

Suicide by Sodium Nitrite Ingestion: An Autopsy Case Report*

May Vell Mañibo¹ and Raquel del Rosario-Fortun²

¹Department of Laboratories, University of the Philippines – Philippine General Hospital

²Department of Pathology, University of the Philippines College of Medicine

ABSTRACT

Sodium nitrite (SN, NaNO₂) is a water-soluble, white-yellow crystalline powder with broad applications in food preservation, automotive maintenance, and animal control. It is a strong oxidizing agent that can oxidize hemoglobin iron (Fe) to its oxidized state, leading to methemoglobin formation. An increasing trend of suicide cases by SN ingestion has been reported globally following its popularization in online suicide forums providing detailed instructions of its use solely or as part of a "suicide kit." We report a case of a 21-year-old male who was found continuously vomiting, with blood per orem and cyanosis of the mouth and digits. Within minutes of the onset of symptoms, the patient lost consciousness and was pronounced dead on arrival at the nearest emergency room. Autopsy findings showed lip erosions, hemorrhage, and perioral and peripheral cyanosis. Internal examination showed characteristic bright red muscle discoloration, dark brown arterial blood, red-brown congested visceral organs, and hyperemic esophageal and gastric mucosa. Methemoglobin studies from sampled arterial blood showed elevated levels (17.5%). Further investigation of the decedent's belongings, social media posts, and recent online purchases reinforced the intentional sodium nitrite ingestion. While there are plenty of reported SN poisoning in suicide cases internationally, limited reports have been published locally. Death by SN poisoning is preventable with Methylene blue. The role of forensic pathologists through autopsy may be the last chance to detect such cases. The lack of systemic death investigation, experts, and local laboratories to reliably detect the signs of SN poisoning may have affected the low detection rate of cases locally. Further reporting of cases can raise the awareness of medical professionals that is fundamental to the ultimate saving of lives.

Key words: : sodium nitrite, suicide, poisoning, forensic pathology, autopsy

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Corresponding author: May Vell M. Mañibo, MD

E-mail: mmmanibo@up.edu.ph

ORCID: <https://orcid.org/0009-0002-5267-1028>

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INTRODUCTION

Sodium nitrite (SN, NaNO₂) is best known as a food additive preventing the growth of microorganisms in meat. It is an odorless inorganic salt that is a white-yellow, crystalline powder and is highly water-soluble.¹ Being a strong oxidizing agent, SN, when absorbed into the body, can oxidize iron (Fe) in the hemoglobin from ferrous (Fe⁺²) to ferric (Fe⁺³) state, producing methemoglobin (MetHb). MetHb is unable to bind and transport oxygen, resulting in cellular hypoxia and ultimately leading to organ damage and death.¹ Recently, an alarmingly increasing trend in the use of SN for suicide purposes has been seen around the world following its popularization online.²

CASE

We report a case of a 21-year-old male with no known comorbidities. Relatives reported previous attempts of self-harm. Minutes prior to his demise, the decedent was seen continuously vomiting with blood per orem and cyanosis of the mouth and digits. He was immediately brought to the nearest hospital where cardiopulmonary resuscitation (CPR) was done but was then pronounced dead on arrival.

An autopsy was done 24 hours post-mortem. External examination findings showed no traumatic injuries or signs of assault. There was significant perioral and peripheral cyanosis (Figure 1). Non-specific grey skin discoloration in the dependent areas of the body was also noted. Lip erosions and hemorrhages were identified, consistent with



caustic burns (Figure 1). Internal examination showed characteristic bright red muscle discoloration, red-brown congested visceral organs, and hyperemic esophageal and gastric mucosa (Figure 2). The gastric contents consisted of brown, mucoid material (80 cc).

Further investigation of the decedent’s belongings at the scene revealed multiple-page suicide notes, a black rope, and a suspicious clear fluid in a commercial bottle of water. A review of phone and computer files was done revealing a recent online purchase history of sodium nitrite (1 kg) from a local distributor. Social media posts surrounding the time of death revealed ingestion of a bitter substance and vomiting as the initial symptoms.

