



# Internet-Acquired Sodium Nitrite as an Emerging Suicide Method: Clinical Presentation and Management of Profound Methemoglobinemia

*Yeni Gelişen bir İntihar Yöntemi Olarak İnternette Temin Edilen Sodyum Nitrit: Şiddetli Methemoglobineminin Klinik Prezantasyonu ve Yönetimi*

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## ABSTRACT

Sodium nitrite (NaNO<sub>2</sub>), an industrial food preservative, has emerged as an accessible means for suicide with increasing online availability. This compound induces methemoglobinemia by oxidising haemoglobin, impairing oxygen transport and causing potentially fatal tissue hypoxia.

We report the case of a 30-year-old male who presented to the emergency department after intentionally ingesting approximately 5000 mg of sodium nitrite purchased online. The patient arrived in a confused state, exhibiting severe cyanosis, tachypnoea, and a blood oxygen saturation of 40%. Laboratory analysis revealed a methaemoglobin level of 58%. Despite diagnostic limitations and treatment delays, the patient was successfully treated with two doses of intravenous methylene blue (1 mg/kg), resulting in complete normalisation of methaemoglobin levels within 24 hours. This case highlights critical challenges in managing sodium nitrite poisoning, including misleading standard pulse oximetry readings and the importance of recognising clinical indicators such as “chocolate brown” blood. Despite significant methemoglobinemia (58%), the successful treatment outcome underscores the efficacy of methylene blue therapy when administered promptly. The increasing accessibility of sodium nitrite online poses a significant public health concern. Healthcare providers should maintain a high level of suspicion for methemoglobinemia in patients presenting with unexplained cyanosis resistant to oxygen therapy, prioritise co-oximetry for accurate diagnosis, and ensure the immediate availability of methylene blue. This case emphasises the need for improved regulation of industrial chemicals with lethal potential and enhanced mental health interventions.

**Key words:** methemoglobinemia; sodium nitrite; methylene blue; cyanosis; hypoxia

## ÖZET

Endüstriyel bir gıda koruyucusu olan sodyum nitrit (NaNO<sub>2</sub>), internet üzerinden erişilebilirliğinin artmasıyla birlikte kolay ulaşılabılır bir intihar yöntemi olarak kullanımı ortaya çıkmıştır. Bu bileşik, hemoglobini oksitleyerek oksijen taşınımını bozar ve potansiyel olarak ölümcül doku hipoksisine neden olan methemoglobinemiye yol açar. Bu olgumuzda, internette satın aldığı yaklaşık 5000 mg sodyum nitriti kasıtlı olarak içtikten sonra acil servise başvuran 30 yaşında bir erkek hasta sunulmaktadır. Hasta, acil servise başvurduğunda bilinç bulanıklığı, şiddetli siyanoz, takipne (hızlı solunum) ve %40 kan oksijen satürasyonu göstermekteydi. Laboratuvar analizinde methemoglobin seviyesi %58 olarak saptandı. Tanısal kısıtlılıklara ve tedavideki gecikmelere rağmen hasta, iki doz intravenöz (damar içi) metilen mavisi (1 mg/kg) ile başarılı bir şekilde tedavi edildi ve 24 saat içinde methemoglobin seviyelerinin tamamen normal düzeylere döndüğü gözlemlendi. Bu vaka, sodyum nitrit zehirlenmesinin yönetimindeki kritik zorlukları vurgulamaktadır. Bunlar arasında standart nabız oksimetresi ölçümlerinin yanıltıcı olabilmesi ve “çikolata kahverengisi” kan gibi klinik belirtilerin tanınmasının önemi bulunmaktadır. %58 gibi anlamlı bir methemoglobinemi düzeyine rağmen tedavinin başarılı sonucu, metilen mavisi tedavisinin zamanında uygulandığında ne kadar etkili olduğunu göstermektedir. Sodyum nitritin internet üzerinden artan erişilebilirliği, önemli bir halk sağlığı sorunu teşkil etmektedir. Sağlık hizmeti sağlayıcıları, oksijen tedavisine dirençli ve açıklanamayan siyanoz ile başvuran hastalarda methemoglobinemiden yüksek düzeyde şüphelenmeli, doğru tanı için ko-oksimeetriye öncelik verilmeli ve metilen mavisinin acil olarak temin edilebilirliğini sağlamalıdır. Bu vaka, ölümcül potansiyele sahip endüstriyel kimyasalların temin edilme sürecindeki regülasyonların iyileştirilmesi ve toplum ruh sağlığı müdahalelerinin iyileştirilmesinin gerekliliğini vurgulamaktadır.

