi: 10.4103/jad.jad_192_25

Nitrite food poisoning from cold pork skin jelly: A report of two cases

Yuru Liu^{1,2}, Qi Zhang¹, Guiping Wang¹, Xiangdong Jian^{2✉}, Baotian Kan^{1,3✉}¹School of Nursing and Rehabilitation, Cheeloo College of Medicine, Shandong University, Jinan, China²Department of Poisoning and Occupational Diseases, Emergency Medicine, Qilu Hospital of Shandong University, Cheeloo College of Medicine, Shandong University, Jinan, China³Department of Nursing, Qilu Hospital, Cheeloo College of Medicine, Shandong University, Jinan, China

ABSTRACT

Rationale: Nitrite, a common meat preservative, can cause life-threatening methemoglobinemia.

Patient's concern: Two men (52 and 60 years) developed sudden cyanosis, dizziness, and nausea 10–20 minutes after consuming pork skin jelly from a local deli.

Diagnosis: Acquired methemoglobinemia (methemoglobin 50.7% and 37.2%); jelly contained 420 mg/kg nitrite (GB 2760-2014 limit: 30 mg/kg).

Interventions: High-flow oxygen and intravenous 1% methylene blue 1–2 mg/kg in 20 mL 5% glucose over 15 minutes.

Outcomes: Cyanosis was relieved within 30 minutes; methemoglobin became normalized within 2 hours. Both recovered fully and were discharged on June 24, 2024.

Lessons: Sudden cyanosis after meat consumption requires immediate hospitalization, methemoglobin testing, and methylene blue treatment; food residue analysis confirms diagnosis, while supplier monitoring and record-keeping prevent recurrence.

KEYWORDS: Nitrite; Food poisoning; Pork rind jelly; Methemoglobinemia

1. Introduction

Nitrite poisoning can cause symptoms such as palpitations, dizziness, headaches, general discomfort, cyanosis of the lips and nail beds, cold extremities, and, in some cases, it may lead to

methemoglobinemia[1]. Nitrite is a white or pale yellow crystalline industrial salt with a slightly salty taste and no odor. It has strong oxidizing properties and is found in fresh pickled vegetables, cooked vegetables that have been stored for too long, and rotten or spoiled vegetables. Ingestion of 0.3–0.5 g can cause poisoning, and 3 g can be fatal[2]. In China, nitrite poisoning is primarily caused by the accidental ingestion or misuse of foods containing excessive levels of nitrites. Symptoms typically manifest within 1–3 h after ingestion, with the shortest onset time being 10–15 min and the longest being 20 h. Nitrite poisoning is characterized by rapid onset, rapid progression, and severe symptoms. Timely diagnosis and treatment are critical for patient recovery. Nitrites oxidize ferrous iron ions in hemoglobin to ferric iron ions, preventing methemoglobin from binding oxygen and altering the affinity of other hemoglobin for oxygen, thereby impairing oxygen delivery to tissues. Under normal conditions, the methemoglobin levels are regulated to <3%. When this regulatory mechanism is disrupted, it can lead to clinically significant methemoglobinemia[3]. Two patients admitted to our department experienced dizziness and nausea after consuming

✉To whom correspondence may be addressed. E-mail: Jian XD, jianxiangdongvip@vip.163.com; Kan BT, kanbaotian@163.com.

This is an open access article distributed under the terms of the Creative Commons Attribution-Non-Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal

For reprints contact: reprints@medknow.com

©2026 Journal of Acute Disease Produced by Wolters Kluwer-Medknow.

How to cite this article: Liu Y, Zhang Q, Wang G, Jian X, Kan B. Nitrite food poisoning from cold pork skin jelly: A report of two cases. J Acute Dis 2026; 15: 6.

Article history: Received 20 September 2025; Revision 21 November 2025; Accepted 23 March 2026; Available online 28 March 2026

cold pork skin jelly. Arterial blood gas analysis revealed that their methemoglobin levels exceeded the normal range. We analyzed the clinical manifestations and treatment of these food poisoning cases to enhance residents' understanding of nitrite food poisoning, improve preventive awareness, and prevent further occurrences of nitrite poisoning. This study was approved by the Ethics Committee of Qilu Hospital, Shandong University, and written informed consent was obtained from both patients, who signed the Case Report Publication Consent Form.