Unfortunately, reliable direct assessment of sodium nitrite in the blood sample, gastric contents, and clear fluid in the bottled water is not available in the local setting. Blood sample from the root of the aorta was tested for

Methemoglobin eight (8) days postmortem, yielding elevated levels of 17.5% (normal values 1-1.5%).¹ With great efforts but limited resources, other toxicological studies of the gastric contents were not deemed to be any more contributory.

The cause of death is determined to be hypoxia secondary to methemoglobinemia from sodium nitrite poisoning. The manner of death is suicide.

DISCUSSION

Suicide accounts for 1% of all causes of death worldwide and is the 4th most common cause of death in the young (15-29 years old).² Since 2017, a significant increase in the use of sodium nitrite for suicide purposes has been seen around the world. Websites such as online suicide forums provide detailed instructions for the use of SN solely or as a part of a “suicide kit.”²

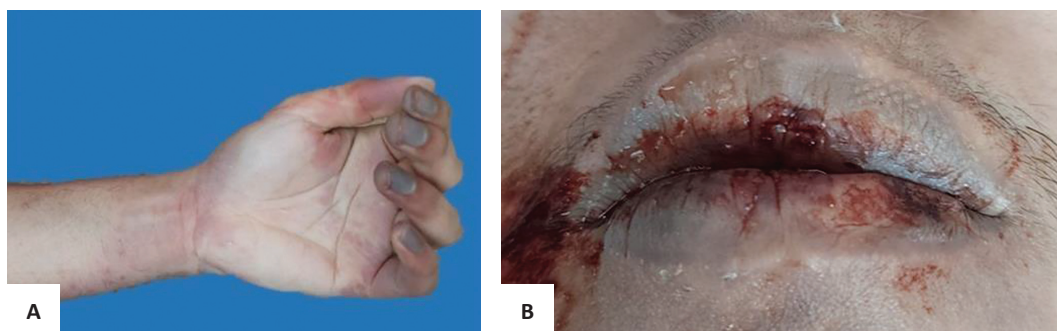


Figure 1. External examination findings. (A) Peripheral cyanosis and (B) perioral cyanosis, lip erosions, and hemorrhages.

