



Severe Methaemoglobinaemia and Acute Kidney Injury Following Intracavernosal Injection of a Recreational Alkyl Nitrite Product: A Case Report

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Abstract

Poppers (alkyl nitrites) are commonly used recreational vasodilators that are typically inhaled. Severe methaemoglobinaemia is a recognised complication of nitrite exposure, but life-threatening toxicity from unconventional routes of administration is rarely described. A 27-year-old man presented with profound cyanosis and persistently low oxygen saturation approximately 2 hours after self-injecting an estimated 10–20 mL of an alkyl nitrite product into the base of the penis, based on the patient’s report. Co-oximetry confirmed severe methaemoglobinaemia (41.7%) with metabolic acidosis, and examination demonstrated ischaemic priapism with a large scrotal haematoma. He was treated with high-flow oxygen, intravenous fluids, and two doses of methylene blue (2 mg/kg each), resulting in resolution of cyanosis and normalisation of oxygen saturation. Renal function was mildly impaired at presentation despite previously normal renal function, then progressed rapidly to Stage 3 acute kidney injury with oliguria on repeat testing the following morning, requiring critical care admission and urgent haemodialysis. Renal function fully recovered by 2-week follow-up. This case underlines the need to consider methaemoglobinaemia in patients with persistent cyanosis despite oxygen therapy, particularly where there is a clear saturation gap between pulse oximetry and arterial oxygen tension. It also demonstrates the risk of significant multi-organ toxicity when alkyl nitrites are used via non-inhalational routes.

Key Points

- 1. This report describes severe methaemoglobinaemia (41.7%) with dialysis-requiring acute kidney injury after patient-reported intracavernosal injection of an alkyl nitrite product. It shows that parenteral nitrite exposure may lead to rapid severe systemic toxicity.

2. Methaemoglobinaemia should be considered when cyanosis does not improve with oxygen therapy, especially where pulse oximetry readings remain low despite relatively preserved arterial oxygen tension. Early co-oximetry and prompt methylene blue treatment are essential.

3. Intracavernosal injection may precipitate ischaemic priapism and local haematoma, which can complicate management. Early multidisciplinary involvement from emergency, critical care, nephrology, and urology teams helps optimise outcomes, and harm-reduction advice should be offered.

INTRODUCTION

Poppers, a colloquial term for volatile alkyl nitrites, are widely used recreational substances because of their rapid vasodilatory and smooth muscle relaxing properties. Most exposures occur via inhalation, but serious toxicity can occur, including methaemoglobinaemia and systemic hypotension.^{1–3} Although oral ingestion and other atypical exposures have been reported, parenteral exposure remains uncommon, with only isolated reports of intravenous nitrite misuse.⁴ Methaemoglobinaemia can be challenging to recognise because cyanosis persists despite oxygen therapy, and pulse oximetry may be discordant with arterial oxygen tension (the saturation gap).^{5,6}

Acute kidney injury following nitrite exposure is rarely reported, but severe cases requiring haemodialysis have been described.⁷ This report describes life-threatening methaemoglobinaemia with rapidly progressive acute kidney injury following intracavernosal injection of an alkyl nitrite product, with concurrent ischaemic priapism.

CASE DESCRIPTION

A 27-year-old man with no known past medical history presented to the emergency department by ambulance approximately 2 hours after self-injecting an estimated 10–20 mL of an alkyl nitrite product into the base of the penis, based on the patient's report. The patient described the injected product as 'amyl nitrite'; however, the product container was not available for inspection or analytical testing, and the precise alkyl nitrite composition could not be confirmed. He stated that the injection was performed to enhance

sexual experience and denied co-ingestants. Previous blood tests performed approximately 1 month earlier had demonstrated normal renal function.

On arrival, the patient was alert and fully orientated but appeared markedly cyanotic. Observations showed blood pressure of 170/85 mmHg, heart rate of 135 beats per minute (sinus tachycardia), respiratory rate of 20 breaths per minute, and temperature of 36.8 °C. Pulse oximetry remained persistently low despite high-flow oxygen delivered via a non-rebreather mask. Chest examination revealed clear breath sounds bilaterally.

The documented local findings were penile swelling, painful rigid erection, and scrotal haematoma at the injection site. No further description of cutaneous chemical injury was available in the clinical record. Peripheral perfusion was otherwise adequate and no focal neurological deficit was identified.

Initial venous blood samples appeared dark chocolate brown. Arterial blood gas analysis on high-flow oxygen demonstrated pH: 7.21; PaCO₂: 41 mmHg; bicarbonate: 14 mmol/L; base excess: –12 mmol/L; lactate: 4.5 mmol/L; and PaO₂: 308 mmHg. Co-oximetry confirmed a methaemoglobin level of 41.7%.