2. Case report

Patient 1 was a 60-year-old male. On the evening of June 20, 2024, around 7 p.m., the patient purchased a food item consisting of pig skin jelly and bean sprouts from a deli in a community in Huaiyin District, Jinan City. Approximately 10 min later, the patient experienced symptoms such as nausea, convulsions, and cyanosis and was admitted to a local hospital. The patient reported dizziness, nausea, and significant discomfort. On the morning of June 21, 2024, the patient was transferred to our hospital. On admission, cyanosis was observed in the extremities, earlobes, lips, and oral cavity. The body temperature was 36.5°C, the heart rate was 91 beats per min, the respiratory rate was 16 breaths per min, the blood pressure was 127/78 mmHg, and the oxygen saturation was 93%. Blood gas analysis showed methemoglobin (MetHb) at 50.7% (normal range: 0.0%–1.5%). A preliminary diagnosis of nitrite poisoning was made. After completing the relevant examinations, the patient was administered oxygen, organ protection, toxin excretion promotion, and nutritional support. A 1% methylene blue solution was given intravenously at 1–2 mg/kg (0.1–0.2 mL/kg) dissolved in 20 mL of 5% glucose and infused slowly over 15 min; the infusion could be repeated at half dose if MetHb remained >10% after 1 h. Thirty minutes after methylene blue infusion, the cyanosis of the patient's lips and extremities was significantly relieved, the facial and nail bed cyanosis gradually faded, and the oxygen saturation rose to 97%. On the second day, cyanosis of the extremities, lips, and ears improved compared with that on admission. Toxicological testing showed a caffeine concentration of 0.2 μ g/mL and a nitrite concentration of 0.1 μ g/mL in the urine. Repeated blood gas analysis revealed that the methemoglobin levels had returned to normal.

Patient 2 was a 52-year-old male. On June 20, 2024, the patient consumed a mixture of pig skin jelly and bean sprouts purchased from the same location at approximately 8:00 p.m. Approximately 20 min later, he developed symptoms of nausea and vomiting and was admitted to a local hospital before being transferred to our department. The patient reported feeling nauseous and vomiting.

On admission, cyanosis was observed in the extremities, earlobes, lips, and oral cavity. Vital signs on admission were as follows: temperature, 36.3°C; heart rate, 90 beats per min; respiratory rate, 14 breaths per min; and blood pressure, 120/67 mmHg. Blood gas analysis showed methemoglobin levels of 37.2% (normal range: 0.0%–1.5%). A preliminary diagnosis of nitrite poisoning was made. After completing relevant examinations, comprehensive treatment was administered, including oxygen therapy, organ protection, toxin excretion promotion, and nutritional support. The same methylene blue regimen (1–2 mg/kg) was administered as described for Patient 1. Thirty minutes after methylene blue infusion, the cyanosis of the patient's lips and extremities was significantly alleviated, and the oxygen saturation increased from 95% to 98%. On the second day, cyanosis in the patient's limbs, lips, and earlobes had improved compared with that on admission, and no other symptoms of discomfort were present. Toxicological testing revealed caffeine (0.2 μ g/mL) and nitrite (0.1 μ g/mL) in the urine sample. Repeated blood gas analysis revealed that the methemoglobin levels had returned to normal.

Both patients were discharged on June 24, 2024. At discharge, the patients had no cyanosis of the skin or lips, normal blood gas analysis, and normal laboratory test results, dynamic changes in laboratory findings are shown in Table 1. They were informed of the need for regular follow-up visits. Because both patients purchased the same food from the same store, as shown in figure 1, the cases were reported to the disease control center, and the patients' pork skin jelly was also sent to the disease control center for testing. The local disease control center collected samples of the food consumed by the patients and tested all food samples using the second method (spectrophotometric method) specified in GB 5009.33—2016 “National Food Safety Standard: Determination of Nitrites and Nitrates in Food”[4]. The nitrite content in the pork skin jelly was 420 mg/kg (reported as NaNO₂), far exceeding the 30 mg/kg maximum residue limit for cooked meat products set by GB 2760-2014. This finding, combined with the patients' clear exposure history and typical clinical manifestations, confirmed the diagnosis of nitrite food poisoning.

3. Discussion

Nitrites are salts containing highly toxic nitrite ions. Ingesting 0.3–0.5 g of nitrites can cause poisoning, with clinical symptoms including cyanosis, hypoxia, impaired consciousness, arrhythmia, and death[5]. They are commonly used as food colorants, preservatives, and antimicrobial agents in meat, fish, and certain cheeses[6]. Nitrites react chemically with myoglobin and hemoglobin to form nitrosomyoglobin during the curing process

Table 1. Dynamic changes in arterial blood oxygen partial pressure and methemoglobin in patients, %.