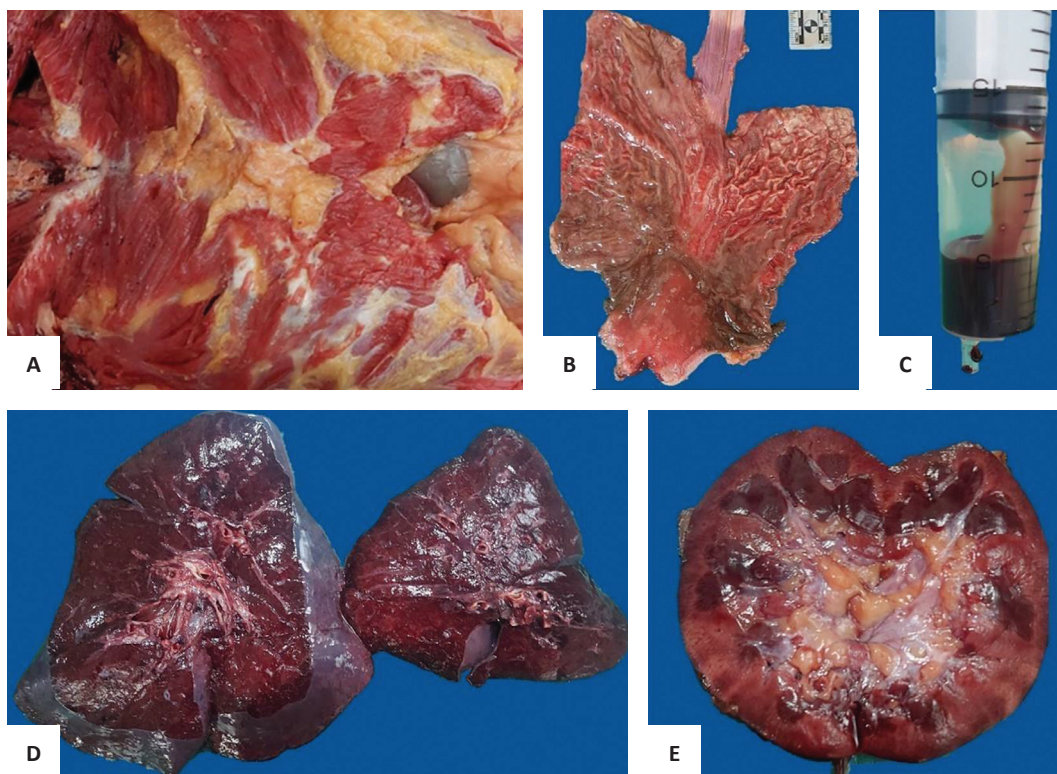


Figure 2. Internal examination findings. (A) Bright red muscle discoloration; (B) hyperemic esophageal and gastric mucosa; (C) dark-brown arterial blood. Red-brown congested visceral organs (D) lungs and (E) kidney.

While there are plenty of reported cases internationally, no local report of sodium nitrite poisoning in suicide cases has been published in the Philippines. A local journal article related to SN poisoning was published in 1996.³ The authors reported a case of accidental SN poisoning of a child after consumption of cured meat. This patient presented with vomiting, abdominal cramps, cyanosis, tachycardia and hypotension within one (1) hour of ingestion and yielded normal methemoglobin levels. The normal methemoglobin result was attributed to late testing (beyond 24 hours). This patient recovered after administration of ascorbic acid and intravenous fluid.³ To the best of the authors' knowledge and review of literature, no other local case of suicide by SN poisoning has been reported as of date.

Sodium nitrite is a potent oxidizing agent and is popularly known as a meat-curing compound. However, dietary exposure to small amounts is harmless.¹ A maximum level of less than 500 mg/kg (5 to 416 mg/kg) of cured food products has been regulated by the Bureau of Food and Drugs (BFAD) in the Philippines.⁴ The estimated fatal dose for humans is approximately 2.6 g, although cases of death from 1g and survival after 6 g ingestion were previously reported. Clinical symptoms of sodium nitrite poisoning include cyanosis, hypoxia, altered consciousness, dysrhythmias, and death.¹ Other uses of SN include corrosion inhibitors found in anti-freeze, antimicrobial, coloring agent and antidote to cyanide poisoning.²

A reliable measurement of SN levels in the blood is difficult due to nitrites rapidly converting into nitrates, resulting in a falsely low value.¹ The measurable value of methemoglobin is helpful in the indirect assessment of SN poisoning.¹ Methemoglobinemia is caused by the oxidation of ferrous to ferric iron leading to increased affinity of hemoglobin to oxygen hence reducing oxygen delivery to tissues leading to hypoxia. Increased levels of methemoglobin in blood account for the clinical manifestations. At 10%, cyanosis will be apparent. If the level reaches above 20%, other symptoms such as headache, dizziness, polypnea, tachycardia, and general asthenia may be observed. Values close to 60% may cause loss of consciousness and death at values higher than 70%.¹ Our case presented with vomiting, cyanosis, and loss of consciousness just minutes before death. Although the methemoglobin level of our case is at 17.5%, other factors including delay in testing (8 days) may have contributed to the falsely low levels, that are not compatible with the literature.¹

In general, methemoglobinemia may be caused by a congenital defect or an acquired disorder.⁵ Congenital causes include autosomal recessive variants in the *CYB5R3* gene or autosomal dominant variants in the globin genes, collectively known as HbM disease.⁵ These conditions present early on in life with cyanosis and hemolysis. Given the age of the decedent in our case, the absence of symptoms during childhood and the lack of family history, a congenital cause of methemoglobinemia is unlikely. On the other hand, acquired causes of methemoglobinemia include consumption of numerous drugs and toxic agents. The most common documented drugs are benzocaine and lidocaine – both of which are local anesthetics administered topically or parenterally and will not present with continuous vomiting and caustic burns such as in our case.⁵

Other recreational drugs such as amyl nitrate (poppers), nitrous oxide (laughing gas), and adulterants in cocaine were also reported to cause fatal methemoglobinemia.⁵ However, there is no documentation of previous use, possession, and/or purchase of any of these drugs by the decedent.

