A fatal case by a suicide kit containing sodium nitrite ordered on the internet

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ABSTRACT
Fatal sodium nitrite poisonings are rare in the forensic context. The present work describes a first fatal case of sodium nitrite contained in a suicide kit that the victim acquired over the internet. The results of the autopsy showed general signs of asphyxia, such as intense cyanosis of the extremities, brown-gray-blue-red livor mortis, and some Tardieu petechiae in addition to intense visceral congestion. It is clear that forensic experts must be aware of the proliferation of this market and the risks of improper selling of these substances through suicide support networks available on the internet. The lack of knowledge of this reality may become unidentifiable, when toxicological analysis contemplates only the most classical and frequent substances involved in poisoning and reinforce the importance of a careful analysis of the death scene.

1. Introduction

Sodium nitrite (NaNO₂) is an easily accessible food additive (i.e., E250), widely used as preservative, antimicrobial agent by inhibiting effect of Clostridium botulinum, an anaerobic bacteria responsible for botulism, and by its capacity to fix the pink color of the cured meat and fish and some cheeses. Sodium nitrite is also used as antifreeze admixture and to prevent corrosion of pipes and tanks, as an antidote for cyanide poisoning and it is a proved carcinogen since nitrites react with amines and amides to form nitrosamines.

While non-lethal intoxications by contaminated water and food is widely reported, lethal cases are rarely described. The estimated lethal dose of sodium nitrite in adults is approximately 2.6 g, but survival was also reported for ingestion of 6 g sodium nitrite. This work presents the first case described in the literature of a suicide by sodium nitrite after ordering a “suicide kit” on the internet.

1.1. Case report

A 37-year-old man was found dead inside his car next to a box that contained two glass cups, the medicines Primperan® (i.e., metoclopramide, 10 mg) and ranitidine (75 mg) and a 35 g package of crystalline sodium nitrite (Fig. 1). The social investigation revealed that the victim, who suffered from depression and schizophrenic disease without major expression, previously attempted suicide and recently consulted suicide support websites on the internet.

The most relevant autopic findings were the general signs of asphyxia, such as intense sceral congestion (Fig. 2A) and cyanosis of the extremities (Fig. 2B), namely hands and lips, and brown-gray-blue-red livor mortis in various parts of the body (Fig. 2C), some Tardieu petechiae in addition to intense polyvisceral congestion. The blood was tinted a chocolate brown color. Histological examination revealed only nonspecific changes, such as pulmonary edema and coronary artery disease without major expression.

The initial toxicological analysis, by gas and liquid chromatography both coupled to mass spectrometry, in femoral blood showed concentrations of desmethylcitalopram 90 ng/mL, citalopram 252 ng/mL and trazodone 280 ng/mL and negativity for other substances investigated. However, considering the evidence found at the crime scene, the femoral blood and gastric content were reanalyzed with a specific spectrophotometric methodology for sodium nitrite. Briefly, the method was based...
on a diazotization-coupling reaction between dapsone and N-(1-naphthyl)ethylenediamine in a hydrochloric acid medium.\(^{12}\) In the gastric content 16 g/L of nitrites and 24 g/L of sodium nitrite were obtained. In blood 30 ng/mL of nitrites were obtained.

2. Discussion

Sodium nitrite is a white crystalline powder with a similar appearance to kitchen salt, easily soluble in water, which eventually allows it to also be used in homicidal form concealed in drinks.\(^{13}\) Searching on the internet some websites advertise that: i) “sodium nitrate offers a number of features that make it an ideal human euthanasia agent. It is cheap, breaks down readily in the environment”; ii) “this is the secret powder to help old people who have a fulfilled life who are not terminally ill and want to die in a peaceful way. Only 2 g of the powder are needed.” Besides sodium cyanide and sodium azide, sodium nitrite due to its availability, preparation, un-detectability, speed, safety, and storage is advocated to be peacefulness suicide method.\(^{14}\)

Sodium nitrite causes asphyxia, since hemoglobin is oxidized to methemoglobin, precipitating methemoglobinemia. In other words, as an oxidizer, nitrite can convert the ferrous iron (Fe\(^{2+}\)) of hemoglobin to ferric iron (Fe\(^{3+}\)), forming methemoglobin, and thereby impairing oxygen transport. Similarly to carbon monoxide poisonings, due to the allosteric changes of hemoglobin, a left shift to the oxygen-hemoglobin...
dissociation curve occurs, increasing the affinity of hemoglobin to oxygen resulting in an impairment of oxygen delivery to the tissues and thus a vital anemic hypoxemia builds. Bradyasystolic cardiac arrest usually rapidly develops. Symptoms such as nausea, vomiting, lethargy, shortness of breath, convulsions and coma are evident soon after intoxication, especially if levels of methemoglobinemia are higher than 60%. Methemoglobinemia levels above 70% usually proved to be fatal, but cases of survival with levels up to 94% have been reported.

