Forensic Science, Medicine and Pathology (2019) 15:102–105
https://doi.org/10.1007/s12024-018-0036-1

CASE REPORT

Sodium nitrite food poisoning in one family

Danica Cvetković[1] · Vladimir Živković[1] · Vera Lukić[1] · Slobodan Nikolić[1]

Accepted: 24 September 2018 / Published online: 6 October 2018
© Springer Science+Business Media, LLC, part of Springer Nature 2018

Abstract

Sodium nitrite is used as a coloring agent or preservative in food, as well as an antimicrobial agent in meat and fish and some cheeses. In high amounts it can be toxic for humans, causing methemoglobinemia. This is an unusual and potentially fatal condition in which hemoglobin is oxidized to methemoglobin (MHb), reducing the amount of oxygen that is released from hemoglobin, similar to carbon monoxide poisoning. MHb levels of 70% are generally lethal, but the existence of underlying anemia, acidosis, respiratory compromise, and cardiac disease may exacerbate the toxicity of MHb. We present a case of poisoning with sodium nitrite in three family members after eating homemade sausages given to them by their neighbor who was a butcher. According to the findings of the veterinary inspectorate in charge of food control in this case, the concentration of sodium nitrite in the homemade sausages was about 3.5 g per 1 kg of meat, almost 30 times higher than allowed according to legislation. In this case report, a 70-year-old man died about 7 h after consuming the meal, while two women, 53 and 67 years of age, respectively, were admitted to a toxicology clinic the following day due to food poisoning, with the maximum concentration of MHb in blood of 33.7 and 20.4%, respectively. They were discharged 3 days later. The autopsy of the deceased man showed sodium nitrite poisoning with a relatively low concentration of MHb in his blood – 9.87%. Death was attributed to the exacerbation of hypertensive and ischemic heart disease, resulting from accidental sodium nitrite poisoning. The presented cases illustrate the necessity of close cooperation between the authorities, medical staff, veterinary inspectorate, and forensic pathologists in determining the source of poisoning, the cause of death of the victim, and preventing the outbreak of poisoning among a greater number of consumers.

Keywords Sodium nitrite · Poisoning · Food poisoning · Accident · Methemoglobinemia

Introduction

Sodium nitrite is a slightly salty, white or yellow powder, which is like table salt in appearance and taste. It has been widely used in both industry and construction [1, 2]. Since the early 1900s, it has been used as a coloring agent or preservative in food, and as an antimicrobial agent in meat, as well as in fish and some cheeses. Sodium nitrite inhibits the growth of microorganisms, gives taste and red color to meat, and inhibits lipid oxidation that leads to rancidity [1–4]. In high amounts sodium nitrite can be toxic for humans. Within a period ranging from 20 min to 3 h after ingestion of 200 mg to 500 mg, signs of acute intoxication appear [2]. The estimated lethal dose of sodium nitrite in adults is approximately 2600 mg; however, a case of a patient surviving after ingesting 6000 mg of sodium nitrite has been reported [3, 5]. Nitrite can cause methemoglobinemia, which is an unusual and potentially fatal condition in which hemoglobin is oxidized to MHb, reducing the amount of oxygen that is released from hemoglobin [1–5]. Nitrite is also a potent vasodilator and can cause coronary ischemia and stroke as a result of hypotension, tachycardia, and hypoxia [3].

Methemoglobinemia refers to the oxidation of ferrous iron (Fe++) to ferric iron (Fe+++) within the hemoglobin molecule. This reaction impairs the ability of hemoglobin to transport oxygen and carbon dioxide, leading to tissue hypoxemia and, in severe cases, death. Additionally, due to allosteric changes to the hemoglobin molecule, there is an increased affinity for oxygen binding in partially oxidized hemoglobin molecules, causing a left shift of the hemoglobin-oxygen saturation curve (same as in carbon monoxide poisoning), i.e. aggravating cellular hypoxia [6]. The severity of the symptoms depends on the MHb level, and the levels are reported as a percentage of total hemoglobin. The concentration of MHb

✉ Slobodan Nikolić
bobanvladislav@yahoo.com; slobodan.nikolic@med.bg.ac.rs

[1] Institute of Forensic Medicine, University of Belgrade – School of Medicine, 31a Deligradska str, Belgrade 11000, Serbia

does not exceed 1%–2% in normal physiological state, while levels of 10%–20% generally cause cyanosis. As the levels reach 30%–40%, symptoms such as headache, fatigue, tachycardia, weakness, and dizziness are experienced. MHb levels of 60% produce lethargy, convulsions, and coma. MHb levels of 70% are generally lethal, although survival has been reported with a level of 94% [3, 5–7]. The existence of underlying anemia, acidosis, respiratory compromise, and cardiac disease may exacerbate the toxicity of MHb [6].