**Anahtar kelimeler:** methemoglobinemi; sodyum nitrit; metilen mavisi; siyanoz; hipoksi

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## Introduction

Sodium nitrite ( $\text{NaNO}_2$ ) is a yellowish-white compound commonly used as an industrial food preservative<sup>1,2</sup>. Recently, it has been recognized as an easily accessible means for suicide, often obtained online.

Reports from U. S. Poison Centres indicate a significant increase in suicidal use of chelating agents such as sodium nitrite in recent years. This trend predominantly affects young adults aged 15 to 34, who constitute approximately 68% of these cases. Although these cases currently account for less than 1% of all suicides, there were 768 fatalities involving antidotes and chelating agents, such as sodium nitrite, from 2018 to July 2023, out of a total of 268,972 suicides in that time-frame. The case fatality rate is notably high at approximately 41.5%, underscoring the severity of using these agents in suicide attempts<sup>3,4</sup>.

From a toxicological perspective, sodium nitrite facilitates the oxidation of haemoglobin iron ( $\text{Fe}^{2+}$  to  $\text{Fe}^{3+}$ ), forming methaemoglobin<sup>2,5,6</sup>. This consequently impairs oxygen transport and induces nitric oxide formation, leading to vasodilation, subsequent hypotension, and reflex tachycardia. Clinically, this manifests in a dose-dependent manner, presenting as tissue hypoxia characterized by cyanosis, dyspnoea, altered mental status, seizures, and cardiovascular collapse<sup>7,8</sup>.

While MetHb levels above 70% are generally linked to mortality, survival was shown with a MetHb level of 92%<sup>1,8,9</sup>. The lethal dose of sodium nitrite for adults is roughly 2,600 mg; however, fatalities have been recorded after consuming 1,000 mg, while survival has been noted even following doses of 6,000 mg<sup>8</sup>.

Diagnostic challenges in clinical settings often arise with standard two-wavelength pulse oximetry, which can yield misleading  $\text{SpO}_2$  values, typically around 85%, despite severe hypoxemia in patients with methemoglobinemia. This can obscure the actual severity of hypoxia, highlighting the need for accurate diagnosis through arterial blood gas sampling or specialized multi-wavelength oximetry<sup>3</sup>.

While sodium nitrite ingestion can rapidly progress to fatality<sup>7</sup>, prompt administration of intravenous methylene blue (1–2 mg/kg) serves as the definitive antidote by facilitating methaemoglobin reduction to functional haemoglobin, demonstrating efficacy even in severe intoxication<sup>3,8,10</sup>. The treatment protocol for methemoglobinemia involves intravenous methylene blue at 1–2 mg/kg over 5 minutes, with a maximum of

7 mg/kg. Methylene blue promotes the conversion of methaemoglobin to normal haemoglobin by inhibiting endothelial and inducible nitric oxide synthase and guanylate cyclase. Notably, large doses may also cause methemoglobinemia<sup>10,11</sup>.

This report details a case of methemoglobinemia resulting from the ingestion of sodium nitrite purchased online, which was effectively treated with methylene blue. The easy access to sodium nitrite on the internet has contributed to its use in self-harm, presenting significant toxicological challenges associated with elevated mortality rates<sup>4,11</sup>. The potential for rapid clinical deterioration and severe hypoxia due to nitrite-induced methemoglobinemia emphasizes the importance of prompt recognition and immediate administration of antidote therapy, such as methylene blue, to ensure effective management and improved patient outcomes.