Renal function was mildly impaired at presentation and worsened on repeat testing the following morning, consistent with Stage 3 acute kidney injury with oliguria classified according to Kidney Disease Improving Global Outcomes (KDIGO) criteria. Other biochemical parameters, including markers of muscle injury, were within normal limits. CT imaging of the kidneys, ureters, and bladder showed no evidence of obstruction or structural renal abnormality.

High-flow oxygen and intravenous crystalloid resuscitation were initiated immediately. Intravenous methylene blue was given at 2 mg/kg, with partial improvement in oxygen saturation. A second 2 mg/kg dose was administered after reassessment due to incomplete response, in keeping with published guidance for severe or refractory cases.⁵ Following the second dose, cyanosis resolved and oxygen saturation normalised.

Urgent urology review was requested. Corporal aspiration was attempted but did not achieve adequate decompression. A suprapubic catheter was inserted to secure urinary drainage and permit accurate fluid balance monitoring.⁸⁻¹⁰

The patient was transferred to critical care and required urgent intermittent haemodialysis for worsening metabolic acidosis and oliguria. Over the subsequent week, urine output improved and renal function recovered. At 2-week outpatient follow-up, renal function had normalised and renal replacement therapy was no longer required.

The patient received harm-reduction counselling and was offered referral to substance misuse services. Poppers maculopathy and the need to report visual symptoms were discussed.¹¹

DISCUSSION

Methaemoglobinaemia arises when the iron in haem is oxidised from the ferrous (Fe^{2+}) to the ferric (Fe^{3+}) state, impairing oxygen delivery despite adequate ventilation.⁵ Clinically, it presents with cyanosis that does not resolve with supplemental oxygen and a saturation gap where pulse oximetry underestimates oxygenation compared with arterial PaO_2 .^{5,6} Alkyl nitrites are established causes of acquired methaemoglobinaemia in recreational settings.^{1,2,6}

In this case, intracavernosal injection likely permitted rapid systemic absorption of the alkyl nitrite product, resulting in a severe methaemoglobin level of 41.7% and clinically significant functional hypoxia.^{4,5}

Unless analytical testing of the product is performed, it is more accurate to refer to such products as alkyl nitrites, as commercially available products may contain butyl nitrite, isobutyl nitrite, or other alkyl nitrites rather than amyl nitrite specifically.^{12,13}

Methylene blue is first-line therapy for clinically significant methaemoglobinaemia. It acts as an electron carrier to accelerate the nicotinamide adenine dinucleotide phosphate (NADPH)-dependent reduction of methaemoglobin back to functional haemoglobin. An initial dose of 1–2 mg/kg intravenously is recommended, with repeat dosing considered if response is inadequate.^{5,14}

Severe acute kidney injury following nitrite exposure is uncommon but has been reported, including dialysis-requiring cases.^{7,15} In this patient, normal baseline renal function 1 month earlier and the subsequent full recovery support an acute, reversible process. The rapid progression between presentation and the following morning is consistent with acute tubular injury, plausibly mediated by combined oxidative stress from nitrite exposure and renal hypoxia from impaired oxygen delivery.^{7,15}

Pigment-related renal injury typically involves renal vasoconstriction, intratubular cast formation, and direct tubular toxicity from haem proteins.¹⁶ Although there was no evidence of rhabdomyolysis on available testing, severe oxidative haem protein stress may contribute to tubular injury in profound methaemoglobinaemia.^{15,16}

The mechanism of priapism in this case is uncertain. Alkyl nitrites are vasodilators and have previously been described in the context of priapism treatment; therefore, a direct pharmacological causal link should be interpreted cautiously. In this case, the more plausible explanation is local injection-related injury, including direct cavernosal trauma, irritant chemical exposure, and haematoma, causing impaired venous outflow and low-flow priapism.⁸⁻¹⁰

It is worth noting that alkyl nitrite liquids are documented to be highly irritant and potentially corrosive. Cases of chemical

burns and mucosal injury following direct liquid contact have been reported in the literature. This is relevant in cases of non-inhalational exposure, where local tissue damage at the site of contact may compound systemic toxicity.^{3,17}

Non-inhalational routes of exposure may be associated with a higher risk of severe systemic toxicity. Clinicians should specifically enquire about the route and product used, and ask about visual symptoms in view of reported cases of poppers maculopathy.¹¹

A limitation of this report is that the product was identified from the patient's history and was not available for analytical confirmation. Commercial 'poppers' products may contain

different alkyl nitrites or mixtures, so the exposure is best described as a recreational alkyl nitrite product rather than confirmed amyl nitrite.^{12,13}

CONCLUSION

This case demonstrates that intracavernosal administration of an alkyl nitrite product may lead to severe methaemoglobinaemia and rapidly progressive acute kidney injury requiring haemodialysis, alongside local complications such as ischaemic priapism and scrotal haematoma. Early recognition of methaemoglobinaemia, prompt methylene blue treatment, and coordinated multidisciplinary management are important to optimise patient outcomes.

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