Parameter	Patient 1	Patient 2
Blood oxygen saturation		
On admission	93	95
2 h after admission	99	98
At discharge	99	99
Methemoglobin		
On admission	50.7	37.2
2 h after admission	5.3	3.2
At discharge	0.6	0.8

of meat, giving the meat a bright red color and thereby improving the sensory quality of meat products. Nitrite plays a crucial role in inhibiting *Clostridium botulinum* growth and toxin production. Nitrite generates nitrous acid under slightly acidic conditions, which decomposes to release oxygen, transforming the anaerobic environment into an aerobic condition unfavorable for the growth of *Clostridium botulinum*, thereby inhibiting its growth, reproduction, and production of botulinum toxin[7]. In this case, the nitrite content in the pork skin jelly exceeded the limit because the shop did not add nitrite in accordance with the specified dosage. The EU legislation specifies the usage levels of nitrite and nitrate in meat products, and the US Department of Agriculture (USDA) has clear regulations aimed at achieving technical objectives while safeguarding public health[8]. China has also established relevant national food safety standards that specify the nitrite dosage to ensure food safety[9].

Nitrite poisoning is a common cause of acquired methemoglobinemia. Nitrate contains three oxygen atoms and is stable; it only oxidizes hemoglobin to methemoglobin after intestinal bacteria reduce nitrate to nitrite. Nitrites contain two oxygen atoms and can directly oxidize hemoglobin. Methemoglobinemia refers to the oxidation of ferrous iron ions (Fe^{2+}) in hemoglobin molecules to ferric iron ions (Fe^{3+}). This reaction impairs hemoglobin's ability to transport oxygen and carbon dioxide, leading to tissue hypoxia, which can be fatal in severe cases. Clinical manifestations primarily depend on the methemoglobin concentration, which reflects the remaining oxygen-carrying capacity[10]. Under normal physiological conditions, the methemoglobin concentration does not exceed 0.0%–1.5%[11]. When methemoglobin levels reach 30%–40%, symptoms such as headache, fatigue, palpitations, weakness, and dizziness may occur. Methemoglobin concentrations as high as 60% can cause drowsiness, seizures, and coma, while methemoglobinemia concentrations exceeding 70% are typically fatal[12]. Both patients had methemoglobinemia concentrations exceeding 30% and exhibited cyanosis and nausea. Treatment options for severe methemoglobinemia are limited and primarily

include active detoxification measures, hyperbaric oxygen therapy, and the use of reducing agents[13]. Oxygen therapy is an important component of nitrite poisoning treatment. Methemoglobinemia caused by nitrite poisoning impairs the blood's ability to carry oxygen. Oxygen therapy can increase oxygen supply through high-flow oxygen (5–10 L/min) or mask oxygenation, thereby helping to correct hypoxemia and improve tissue oxygenation. Oxygen therapy can significantly alleviate symptoms such as dyspnea in severely hypoxic patients. Methylene blue is a thiazole dye with antimicrobial and dose-dependent redox properties that serves as the first-line antidote for patients with severe methemoglobinemia[14]. Typically, a 1% methylene blue solution is administered via intravenous infusion at a dose of 1–2 mg/kg body weight dissolved in 20 mL of 5% glucose solution. At low doses (1–2 mg/kg), methylene blue is reduced to reduced methylene blue in the body, transferring hydrogen ions to methemoglobin and promoting its conversion to normal hemoglobin. Cyanosis typically improves rapidly, and methemoglobin levels usually return to normal within 30–60 min after methylene blue injection. Methylene blue, the first-line antidote for symptomatic methemoglobinemia, was given at 1–2 mg/kg (0.1–0.2 mL/kg of 1% solution) IV over 15 min; MetHb normalized within 2 h without need for repeat dosing. Combined with the patients' symptoms, the results indicated that the treatment was effective; therefore, rapid diagnosis and early intervention with methylene blue infusion can prevent fatal outcomes. Additionally, blood purification is an effective method to promote toxin excretion in patients with severe poisoning and concomitant multi-organ dysfunction. Both patients reported relief of discomfort after oxygen therapy and methylene blue injection. Two hours post-treatment, the blood gas analysis results were normal, and no further hemodialysis was required.

Of note, both patients presented with markedly elevated blood MetHb (50.7% and 37.2%), yet urinary nitrite was only $0.1 \mu\text{g/mL}$. This apparent discrepancy can be explained by the rapid toxicokinetics of nitrite: its plasma half-life is short (≈ 1.3 – 1.8 h), and it is promptly excreted by the kidneys and

reduced by erythrocytes and vascular endothelium. Because blood samples were drawn 12–14 h after ingestion, circulating nitrite had already been largely eliminated, whereas MetHb, once formed, persists until re-reduced. Additionally, the urine specimens were collected at first micturition after hospital arrival (2–4 h later), further diluting residual nitrite. Therefore, a low or even negative urinary nitrite does not rule out acute severe nitrite-induced methaemoglobinaemia; blood MetHb remains the diagnostic gold standard. To prevent nitrite food poisoning, in addition to strengthening regulatory measures, targeted health education should be carried out for residents. Residents should avoid purchasing homemade meat products from unlicensed delis, and check food labels and qualification certificates when buying processed meat products. For homemade pickled foods, the pickling time should be strictly controlled (at least 2 weeks, until the nitrite content drops to a safe level), and excessive addition of salt and nitrite-containing additives should be avoided. The finished products should not be stored at room temperature for a long time, but should be refrigerated and consumed as soon as possible. If symptoms such as lip cyanosis, dizziness, and nausea occur after eating meat products, immediate emetic treatment and medical attention are required, and the remaining food should be retained for subsequent testing to clarify the cause of poisoning.

