Fatal methaemoglobinaemia in a dental nurse. A case of sodium nitrite poisoning

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SUMMARY. Methaemoglobinaemia may be caused by ingestion of a number of drugs, among them nitrites, some of which are remarkably toxic. This was tragically highlighted by the death of a 17 year old dental nurse after taking a single 1 g tablet of sodium nitrite. These tablets are widely used in the medical and dental profession to prevent rusting of instruments while immersed in disinfectant solutions. The toxicity of this chemical should be more widely recognized and its storage made correspondingly more secure. Methaemoglobinaemia should be considered as a diagnosis in any patient with significant central cyanosis in whom there is no obvious cardiorespiratory cause.

Case history

MISS H, a 17 year old dental nurse, was admitted to the casualty department at 18.30 hours after being found at home in an hysterical condition and bright blue. On admission she had central cyanosis, tachycardia and tachypnoea. Her chest was clear and she had a systolic blood pressure of 90 mmHg. No history was obtainable from her but her boyfriend reported that she had been fit and well earlier in the day and that he had not seen any evidence of drug ingestion at her house.

Fifteen minutes after admission she vomited, aspirated and suffered a respiratory arrest. She was intubated and ventilated. Despite her cyanosis her arterial blood gases were normal. Apart from recent aspiration there was no other indication that her cyanosis was of cardiorespiratory origin. A venous blood sample was noted to be chocolate brown in colour. Methaemoglobinaemia was suspected and spectroscopy of a blood sample confirmed 'severe methaemoglobinaemia'. Forty five minutes after admission, methylene blue was administered intravenously (2 mg kg^{-1} over 10 minutes). However, by 19.30 hours her blood pressure was unrecordable, a series of cardiac arrythmias ensued and, despite the insertion of a temporary pacing wire together with standard resuscitation measures, she failed to recover. She was certified dead at 20.30 hours, two hours after admission.

Postmortem findings confirmed a normal cardiorespiratory system apart from evidence of minor aspiration into the upper lobe of the right lung. Biochemical analysis after death revealed that the level of methaemoglobinaemia was 35%, implying a much higher level on admission to hospital. The serum nitrite ion level was 13 mg l^{-1}. This level is consistent with death from nitrite poisoning following the ingestion of a single 1 g tablet of sodium nitrite.

Discussion

The haemoglobin molecule is made up of four haem molecules each of which contains one iron atom in the Fe(II) state. Methaemoglobinaemia is caused by oxidation of one or more of the iron atoms to the Fe(III) state. Oxidation of a single haem changes the conformation of the haemoglobin molecule such that the oxygen affinity of the remaining three haem molecules is increased. The oxygen dissociation curve of methaemoglobin is shifted to the left compared with that for haemoglobin, resulting in impaired oxygen unloading to the tissues. The clinical effect of 30% methaemoglobinaemia is therefore much greater than an equivalent reduction in haemoglobin level. In more severe cases of acquired methaemoglobinaemia a haemolytic anaemia may ensue. A characteristic feature of methaemoglobinaemia is the chocolate brown appearance of whole blood which is said to be present at methaemoglobin concentrations as low as 15–20%.

Acquired methaemoglobinaemia is caused by ingestion of or exposure to various oxidant drugs or chemicals. Chronic exposure to a low dose of oxidant may produce minimal methaemoglobinaemia and no symptoms whereas the acute ingestion of the same total dosage may be lethal. Treatment of chronic acquired methaemoglobinaemia involves withdrawal of the offending drug or chemical. If continued therapy is indicated then the dose of oxidant to which the patient is exposed should be reduced. Acute methaemoglobinaemia, if life threatening, should be treated with oxygen and intravenous methylene blue; both reduce levels of methaemoglobin. If there is intravascular haemolysis then plasmapheresis or exchange transfusion is indicated.

The list of drugs which cause methaemoglobinaemia is long. It includes organic and inorganic nitrites, nitrates, local anaesthetics, dapsone, aniline dyes and sodium chlorate. Numerous cases of infant methaemoglobinaemia have resulted from consumption of rural well water high in nitrates which are converted into nitrites in the gut. Amyl or butyl nitrite is often abused as an inhalation intoxicant and to heighten orgasm and several cases of severe poisoning and deaths have been reported.[1-5]

Serum nitrite levels in cases where death has followed accidental or intentional ingestion of sodium or potassium nitrite have varied from 0.5 to 340 mg l^{-1}.[6,7] Serum nitrite levels vary widely between individuals who have ingested roughly equivalent doses of the same nitrite preparation. There are several reported cases of fatal sodium or potassium nitrite overdose in which the chemical has been identified in gastric contents but not in other body fluids or tissues.[8-12] The correlation between serum methaemoglobin concentrations and symptoms is more consistent. Cyanosis is detectable at serum methaemoglobin concentrations of 15%, 40% causes severe headache with weakness and ataxia while levels above 70% will cause severe cyanosis, tachycardia and dyspnoea with the risk of cardiovascular collapse and death.[13]

Sodium nitrite tablets (1 g) are supplied as an adjunct to disinfectant fluids such as chlorhexidine. When dissolved in the fluid sodium nitrite acts as a strong oxidizing agent preventing rusting of the instruments being disinfected. The label on a sodium nitrite container indicates that the material is toxic by ingestion both in text and using the standard symbol of a skull and crossbones in black on an orange background.

In the case just cited the dental nurse almost certainly obtained the tablet(s) from the practice in which she was employed. They were routinely left by the side of the disinfectant bath in which they were used. Furthermore, she probably took only one tablet; this being enough to produce a serum nitrite level that was lethal despite active treatment. The toxicity of sodium nitrite

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Submitted: 14 November 1989; accepted: 26 June 1990.

tablets is not widely known, despite their widespread use in the medical and dental professions. If they are to be kept on surgery premises at all then secure storage must be guaranteed.

This case serves to remind general practitioners to consider methaemoglobinaemia as a diagnosis in any patient with significant central cyanosis in whom there is no obvious cardiorespiratory cause.

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