Saturday night blue—a case of near fatal poisoning from the abuse of amyl nitrite

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Abstract
A case of severe methaemoglobinaemia caused by the abuse of volatile nitrates is reported. The agents are commonly abused, but this complication is rare. The clinical presentation can make diagnosis difficult; however, the subsequent treatment needs to be rapid to avoid serious morbidity or mortality. This report presents the clinical picture and the background information leading to the detection and treatment of this unusual problem.

Keywords: methaemoglobinaemia; volatile nitrates; diagnosis; treatment.

The use of volatile nitrates as drugs of abuse is commonplace. We present here an uncommon but potentially fatal complication of their use. Rapid diagnosis and treatment in this case resulted in a full recovery and we have highlighted the important features of this uncommon toxicological problem.

Case report
An unknown white woman in her early twenties was found collapsed outside a police station. She was initially thought to be dead, as she was blue, there was no obvious breathing, and no pulse was palpable. An ambulance was called and on their arrival the crew detected a pulse, some respiration was evident, and she was treated with 100% oxygen and a total of 1.6 mg naloxone. She remained blue, her respiration was unchanged, and a pulse oximeter read 85%.

On arrival in accident and emergency (A&E) she was profoundly cyanosed with rapid irregular respiration, pulse 130/min, blood pressure 80/30 mm Hg, and marked peripheral vasodistention. Her pupils were dilated but weakly reactive, Glasgow coma score 5. She was actively resuscitated with a rapid infusion of colloid and was intubated and ventilated with 100% oxygen. Arterial blood gases...
showed pH 7.1, PO₂ 56 kPa, Sao₂ 100%, base excess -16. Pulse oximetry read 85%. The methaemoglobin level was 83% on co-oximetry. Gastric lavage produced a pungent smelling fluid, and a diagnosis of methaemoglobinemia due to volatile nitrite ingestion was made.

She was given 2 mg/kg methylene blue intravenously and admitted to intensive care. After one hour her methaemoglobin had fallen to 26% and she was given a further 1 mg/kg methylene blue. An hour later the methaemoglobin was 7%. She continued to improve and was extubated seven hours later. She made a full recovery.

It transpired that she had purchased a bottle of “poppers”, and had drunk the contents (15 ml) rather than sniffing it. Feeling unwell, she had been put by her boyfriend in a taxi and when she became unconscious he fled, leaving the driver to take her to the police station.

Discussion

“Poppers” is the street name for volatile nitrites taken by inhalation. The exact composition varies, and amyl, butyl, and isobutyl nitrite are all used.1 “Liquid Gold” is isobutyl nitrite, sold as a “room odoriser”.2 Clinical effects lasting a minute or two include vasodilatation causing tachycardia and hypotension.2 Smooth muscle relaxation and euphoria are said to prolong orgasm, hence its popularity. Perioral dermatitis may occur after repeated use1 and tracheobronchitis and haemolytic anaemia have been reported.

Methaemoglobin is formed by oxidation of ferrous (Fe²⁺) haem to ferric (Fe³⁺), which is incapable of binding oxygen for transport. Normal blood levels are 0–2%, and below 15% clinical effects are unlikely. The characteristic slate grey cyanosis appears at up to 30%, with headache, fatigue, and dizziness. At up to 50%, tachycardia, dyspnoea, and severe weakness are seen. At levels of more than 50% the effects of cellular hypoxia appear, with acidosis, cardiac dysrhythmias, respiratory depression, and coma.

There are two rare congenital forms: haemoglobin M disease, and a deficiency of the enzyme methaemoglobin reductase. Poisoning is far more common. Agents causing methaemoglobinemia include nitrates and nitrites, aniline dyes, chlorate salts, nitrobenzene, and the drugs benzocaine, chloroquine, dapson, lignocaine, phenacetin, prilocaine, and sulphonamides. Susceptibility is variable but pre-existing cardiopulmonary disease increases the severity of clinical effects.

Definitive treatment is based on reducing the Fe³⁺ with methylene blue and preventing further absorption of the oxidising agent. In extreme cases exchange transfusion has been used and has been effective in improving oxygen carriage. Methylene blue acts as a substrate for NADPH dependent methaemoglobin reductase, producing leucomythylene blue, which increases the rate of conversion of methaemoglobin. Contraindications to the use of methylene blue include cyanide poisoning, where sodium nitrite is given, producing methaemoglobin which binds to the cyanide ion to form inert cyanomethaemoglobin. In this situation methylene blue frees the bound cyanide. Glucose-6-phosphate deficiency is a relative contraindication, as methylene blue may induce haemolytic anaemia.3

It is important to appreciate that pulse oximetry can be misleading. It measures the ratio of absorption of light at 660 nm (the point of maximum difference between oxyhaemoglobin and deoxyhaemoglobin) and at 940 nm (where absorbances are virtually identical). Methaemoglobin shifts this ratio towards 1.0, corresponding to a saturation of 85%, so at high concentrations the reading becomes meaningless. Methylene blue mimics reduced haemoglobin, so a falsely low reading is obtained.3,4

Arterial blood gas analysis measures dissolved oxygen concentrations and hence partial pressures, so it is unaffected by dyshaemoglobins. The saturation value is derived, assuming normal haemoglobin, and can give a false sense of security.

Co-oximetry uses spectral absorption to measure oxy-, deoxy-, carboxy-, and methaemoglobin directly. This is more useful since tissue oxygen delivery depends on blood oxygen content, that is, oxyhaemoglobin levels.

Methylene blue does affect co-oximetry. It alters values, but the level of effect is a 4.3% negative error in methaemoglobin readings at a concentration of 25 mg/dm³ and this measurement change is probably below clinical significance in this scenario.

Cases of poisoning by volatile nitrites are regularly reported, both ingestion and inhalation having fatal consequences.3-6 In this case the level of 83% is extremely high and was likely to be fatal. Astute clinical diagnosis, the availability of co-oximetry, and rapid treatment with methylene blue prevented an almost fatal overdose of what may be becoming an increasingly popular drug.

1 Dixon DS, Reisch RF, Santinga BS. Methaemoglobinemia resulting from ingestion of isobutyl nitrite, a “room odoriser” widely used for recreational purposes. J Forensic Sci 1987;22:587–93.