Case 2—Another woman, aged 73, had longstanding severe osteoporosis. She also complained of dysphagia and dyspepsia. Radiological investigation showed multiple compressed vertebrae, appreciable kyphosis, and profound osteoporosis and also noticeable hold-up of barium in the lower oesophagus owing to a tortuous aorta with the reversed C configuration.³ There was mild left ventricular enlargement. She was normotensive.

The remaining four patients all complained of dysphagia, and similar hold-up of barium by a tortuous aorta was noted on radiological investigation. In addition three of the four showed kinking obstruction similar to that seen in case 1; in one patient this was more severe than the original compressive obstruction. Three of the four patients had evidence of left ventricular enlargement. Two were hypertensive. One patient died from inanition owing to complete oesophageal obstruction. Oesophageal compression by the aorta was confirmed as the only abnormality post mortem.

X-ray film obtained on barium swallow (case 1) showing reversed S configuration of aorta compressing mid and distal oesophagus and also secondary kinking obstruction of upper oesophagus.

Comment

Twenty-one patients, including our own, have now been described with this condition. Most are elderly women with a long history of dysphagia or anorexia and often weight loss.

The aortic unfolding that causes oesophageal compression is seen on barium studies to fall into two clear categories—that is, the reversed S and reversed C configurations.³ The reversed S aorta is likely to compress the oesophagus in its mid and lower thirds and the reversed C only in the lower third. Four of our patients showed the reversed C and two the reversed S aorta.

Because of the oesophageal compression oesophageal dilatation and then elongation occur. The elongation introduces a kinking obstruction which usually occurs just below the aortic arch and may be more severe than the original compressive obstruction.

Why this condition predominates in women and whether aortic unfolding is more common in women are not known; while kyphosis (which may contribute to the condition) resulting from osteoporosis is more common in women, it was not present in all of our patients. Hanna and Derrick suggested that the oesophageal obstruction is due to compression between the unfolded aorta and an enlarged left ventricle⁴; left ventricular enlargement may well aggravate the condition but is clearly not an essential feature since it was absent in one of our patients.

Prognosis and treatment of the condition vary. One of our patients was subsequently untroubled by symptoms, one was satisfactorily treated with and one was trying a semi-fluid diet, and another died of inanition. The fate of the other two was not known. The course in previously reported patients has also varied.²⁻⁵ Patients have been asymptomatic, had slight or intermittent dysphagia, or shown severe oesophageal obstruction often with accompanying oesophagitis. Treatment for the condition varies with severity and includes a semi-fluid diet and oesophagoscopy or bougienage, or both. In the most severe cases surgical repositioning of the oesophagus has been reported to relieve the compression and symptoms completely.⁵

Recognising this condition is important as it is a remediable cause of dysphagia, anorexia, and weight loss and, though usually more likely to cause mild rather than severe symptoms,⁵ is nevertheless potentially fatal, causing aspiration pneumonia and inanition. We suggest that it is not uncommon in elderly women and if it was recognised more widely than at present many of the ensuing symptoms might be avoided by using a fluid or semi-fluid diet and resorting to bougienage or surgical relief of the compression in those unhelped by diet.⁵

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Multisystem disorder after exposure to paint stripper (Nitromors)

Nitromors is a household and industrial paint stripper whose active ingredient is methylene chloride (CH_2Cl_2). It is reputedly harmless in ordinary use, but methylene chloride is a halogenated hydrocarbon and these compounds are a recognised cause of hepatic and renal damage.¹ Inhalation of methylene chloride vapour may cause pulmonary oedema² and depression of the central nervous system.³ We report a serious multisystem disorder that followed exposure to Nitromors and was complicated by the development of temporary diabetes mellitus.