In our case, autopsy results revealed brown/gray/blue/red livor mortis in various anterior and posterior parts of the body, namely lips, hands, and shoulders. It is suggested to be an adverse consequence of the vasodilation, since sodium nitrite is an precursor of nitric oxide. Curiously, due to the increase of the blood flow, compounds such as sodium nitrite/nitrate have been subject to patent protection for performance enhancement in sport.

The brown color of the livor mortis is one of the major textbook signs of nitrite poisoning that can help to suspect about this poisoning. Nevertheless, the lack of this sign in some cases can be attributed to a low concentration of methemoglobin at the time of death. The classical chocolate-colored blood was slightly present in our case. Moreover, one of the most characteristic postmortem change was the blue-gray discoloration of the fingernail beds as signs of hypoxia due to methemoglobinemia.

The toxicological tests revealed increased nitrite ion levels in the blood and gastric contents. In other studies, methemoglobinemia of 9.87% and 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 were reported. Blood nitrite levels in death cases following accidental or intentional ingestion were reported to be 0.55–13 μg/mL. Methemoglobin concentrations were not analyzed in our case. An enzyme-linked immunosorbent assay (ELISA) kit, following the manufacturer’s instructions would be useful, but higher values than the reference range (1–2%) were suspected. In some deaths, sodium nitrite was detected in gastric contents but not in other body fluids or tissues. Therefore, as previously suggested, if the presence of nitrite is suspected in the blood, quantification of the methemoglobin levels may be more important than the nitrite or nitrate levels. Moreover, since in some cases nitrate levels were much higher than nitrite levels, the former should also be quantified. Indeed, in comparison to our results, similar nitrite levels (<0.05 and 0.09 μg/mL in cardiac and femoral blood, respectively) were also reported, for which nitrate levels (71.69 and 83.48 μg/mL in cardiac and femoral blood, respectively) were significantly high. The justification could be due to nitrite conversion to nitrate, which is incapable of oxidizing hemoglobin to methemoglobin in the body. Importantly, since nitrites and nitrates are not substances commonly analyzed in forensic toxicology services, false negative results of intoxication may be obtained. Metoclopramide and ranitidine were not detected in toxicological analysis. Although speculative, we hypothesized that these drugs are included in the suicide kit to reduce emesis and therefore to increase ingestion and tolerance to sodium nitrite.

In conclusion, based on the results of the forensic, toxicological, and death scene investigation, the cause of death was due asphyxiation resulting from methemoglobinemia induced by sodium nitrite overdose. Although, poor health condition or pre-existing disease, such as anemia, acidosis, respiratory compromise, and cardiac disease, increases the susceptibility to the fatal consequences of nitrite poisoning, autopsy did not reveal any remarkable signs of previous disease. It is also important to highlight that benzene, sodium nitrite intoxications, methemoglobinemia may arise from a variety of etiologies including but not limited to, kidney failure, sickle cell, cyanide and methemoglobinemia intoxications. The treatment of severe methemoglobinemia usually focus on aggressive decontamination, hyperventilation, and administration of reducing substances such as the thiamine dye methylene blue. Failure to detect sodium nitrite in routine toxicological analysis can result in false negatives. In this regard, the inclusion of methemoglobin, nitrite and nitrate quantification should be considered if crime scene is particularly suggestive. This case also reveals the danger and the importance of preventing and fighting existing suicide support networks over the internet, which allow the purchase of a “suicide kit”, by circumvent the customs services.

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Ethical approval

All procedures were performed according to the ethical and legal standards of the institution and the principles of the Declaration of Helsinki. This study did not require informed written or oral consent from the victims’ families since this is a scientific research conducted during routine forensic procedures.

Informed consent

Not applicable.

Declaration of competing interest

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