Case report

A neighbor, who was a butcher, brought some homemade sausages to two spouses (a 70-year-old man and 53-year old woman). They ate some of them for lunch around 13:00. Shortly after lunch, in the afternoon, they started feeling ill and both experienced headache, malaise, dizziness, walking instability, nausea, and vomiting. During the same evening the man experienced chest pain, and shortly after suddenly collapsed and died around 20:00. For several years prior to his death, he had been suffering from hypertension, ischemic cardiac disease, and asthma. Early the following morning, around 03:00, his 67-year-old sister also ate the sausages and, shortly after, experienced similar symptoms. Due to the described health issues, around 10:00 the two women were examined by a physician and admitted to the toxicology clinic for suspected food poisoning and accidental nitrite intoxication. Upon admission, the concentration of methemoglobin (MHb) in the 53-year-old woman's blood was 33.7% (about 21 h after eating the sausages). Seven hours later, after the treatment, the MHb concentration was 6.2% and the following day 1.8%. The other woman's (67 years old) blood showed a concentration of MHb of 20.4% upon admission (about 7 h after eating the sausages). Seven hours later, the MHb concentration was 1.4%, and the following day no MHb was detected. They were treated with oxygen therapy, solutions of glucose, electrolytes, ascorbic acid (vitamin C), and H2 receptor blockers. Both women were conscious and stable the entire time, and they were released the third day after their admission, in good condition with the final diagnosis of methemoglobinemia due to food poisoning with sodium nitrite.

The autopsy of the 70-year-old deceased man was performed about 36 h after death. The body was obese (175 cm in height, weighing 100 kg), and the most conspicuous finding at external examination was very well-developed postmortem hypostasis (*livores*), not only on the posterior parts of the body, but also on the face, anterior side of the neck, and upper and anterior side of the shoulders, with significant postmortem punctiform hemorrhages (*vibices*) in the region of hypostasis. The color of the postmortem hypostasis was a bit darker (dark bluish red) than usual, but it was not brown (Fig. 1).

Internal examination showed incipient postmortem putrefaction changes. However, the main finding was advanced

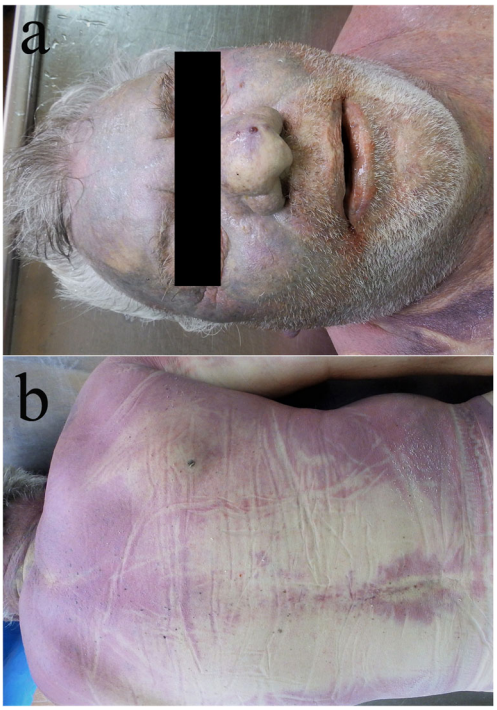

Fig. 1 Macroscopic appearance of the postmortem hypostasis – the color is dark bluish red, instead of brown, as it would be expected in sodium nitrite poisoning: **a** – face, and **b** – posterior aspect of the trunk

hypertensive and atherosclerotic heart disease (heart weighed 520 g, with dilatation; left ventricle thickness was up to 18 mm) (Fig. 2), with several small fibrous scars on the myocardium. Additional findings included lung edema, horseshoe kidney (*ren arcuatus*) and enlarged fatty liver. There was about 500 ml of partially digested, mushy content in the stomach. Besides early postmortem changes, microscopic examination showed leukostasis and margination of neutrophils in the myocardial vessels as signs of early ischemic myocardial lesion, atherosclerotic changes with calcifications in the coronary blood vessels, as well as fields of myocardial fibrosis. Other findings were consistent with hypertension and macroscopic findings.