4. Conclusion

We recommend that local authorities should require cooked-meat workshops to maintain a dual logbook system that records every purchase and daily use of nitrite under real-name registration, with regular inspections by the market-supervision bureau; health departments should distribute free colorimetric test strips (LOD 10 mg/kg) to vendors for daily self-testing, the results of which must be posted in-store; and the commerce bureau should set up a QR-code-based traceability platform that documents the supplier, buyer and quantity of each nitrite transaction, enabling full-chain surveillance from source to sink. In addition, public health education should be strengthened to raise residents' awareness of safe food consumption, which is critical to reducing the incidence of nitrite food poisoning.

Conflict of interest statement

The authors report no conflict of interest.

Ethical statement

This study was approved by the Institutional Review Board of Shandong University (approval number: IRB-SPH-SDU-20250371). All participants provided informed consent and participated voluntarily.

Data availability statement

The data supporting the findings of this study are available from the corresponding authors upon request.

Funding

This study received no extramural funding.

Authors' contributions

LYR: data collection, manuscript drafting. ZQ & WGP: clinical data analysis. JXD case diagnosis and treatment. KBT: study design, manuscript review and editing.

Publisher's note

The Publisher of the *Journal* remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Edited by Liu JJ

References

- [1] Patel P, Konala VM, Adapa S, Gayam V, Sahasranam P, Bose S, et al. Case report of lethal toxin lurking in an edible plant. *J Popul Ther Clin Pharmacol* 2019; **26**(3): e14-e18.
- [2] Wang R, Teng CG, Zhang N, Zhang J, Conway G. A family cluster of nitrite poisoning, Suzhou City, Jiangsu Province, China, 2013. *West Pac Surveill Response J* 2013; **4**(3): 33–36.
- [3] O'Neill C, Najjar Z, Ingleton A, Edwards A, Dawson A, Gupta L. Methaemoglobinaemia associated with the atypical use of sodium nitrite as a food additive. *Med J Aust* 2021; **215**(6): 256-257.
- [4] GB 5009.33-2016 National Food Safety Standard - Determination of Nitrites and Nitrates in Foods. Beijing: Standards Press of China, 2016.
- [5] Neth MR, Love JS, Horowitz BZ, Shertz MD, Sahni R, Daya MR. Fatal sodium nitrite poisoning: key considerations for prehospital providers. *Prehosp Emerg Care* 2021; **25**(6): 844-850.
- [6] Cvetković D, Živković V, Lukić V, Nikolić S. Sodium nitrite food poisoning in one family. *Forensic Sci Med Pathol* 2019; **15**(1): 102-105.
- [7] Pierson MD, Smoot LA. Nitrite, nitrite alternatives, and the control of *Clostridium botulinum* in cured meats. *Crit Rev Food Sci Nutr* 1982; **17**(2): 141-187.
- [8] Ferysiuk K, Wójciak KM. Reduction of nitrite in meat products through the application of various plant-based ingredients. *Antioxidants* 2020; **9**(8): 711.
- [9] GB 2760-2014 National Food Safety Standard: Standard for Uses of Food Additives. Beijing: Standards Press of China, 2014.
- [10] Chui JS, Poon WT, Chan KC, Chan AY, Buckley TA. Nitrite-induced methaemoglobinaemia – aetiology, diagnosis and treatment. *Anaesthesia* 2005; **60**(5): 496-500.
- [11] Stambach T, Haire K, Soni N, Booth J. Saturday night blue – a case of near fatal poisoning from the abuse of amyl nitrite. *J Accid Emerg Med* 1997; **14**(5): 339-340.
- [12] Katabami K, Hayakawa M, Gando S. Severe methemoglobinemia due to sodium nitrite poisoning. *Case Rep Emerg Med* 2016; **2016**: 9013816.
- [13] Harvey M, Cave G, Chanwai G. Fatal methaemoglobinaemia induced by self-poisoning with sodium nitrite. *Emerg Med Australas* 2010; **22**(5): 463-465.
- [14] Umbreit J. Methemoglobin—it's not just blue: a concise review. *Am J Hematol* 2007; **82**(2): 134-144.