

but took his own discharge 12 hours after admission. At that time an abdominal ultrasound was negative and full blood count was normal.

On the present admission routine blood examination revealed a leucocytosis of $23.1 \times 10^9/l$, haemoglobin of 15.0 g/l, and normal serum amylase and liver function tests. Plain abdominal radiographs showed localised dilated loops of bowel in the upper quadrant to the left with a few fluid levels. There was no free intraperitoneal gas and chest x ray showed no rib fractures.

After three hours of resuscitation, laparotomy was undertaken through a midline incision. There was a three inch long ischaemic segment in the upper sigmoid colon, which was severely bruised with areas of gangrene and necrosis. Its surface was thin in places and about to perforate. Nine inches of colon were resected. Five inches proximal to this, the lower descending colon showed a five inch long segment which was contused, indurated, and thickened but viable. Both areas of damage corresponded with the position of seat belt straps. A Hartmann's procedure was carried out without difficulty. The rest of the laparotomy was normal. Postoperative recovery was uneventful.

Histopathology of the resected segment showed extensive mucosal necrosis with focal

ulceration and transmural acute inflammation, with submucosal oedema and vascular congestion. Parts of the wall were completely gangrenous and the appearance was in keeping with ischaemic injury.

Discussion

The use of seat belts has reduced the incidence and severity of road traffic accidents injuries.¹ Nevertheless, seat belt themselves can cause serious injuries. In abdominal trauma the underlying viscera may be crushed between the seat belt, the abdominal wall, and the lumbar vertebrae if the anterior abdominal muscles are relaxed at the moment of impact, or if the viscera are distended or pathologically enlarged. Late presentation occurs if the bowel is not ruptured immediately. In such cases sepsis may develop, with increased mortality. A high index of suspicion is necessary to avoid diagnostic delays. Serious injuries to other passengers are evidence of the violence of the accident, as is abdominal wall bruising.

Patients at risk should be admitted to hospital and regular observations and serial examinations performed. A variety of further investigations including minilaparoscopy may be helpful.

¹ Howdieshell TR, Delaurier G. An unusual injury of the sigmoid colon produced by seat belt trauma. *Am Surg* 1993;59:355-8.

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 nitrites 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.

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The use of volatile nitrites 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 83%.

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 vasoconstriction. 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

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showed pH 7.1, P_{O_2} 56 kPa, Sa_{O_2} 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 methaemoglobinaemia 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. "Liquid Gold" is isobutyl nitrite, sold as a "room odouriser".¹ 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 use³ and tracheo-bronchitis and haemolytic anaemia have been reported.

Methaemoglobin is formed by oxidation of ferrous (Fe^{2+}) haem to ferric (Fe^{3+}), 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 methaemoglobinaemia include nitrates and nitrites, aniline dyes, chlorate salts, nitrobenzene, and the drugs benzocaine, chloroquine, dapsone, 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 Fe^{3+} 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 leucomethylene 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 cyanmethaemoglobin. In this situation methylene blue frees the bound cyanide. Glucose-6-phosphate deficiency is a relative contraindication, as methylene blue may induce haemolytic anaemia.⁴

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.^{5,6}

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.^{7–9} 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.

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