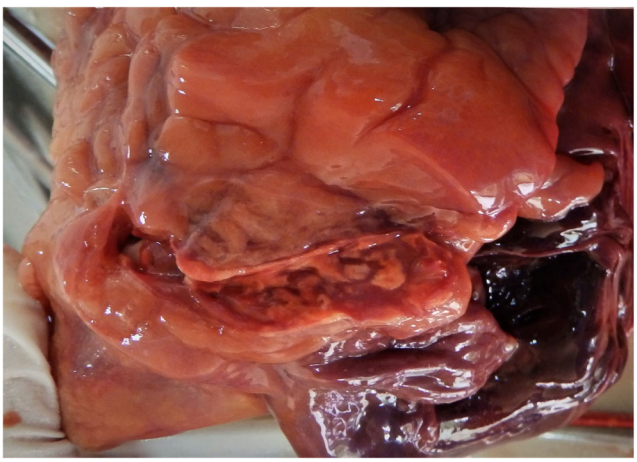

Fig. 2 Severe atherosclerotic changes in the right coronary artery

Toxicological analysis showed a relatively small degree of methemoglobinemia – 9.87% of MHb in the blood, with 24.6 ng/ml of nitrites in the blood, 93.91 mg/kg in the stomach contents, and 0.003 mg/kg in the liver and kidney mixture. Death was attributed to the exacerbation of hypertensive and ischemic heart disease, resulting from accidental sodium nitrite poisoning.

According to the findings of the veterinary inspectorate in charge of food control in this case, the concentration of sodium nitrite in the homemade sausages was about 3.5 g per 1 kg of meat, almost 30 times higher than allowed according to legislation.

Discussion

In 2002, the European Food Safety Authority (EFSA) indicated that 50-100 mg/kg of added nitrites (such as sodium nitrite) was sufficient to "control" microbiological hazards. To reduce the risk of overdose, the EFSA recommended the exclusive use of nitrite-based additives containing 50% of sodium chloride. A much lower quantity (1-14 mg/kg) is required for the pinking of meat. Contamination of food with toxic doses of nitrites is most usually accidental, and it may occur through negligence, inexperience or lack of caution, such as the use of nitrites in the preparation of salt substitutes or other ingredients that have similar appearance [8]. Heat used in cooking destroys nitrites, but this is not to be relied upon, since the amounts destroyed vary with the degree of heat and the length of cooking [9]. In our country, the sale of pure sodium nitrite is strictly controlled by state authorities; its free sale is prohibited, and it cannot be legally purchased for domestic use. Additionally, to avoid intoxication, the most common way of (controlled) sale of sodium nitrite is in the form of the previously prepared mixture of kitchen salt (sodium chloride) and sodium nitrite (marked as the food additive E250) for brining, in a fixed nitrite concentration of 0.5–0.6%. It is usually sold in 25 or 50 kg sacks marked with the warning "poison", with strict instructions for use: 2 to 2.5 kg of salt mixture per 100 kg of meat (which is a maximum of 15 mg of sodium nitrite per 1 kg of meat). The law allows the maximum concentration of 125 mg of sodium nitrite per 1 kg of meat in a final product. In the presented case, the accused butcher did not admit to the authorities how he obtained sodium chloride, claiming he used the industrially prepared mixture. Given that no other case of nitrite intoxication has been recorded in wider area before or since then, and that the concentration in the prepared sausages was almost 30 times higher than allowed, it can be assumed that the accused butcher illegally obtained sodium nitrite, mixed it with kitchen salt, miscalculating and adding too much nitrite to the mixture. Excepting the three presented victims, he did not distribute the sausages further.

Sodium nitrite intoxication due to food poisoning is not very common today. Industrial intoxication (sodium nitrite used in the antifreeze admixture) and drinking water from sodium nitrite-contaminated tanks and pipes (where sodium nitrite is used commercially to prevent corrosion of pipes) are more common causes [4]. The first described cases of fatal sodium nitrite intoxication due to food poisoning date back to 1936, when a family of two adults and a child died due to seasoning their dinner with sodium nitrite that was in the salt cellar (instead of kitchen salt) [10]. In 1945, an "outbreak" of sodium nitrite poisoning occurred in New York, when 11 people were poisoned after eating a meal in a local cafeteria: the oldest of them, an 82-year-old man, died [9]. In recent years, however, only occasional cases of (usually non-fatal) intoxication after accidental sodium nitrite food poisoning have been described [1, 2, 4, 5, 8]. One of them included the poisoning of five people on a ship, with one fatal outcome – a 69-year-old woman [1]. Interestingly, one of the cases included non-fatal sodium nitrite poisoning after taking a Chinese herbal medicine [5].