Case history

A previously fit 25-year-old accountant used Nitromors in a confined space for three to four hours, taking the recommended precautions to avoid skin contact. He suffered no immediate side effects except for mild conjunctival irritation, and returned to work the next day. Forty-eight hours after exposure he felt generally unwell, with swollen and stiff ankles, knee, and wrist joints, and over the next 24 hours he developed a pink, profuse, non-itchy rash. During the next eight days he noticed increasing difficulty with simple intellectual tasks and an inability to concentrate and his work deteriorated; he developed swelling of the face and shortness of breath on minimal exertion. Ten days after exposure he became increasingly thirsty and complained of nocturia, polyuria, and weight loss of 2.7 kg.

On admission he had stiffness of the fingers, wrists, and ankles and a profuse pink nodular rash, parafollicular in distribution, limited to the upper half of the body. Although he was conscious and fully orientated, his concentration was poor and mental arithmetic impaired; his long-term memory was intact but immediate recall poor. There was pitting oedema of both ankles. Jugular venous pressure was raised by 2 cm H_2O , pulse was 98 beats/min and blood pressure 160/110 mm Hg, and a third heart sound was present. Urinary examination showed 2% glycosuria and a few red blood cells per high-power field on microscopy. Laboratory results included normal urea and electrolyte concentrations, a random blood glucose concentration of 12.1 mmol/l (218 mg/100 ml), bilirubin 27 nmol/l (1.6 $\mu\text{g}/100\text{ ml}$), alkaline phosphatase 170 King Armstrong units/l, alanine aminotransferase 47 IU/l, and normal serum protein, creatinine, and calcium concentrations and creatinine clearance. Electrocardiography showed widespread T-wave inversion, and a chest x-ray film showed cardiomegaly with upper-lobe blood-vessel diversion. Virological studies were negative and coxsackie B antibodies not detected.

The rash (a perifollicular papulosis) was treated with only local applications; six months after exposure it had improved but had not completely cleared. His mild arthropathy flared up three months after exposure with pain, swelling, and stiffness in the fingers, wrists, ankles, and knees. Simultaneously the erythrocyte sedimentation rate rose to 129 mm in the first hour but results of Rose-Waaler and latex tests remained negative; this episode settled with mild analgesics in 10 days. Although formal testing showed mental clearing within a few weeks, he could not work as an accountant for six months. The cardiac failure was treated satisfactorily with diuretics and the electrocardiographic changes returned to normal within two weeks of admission. His diabetes needed treatment with glibenclamide and metformin together with diet; after six weeks this treatment was reduced and by six months he needed no hyperglycaemic treatment.

Comment

Acute poisoning by large quantities of Nitromors reportedly causes an initial metabolic upset and unconsciousness with no long-term sequelae.⁴ Chronic exposure has caused cerebral damage probably by the production of endogenous carbon monoxide.⁵ Our patient clearly had mental impairment after one exposure but also had an unusual and severe multisystem disorder. Although several chemical agents cause β -cell destruction, none of the constituents of Nitromors is recognised as causing diabetes in man. As our patient developed temporary diabetes as part of a multisystem disorder, however, Nitromors should be added to the list of chemicals causing diabetes mellitus.

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Exaggerated hypokalaemia in acute myeloid leukaemia

When blood is stored plasma potassium concentration falls.¹ As glycolysis ceases erythrocyte sodium-potassium adenosinetriphosphatase becomes inactive and the expected rise in plasma potassium concentration occurs. We describe a case of acute myelomonocytic leukaemia in which these events were exaggerated, presumably by the metabolically active leukaemic cells, leading to a dramatic fall in plasma potassium concentration in blood stored at room temperature. This suggested that the patient was profoundly hypokalaemic, but the true plasma potassium concentration was only slightly below normal.