## Case Report

A 30-year-old male patient was brought to the emergency department by emergency medical services after self-reporting sodium nitrite ingestion three hours earlier. He arrived at the emergency department (ED) with rapid breathing and in a confused state, presenting a Glasgow Coma Scale (GCS) score of 11 (E: 3, V: 4, M: 4). His initial oxygen saturation was recorded at 40%, and the  $\text{SO}_2$  in the arterial blood gas sample was 30%. After administering 10 liters per minute of oxygen through a reservoir bag, his GCS score improved to 15. The patient had a mean arterial blood pressure of 84 mmHg and a heart rate of 96 beats per minute.

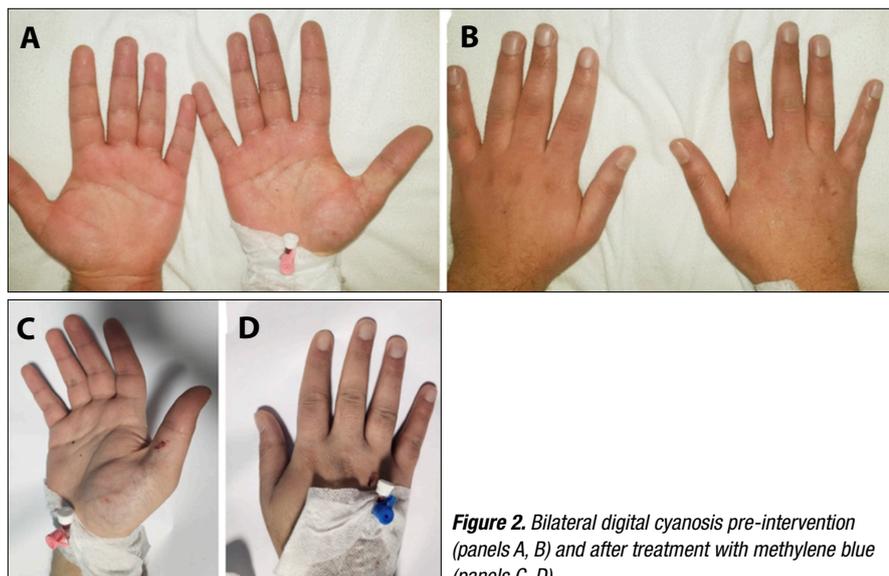
Regarding his medical history, the patient has a confirmed diagnosis of major depression and does not regularly follow any psychiatric medication regimen. Previously, he attempted to use Largactil and lithium but discontinued both. He stated that he is a psychologist and has been contemplating suicide for ten years. He has no other chronic illnesses.

The patient reported ingesting three tablespoons (about 5000 milligrams) of sodium nitrite mixed with fruit juice three hours earlier. Fig. 1 shows the 500 g sodium nitrite container, which is 98.0% active ingredient by weight and was purchased online by the patient.