According to a 2022 systematic review of cases related to SN intoxication and death, there is a concerning 41.67% mortality rate - 80% of which with suicidal intent.⁶ Mortality is significantly higher in suicide cases due to greater quantities taken. A longer survival interval with the possibility of accessing an emergency department is reported in cases that took excessive but non-lethal quantities. Given this data, training of health professionals to quickly identify an acute intoxication and to implement necessary treatments is inferred.⁶ The antidote for methemoglobinemia from SN poisoning is methylene blue.¹ Methylene blue is given at a dose of 1-2 mg/kg and is infused over 5 min and can be repeated if symptoms persist.^{1,7} This antidote acts as a catalyst to reduce methemoglobin to hemoglobin via the enzyme NADPH-methemoglobin reductase.⁸ Ascorbic acid, hyperbaric oxygen, exchange transfusion, and extracorporeal membrane oxygenation are some of the additional treatment approaches for methemoglobinemia.⁸ There are reported cases of patients surviving from SN poisoning.⁹ Unfortunately, the index patient of this case report succumbed to death just minutes post-ingestion.

Documented autopsy findings in SN poisoning include post-mortem signs of methemoglobinemia such as blue-grey hypostasis, cyanosis, and dark-brown discoloration of blood and internal organs.^{1,5,6} Most of these are present in our case.

There is a general methodological discrepancy in the diagnostic process, and SN-related deaths represent a challenge for forensic pathologists.⁶ An important role in framing SN intoxication as cause of death is played by the investigation of the scene, and an accurately done autopsy.⁶ Forensic pathologists involved in such cases are advised to: 1) investigate the scene; 2) ascertain previous web searches, purchases, and consumption of uncontrolled food; 3) pay attention to livor mortis; 4) focus on autopsy findings; and 5) consider nitrite and methemoglobin dosage in suspected cases.⁶ With the permission and help from the relatives of the decedent, these tasks were taken by the forensic pathologist in charge of the case.

Distinguishing between manners of death particularly in suicide and accident cases, is a challenge.¹⁰ The role of pathologists in such cases is highlighted during post-mortem examination. Death investigation using autopsy, toxicological and histological examination is ideal if the cause of death is not readily recognized.^{11,12} While there are established and collaborative systemic death investigations between the police and forensic pathology experts internationally, the current local setting relies on the autopsy findings by pathologists.^{10,12}

CONCLUSION

Sodium nitrite ingestion has an alarming increasing trend of use for suicide purposes around the world. Easy access

to the substance and online forums providing step-by-step instructions on how to use it play a significant role in the continuously increasing number of suicide cases by SN ingestion. We report a case of a young adult who purchased SN online and committed intentional ingestion, with autopsy findings compatible with methemoglobinemia from SN poisoning. The acute, non-specific, and dose-related manifestations of SN poisoning pose a challenge in the clinical recognition and prompt management of cases. The lack of forensic pathology experts, systemic death investigation, and local laboratories to reliably recognize the autopsy signs may have also affected the low detection rate of cases locally. Further reporting of cases can raise the awareness of medical professionals and is fundamental in the ultimate saving of lives.

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ETHICAL CONSIDERATION

An informed consent was secured from the index patient's relative before the conduct of the autopsy and a separate informed consent for the writing of the case report. The completed report has been submitted and approved by the Expanded Hospital Research Office (EHRO) of the affiliated institution.

STATEMENT OF AUTHORSHIP

All authors certified fulfillment of ICMJE authorship criteria.

AUTHOR DISCLOSURE

The authors declared no conflict of interest.

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