Methaemoglobinaemia due to accidental 
sodium nitrite poisoning

Report of 10 cases

ANNA KAPLAN, C. SMITH, D. A. PROMNITZ, B. I. JOFFE, H. C. SEFTEL

Summary

Nitrites and nitrates are widely used in the food and chemical industry. Poisoning with these agents may be potentially life-threatening as a result of the production of methaemoglobin. A group of 10 patients suffering from moderate to severe methaemoglobinaemia after accidental intoxication with a sodium nitrite salt is described. One patient died but the other 9 recovered rapidly. The low mortality rate was attributed to prompt diagnosis and institution of appropriate therapy with methylene blue and ascorbic acid.

The occurrence of methaemoglobinaemia may be more common than is generally accepted. It may be caused by exposure to and absorption of numerous drugs and chemicals, including nitrites, nitrates and aromatic amines, the latter being widely used in the chemical industry. Awareness of this condition may therefore be life-saving in both accidental and deliberate poisonings.

A group of 10 patients with moderate to severe methaemoglobinaemia as a result of accidental intoxication with a nitrite salt, similar in appearance to table salt, is described. As far as we are aware, this is the largest group of patients with accidental methaemoglobinaemia to be documented.

Case reports

Ten previously healthy adults (9 men and 1 woman, age range 20 - 50 years) were referred to Hillbrow Hospital casualty department with suspected food poisoning. Pickling salt, mistaken for table salt, had been sprinkled over a meat and cabbage meal in a hostel. The majority of approximately 30 diners became ill 1 - 2 hours after the meal, but only 10 patients were sent to hospital. One patient died in the casualty department and the remaining 9 were sent to the medical admissions ward.

All patients were tachypnoeic and cyanotic. Additional features were: altered level of consciousness; hypotension; and tachycardia. One patient required immediate intubation and mechanical ventilation. A striking feature was the persistence of cyanosis despite oxygen therapy. However, arterial blood gas estimations revealed abnormally high partial pressures of oxygen. This important clinical information led the ward staff to suspect the presence of methaemoglobinaemia. This was confirmed spectrophotometrically in 2 of the most severely ill patients who showed levels of 79% and 71% respectively. Other routine haematological and biochemical blood tests were normal. Blood screening tests for other toxins were negative.

Immediate gastric lavage was performed, with continued supportive care given to all patients. All patients were treated with 1% methylene blue solution in a dose of 1 mg/kg and ascorbic acid 1000 mg intravenously. The 2 patients most seriously affected required a repeat dose of methylene blue after 2 hours. All patients improved dramatically and there were no after-effects.

On analysis, the pickling salt was found to be sodium nitrite.

Discussion

Acquired methaemoglobinaemia may be caused by ingestion or absorption of drugs and chemicals, commonly sodium nitrite. Nitrite is used as a curing agent for the preservation of meat and fish, acting simultaneously as a colouring agent. Symptoms of poisoning may be expected after nitrite ingestion of 0.4 mg/kg body weight or > 200 mg nitrites. These symptoms include cyanosis without other symptoms or euphoria, flushed face, headache, dizziness, ataxia, followed by dyspnoea, tachycardia and cyanosis.

Accidental nitrite or nitrate poisoning has been known for many years. The largest series published to date is that of 7 patients, all of whom survived. Our series of 10 patients, with 1 fatality, thus appears to be the largest documented.

Methaemoglobinaemia is formed when the iron in haemoglobin, normally in the ferrous state, is oxidised to the ferric state, with resultant loss of oxygen-combining capacity. Normally 99% of haemoglobin iron is in the reduced state, existing in equilibrium with the 1% oxidised compound in the red blood cell.

Methaemoglobinaemia may be suspected when a cyanosed patient fails to respond to oxygen therapy, particularly in the absence of severe cardiopulmonary disease. A blood sample, when shaken in an attempt to reoxygenate it, will not turn a brighter red colour but remain brown-red when methaemoglobin exists. This diagnosis is supported by the chocolate-brown colour of a drop of the patient's dried blood and it may be confirmed by spectrophotometric measurements. It should be noted that persons with glucose-6-phosphate dehydrogenase deficiency are more susceptible to poisoning resulting from methaemoglobinaemia.

The major mechanism for maintaining haemoglobin in the reduced state is via a nicotinamide-adenine dinucleotide phosphate (reduced form) (NADPH)-dependent methaemoglobin reductase pathway. This is not amenable to medical intervention. Another mechanism, inert physiologically, utilises
The enigma of granuloma inguinale in South Africa

A. L. FREINKEL

Summary
Granuloma inguinale (donovanosis) was well described in South Africa in the early part of this century. After 1927 no further reports appeared and with the passage of time, the belief grew that the disease did not occur in this country. This belief held until the 1980s, when several reports of cases of the disease appeared. What happened to the disease in the intervening half century? Did it disappear spontaneously? Or was it just not being recognised even when cases were being reported from 1950 onwards in neighbouring Zimbabwe? How is it that a disease can remain unrecognised when, untreated, it can relentlessly progress to cause severe disfigurement?

REFERENCES

Department of Anatomical Pathology, School of Pathology, South African Institute for Medical Research and University of the Witwatersrand, Johannesburg

A. L. FREINKEL, M.B. B.CH., F.F.PATH. (S.A.)

Accepted 21 June 1989.

Granuloma inguinale is a chronic superficial ulcerative disease of the genitalia that occurs in both sexes. It is of low infectivity and the sexual partners of patients with the disease do not always contract the disease. It occurs among the lower socioeconomic strata and is probably related to poor personal hygiene. The lesions are relatively painless and some patients may have the disease for several months or even years before seeking medical attention (Fig. 1). With recurring cycles of ulceration following partial healing, there can eventually be extensive fibrosis with disfigurement. Extranatal disease is said to occur in 6% of cases.

Since 1980 the author has collected 142 cases of donovanosis, all resulting from which were diagnosed antemortem by clinical examination. Earlier findings were reported in 1983,1 and 1984.2 The majority of these cases have been in the eastern Transvaal centered around Shongwe, Malelane and Barberton. Among these 142 cases were 2 patients who presented with extragenital donovanosis of cervical lymph nodes clinically simulating tuberculous lymphadenitis.3 From the experience gained in differentiating these from other genital lesions, the histological features of sexually transmitted genital lesions were reviewed in 1987.4

The first mention and clinical description of granuloma inguinale is generally accredited to McLeod in India in 1882.

The authors wish to thank all the staff involved in the care of these patients.