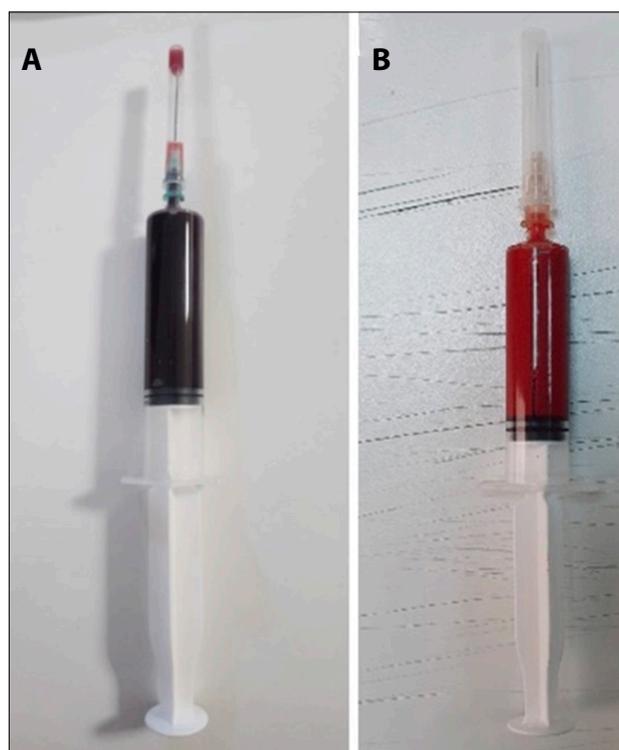
Despite receiving 10 liters of oxygen therapy, the patient remained cyanotic, with notable discoloration of the extremities (Fig. 2). Oxygen saturation, measured using a plethysmograph, was 84%, and a venous blood sample for routine laboratory tests appeared dark



**Figure 1.** Bottle containing 500g of sodium nitrite.



**Figure 2.** Bilateral digital cyanosis pre-intervention (panels A, B) and after treatment with methylene blue (panels C, D).



**Figure 3.** Blood samples demonstrating methemoglobinemia with chocolate-brown appearance (A) and post-methylene blue intervention (B).



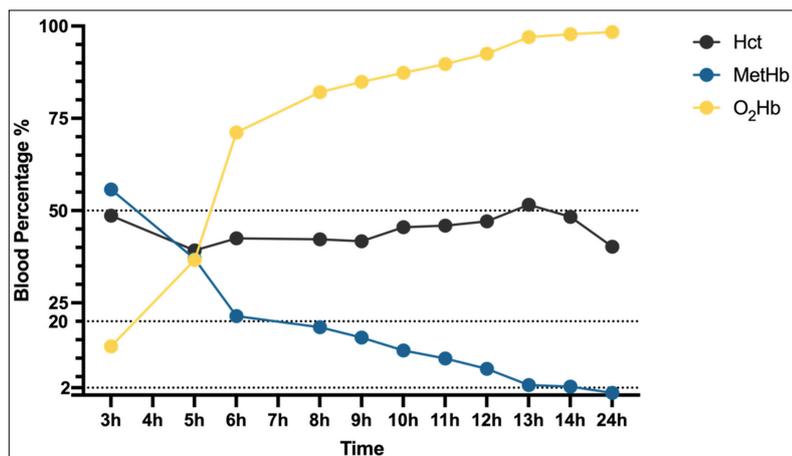
**Figure 4.** Urinary specimens showing a pre-intervention dark discolouration (A), amber transitional phase (B), and resolution (C).

chocolate (Fig. 3), while the urine showed significant dark discoloration (Fig. 4). The patient was breathing spontaneously but exhibited tachypnea at 28 breaths per minute. The rest of the physical examination was unremarkable.

Several differential diagnoses were systematically eliminated. Carbon monoxide poisoning was ruled out due

to the lack of cherry-red skin and relevant exposure history. Cyanide poisoning was also excluded because severe lactic acidosis was not present. Radiological tests ruled out respiratory conditions such as pulmonary embolism, pneumothorax, and pneumonia.

Because our center lacks a kit to measure blood nitrate levels, the patient's blood concentrations remain



**Figure 5.** Graph illustrating the dynamic changes in, methemoglobin, oxyhaemoglobin and haematocrit levels measured over a 24-hour period with arterial blood gas sampling.

unknown. Considering the patient's history of ingesting sodium nitrite, presenting with cyanosis, and having persistently low oxygen saturation not exceeding 84% despite oxygen therapy, along with the observation of dark brown blood during venipuncture, methemoglobinemia was strongly suspected. Laboratory results later confirmed a blood methemoglobin level of 58%.