Case report

A 60-year-old woman was admitted to hospital suffering from acute myelomonocytic leukaemia. Initial blood count showed Hb 9.4 g/dl, white blood cells $254 \times 10^9/l$, and platelet count $70 \times 10^9/l$. Peripheral blood film showed 92% blast cells with myelomonocytic features, and bone-marrow examination confirmed the diagnosis of acute myelomonocytic leukaemia. Serum lysozyme concentration was 33.1 mg/l (normal <20 mg/l) while urinary lysozyme was 17.2 mg/l (normally undetectable). An electrocardiogram was consistent with mild hypokalaemia. Plasma potassium concentration on admission was reported as 0.9 mmol(mEq)/l. This was measured about an hour after venepuncture, blood having been taken into standard glass tubes containing 150-250 units of lithium heparin. Sodium concentration was 131 mmol(mEq)/l, bicarbonate 32 mmol(mEq)/l, chloride 84 mmol(mEq)/l, urea 7.4 mmol/l (44 mg/100 ml), and creatinine 104 μ mol/l (1.2 mg/100 ml). In a repeat sample, analysed within 15 minutes of venepuncture, the plasma potassium concentration was 2.9 mmol/l.

Treatment was started with daunorubicin 25 mg intravenously on day 0, followed by cytarabine 50 mg intravenously 12-hourly for five days and thioguanine 40 mg by mouth 12-hourly for five days. She was febrile, and ticarcillin 5 g eight-hourly intravenously, gentamicin 80 mg eight-hourly intravenously, and metronidazole 400 mg eight-hourly by mouth were also administered. The next day the plasma potassium concentration was reported as 1.0 mmol/l in blood sent routinely to the laboratory and analysed about an hour after sampling, and 3.5 mmol/l in a repeat sample assayed within 15 minutes of venepuncture. We therefore examined the relation between plasma potassium concentration and the interval between venepuncture and separation of plasma.

Blood was taken into the same type of standard lithium heparin tubes as before and stored at room temperature and 4°C. Aliquots were analysed for plasma potassium concentration (flame photometer) at 15-minute intervals, after separation by centrifugation at 8850 rpm for one minute. The first sample was analysed within five minutes of venepuncture. At room temperature plasma potassium concentration fell appreciably and rapidly (figure).

Change in plasma potassium concentration with time after venepuncture.

—○— = White cell count $220 \times 10^9/l$, more than 90% blasts, initial potassium concentration 2.5 mmol(mEq)/l, at room temperature. --●-- = White cell count $110 \times 10^9/l$, 90% blasts, initial potassium concentration 3.2 mmol/l, at room temperature. - -■ - - = White cell count $2.1 \times 10^9/l$, no blasts, initial potassium concentration 4.2 mmol/l, at room temperature.○.... = White cell count $220 \times 10^9/l$, more than 90% blasts, initial potassium concentration 2.5 mmol/l, at 4°C. Shaded area shows mean $\pm$ 2SD values at room temperature in five normal people.

Conversion: SI to traditional units—Potassium: 1 mmol/l = 1 mEq/l.

At 4°C the concentration rose rapidly. There was no change in potassium concentration in plasma separated immediately after venepuncture and stored at room temperature.

As the white cell count decreased with treatment the fall in potassium concentration became less pronounced. Ten days after the start of treatment (white cell count $2.1 \times 10^9/l$) no fall occurred. In samples of blood from five normal volunteers a slight (0.2 mmol/l) fall in plasma potassium concentration over two hours was observed, as expected¹ (figure).

Comment

Hypokalaemia is a common complication of acute myeloid leukaemia and has been ascribed to renal tubular loss of potassium,² increased uptake of potassium by the active leukaemic cell mass,³ and treatment with some antibiotics.⁴ Our patient was mildly hypokalaemic on admission, as shown by the electrocardiographic changes and "rapid" plasma potassium measurement. The hypokalaemia was grossly exaggerated, however, by a sharp fall in plasma potassium concentration, which occurred after blood had been taken. This led us to give inappropriate parenteral potassium replacement until the true explanation became apparent.

Possibly the observed effect was due to increased uptake of potassium by leukaemic cells. When an aliquot of plasma was incubated with buffy coat alone similar changes occurred. The in-vitro fall in potassium concentration disappeared as the total leucocyte count fell to normal. Our finding of temperature dependence would be compatible with an energy-dependent process, and increased leucocyte sodium-potassium adenosinetriphosphatase has been reported in leukaemia.⁵ We suggest that to avoid potentially dangerous and