In addition to the fact that the signs and symptoms, as well as the outcomes of sodium nitrite poisoning are dose-dependent, it seems that older people are more susceptible to a fatal outcome (similar to carbon monoxide poisoning) as in our presented cases. In our three cases, the fatal outcome occurred in the oldest, a 70-year-old victim with the previously existing ischemic heart disease and a relatively low MHb concentration (less than 10%), while the other two survived with significantly higher (although still non-fatal) MHb concentrations (33.7 and 20.4%). His age and existing chronic ischemic heart disease made him susceptible to a relatively small rise in MHb levels, and thus even a small decrease in hemoglobin capacity for oxygen transfer resulted in myocardial ischemia and a fatal outcome. Additional mechanisms may also include the effect of nitrites in circulation as powerful vasodilators, which could have further compromised the supply of oxygen to the myocardium and worsened his heart condition, resembling the effect of sildenafil (Viagra) and similar medications on people with ischemic heart disease (also known as *coronary steal syndrome*).

Still, the outcome of sodium nitrite poisoning mostly depends on the timely recognition of symptoms and signs and adequate treatment [1–5, 8]. In the three cases presented, the fatal outcome in one victim had occurred before they sought medical help, within 7 h after the meal. In the following several hours, the two surviving victims received medical attention. Sodium nitrite poisoning was suspected, and they were transferred to the toxicology clinic the following morning. Since the autopsy of the deceased was performed the next day, the forensic pathologists had already been informed by the police about nitrite poisoning as the possible cause of death. That was particularly important due to the incipient postmortem putrefaction changes, and because external examination lacked one of the major textbook signs of nitrite poisoning – brownish color of the hypostasis, which would suggest to the pathologist to seek for nitrite poisoning as the cause of death. The lack of this sign can be attributed to a low concentration of MHb at the time of death.

The presented cases justify the strict and rigorous control of sodium nitrite turnover, since inappropriate preparation and usage of this substance present a severe public health hazard. These cases also illustrate the necessity of close cooperation between authorities, medical staff, veterinary inspection, and forensic pathologists in determining the source of poisoning, the cause of death in victims, and in preventing the outbreak of poisoning among a greater number of consumers.

Key points

1. The case of sodium nitrite poisoning in three family members, with one fatal outcome, is presented.
2. Sodium nitrite is used as a coloring agent or preservative in food and as an antimicrobial agent in meat.
3. In high amounts sodium nitrite can be toxic, causing methemoglobinemia.
4. Death generally occurs with methemoglobin levels over 70%; however, in individuals with some pre-existing diseases, death may occur with much lower concentrations.

Funding This work was supported by the Ministry of Education, Science and Technological Development of the Republic of Serbia, Grant No. 45005.

Compliance with ethical standards

Conflict of interest The authors hereby declare that they have no conflict of interest.

Ethical approval This article does not contain any studies with human participants or animals performed by any of the authors.

References

1. Lee C, Jang EJ, Yum H, Choi YS, Hong J. Unintentional mass sodium nitrite poisoning with a fatality. Clin Toxicol (Phila). 2017;55:678–9.
2. Wang R, Teng CG, Zhang N, Zhang J, Conway G. A family cluster of nitrite poisoning, Suzhou City, Jiangsu Province, China, 2013. Western Pac Surveill Response J. 2013;4:33–6.
3. Katabami K, Hayakawa M, Gando S. Severe methemoglobinemia due to sodium nitrite poisoning. Case Rep Emerg Med 2016;2016: 9013816.
4. Centers for Disease Control and Prevention (CDC). Methemoglobinemia following unintentional ingestion of sodium nitrite – New York, 2002. MMWR Morb Mortal Wkly Rep. 2002;51:639–42.
5. Chui JSW, Poon T, Chan KC, Chan AYW, Buckley TA. Nitrite-induced methaemoglobinaemia – aetiology, diagnosis and treatment. Anaesthesia. 2005;60:496–500.
6. Wright RO, Lewander WJ, Woolf AD. Methemoglobinemia: etiology, pharmacology, and clinical management. Ann Emerg Med. 1999;34:646–56.
7. Harvey M, Cave G, Chanwai G. Fatal methaemoglobinaemia induced by self-poisoning with sodium nitrite. Emerg Med Australas. 2010;22:463–5.
8. Matteucci O, Diletti G, Prencipe V, Di Giannatale E, Marconi MM, Migliorati G. Two cases of methemoglobinaemia caused by suspected sodium nitrite poisoning. Vet Ital 2008;44:439–453.
9. Greenberg M, Birnkrant WB, Schiftner JJ. Outbreak of sodium nitrite poisoning. Am J Public Health Nations Health. 1945;35: 1217–20.
10. McQuiston TAC, Belf MB. Fatal poisoning by sodium nitrite. Lancet. 1936;228:1153–4.