The patient was moved to the intensive care unit (ICU) for treatment and monitoring. Femoral catheterization and radial artery cannulation were performed to help manage potential hemodynamic issues and for arterial blood gas sampling. However, the patient did not need vasopressors and stayed hemodynamically stable throughout his ICU stay.

The National Poison Consultation Hotline was contacted, and supportive care and methylene blue therapy were recommended. Given the patient's symptomatic condition and a methaemoglobin level of 58%, methylene blue treatment was planned. Since methylene blue was unavailable at our centre, we obtained it from the nearest facility within an hour. In addition to routine monitoring, methaemoglobin levels were closely monitored both invasively via arterial blood gas analysis at 1-hour intervals and non-invasively using a Masimo SpMet<sup>®</sup> probe.

Methylene blue treatment was started at 1 mg/kg over 5 minutes at the fourth hour after ingestion. One hour after the initial dose, since the patient still required 10 liters per minute of oxygen via a reservoir bag and his methemoglobin level remained above 30% (36.9%), a second dose of 1 mg/kg was given. No adverse reactions were observed.

Following treatment, laboratory results showed a significant decrease in methaemoglobin levels. The levels dropped to 21.4 percent at the sixth hour and returned to normal at 24 hours, reaching 0.6 percent (Fig. 5). By the eighth hour, the patient's oxygen needs decreased considerably, with a PO<sub>2</sub> of 314 mmHg while receiving 10 liters of oxygen per minute through a reservoir bag. Oxygen therapy was then reduced to 4 liters per minute via a nasal cannula, resulting in a PO<sub>2</sub> of 126 mmHg. A psychiatry consultation was performed, and based on the psychiatrist's recommendations following a recent suicide attempt, the patient was monitored in the ICU for two days before being transferred to the psychiatric ward in stable condition.

## Discussion

This case report details the successful management of severe methemoglobinemia following intentional ingestion of sodium nitrite, highlighting its alarming emergence as a readily accessible suicide method facilitated by online purchasing<sup>3,4,12</sup>. The ease with which this industrial chemical can be acquired for self-harm presents a significant public health crisis, contributing to high mortality rates and posing unique challenges for emergency care providers<sup>4,12</sup>.

Our patient's presentation —refractory cyanosis, tachypnoea and confusion— typifies the clinical picture associated with methemoglobinemia<sup>2,5-8</sup>. It also highlights the diagnostic pitfall of relying only on standard pulse oximetry (SpO<sub>2</sub>), which misleadingly showed 84% blood oxygen saturation, masking the actual severity confirmed only by co-oximetry and blood-gas sampling<sup>3</sup>. This aligns with the literature emphasizing

the necessity of specific diagnostic tools and recognizing clinical signs such as “chocolate brown” blood<sup>3,8</sup>. The successful reversal of methemoglobinemia after administering intravenous methylene blue, the established antidote, confirms the effectiveness of standard treatment protocols even in severe cases<sup>3,8,11</sup>. This case demonstrates that prompt diagnosis and appropriate treatment can still lead to positive outcomes despite potential system delays.

Although the treatment ultimately proved successful, the inability to quantify blood nitrite levels represented a significant limitation. The delay in acquiring methylene blue, due to its unavailability at our centre, highlights a systemic vulnerability. This barrier could have serious consequences for other institutions that lack immediate access to this cost-effective antidote, as delays in its availability can negatively impact patient outcomes.

In summary, clinicians must maintain a heightened suspicion of methemoglobinemia in refractory cyanosis, prioritize co-oximetry over standard SpO<sub>2</sub> measurements when available, and promptly administer methylene blue. Crucially, healthcare systems should ensure rapid diagnostic capabilities and the immediate availability of essential antidotes, such as methylene blue. Further research and public health initiatives are needed to monitor trends in sodium nitrite misuse, explore potential strategies to limit its accessibility for self-harm purposes, and address the underlying mental health factors contributing to such suicide attempts.

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