The rash (a parafollicular papillosis) 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

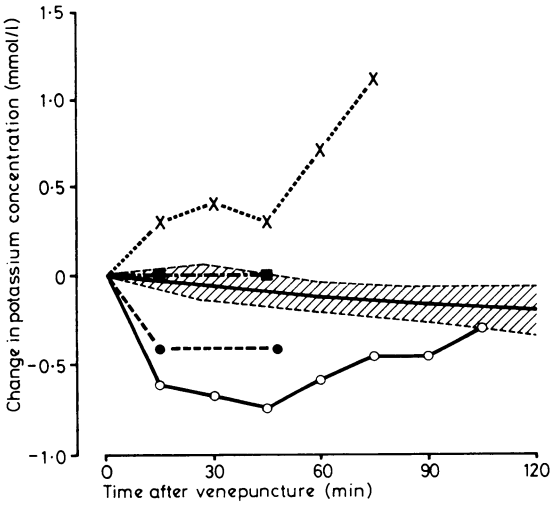
When blood is stored plasma potassium concentration falls.[1] As glycolysis ceases erythrocyte sodium-potassium adenosinetriphosphatase becomes inactive and the expected rise in plasma potassium concentration occurs. We describe a case of acute myelomonocytic leukaemia in which these events were exaggerated, 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 254×10^9/l, and platelet count 70×10^9/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 μmol/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. —o— = White cell count 220×10^9/l, more than 90% blasts, initial potassium concentration 2·5 mmol(mEq)/l, at room temperature. - - ● - - = White cell count 110×10^9/l, 90% blasts, initial potassium concentration 3·2 mmol/l, at room temperature. - - ■ - - = White cell count $2{\cdot}1 \times 10^9$/l, no blasts, initial potassium concentration 4·2 mmol/l, at room temperature. · · · · × · · · · = White cell count 220×10^9/l, more than 90% blasts, initial potassium concentration 2·5 mmol/l, at 4°C. Shaded area shows mean ±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 $2{\cdot}1 \times 10^9$/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 expected[1] (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

inappropriate treatment plasma potassium estimations in patients with acute myeloid leukaemia should be made on plasma separated immediately after venepuncture.

We thank Dr M Qureshi for permission to study his patient and Professor M D Rawlins for his help and advice.

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Brain abscess due to Arachnia propionica

Arachnia propionica, a member of the Actinomycetaceae, has rarely been described as an agent of human infections. We report the isolation of this organism from a brain abscess and emphasise the problems associated with its identification.

CASE REPORT

A 32-year-old man presented with a two-day history of severe central headache not relieved by the usual analgesics, falling to the left when walking, and spatial visual difficulties on looking to the left. His medical history indicated that he had developed Eisenmenger's syndrome as a result of an inoperable ventricular septal defect, confirmed by cardiac catheterisation in 1952. During the procedure a left cerebrovascular accident had occurred, which had resulted in continuing convulsions, right-sided hemiparesis, and growth retardation. He had been started on prophylactic phenoxymethylpenicillin 250 mg twice daily.

Four months before presentation he had been admitted to hospital for treatment and control of cardiac failure. Shortly after discharge he stopped the penicillin prophylaxis. On readmission he was subjected to a full examination and special investigations. A computed tomography scan showed generalised atrophy of the left cerebral hemisphere and a lesion in the right occipital lobe compatible with an abscess. No primary focus of infection was found. He was started on intravenous ampicillin 2 g four times daily and gentamicin 80 mg and metronidazole 500 mg thrice daily and underwent craniotomy, when an abscess close to the medial surface and above the calcarine sulcus of the right occipital lobe was drained and the wall excised.

Pus was cultured aerobically on blood agar, CLED agar, and heated blood agar, and anaerobically on blood agar and a supplemented lysed blood agar containing gentamicin 5 mg/l, cysteine hydrochloride 0·05%, yeast extract 0·5%, and vitamin K_1 0·0002%. Microscopy showed numerous leucocytes, Gram-positive cocci, and branching Gram-positive bacilli. After 48 hours' incubation results of aerobic culture were negative while anaerobic culture produced an anaerobic Gram-positive coccus subsequently identified as *Peptostreptococcus micros* and a branching anaerobic Gram-positive bacillus identified as *A proprionica*. Identification of isolates was based on morphological studies, routine biochemical tests, and analysis of the end products of glucose fermentation as described by Holdeman *et al.*[3] He was treated with intravenous penicillin and metronidazole and discharged three weeks later taking oral penicillin.

Comment

A propionica isolations have usually been associated with lesions of the cervical lymphocutaneous tissues, lacrimal glands, lung, abdomen,[2] and bone.[3] We have found no reports of a brain abscess due to this organism. *A propionica* is a resident of the oropharynx, as is *P micros*, and the organisms isolated from the abscess probably originated from this site despite the absence of oral disease.

Identification of *A propionica* in the microbiology laboratory can be difficult. Morphologically and biochemically the organism is similar to *Actinomyces israelii* and clinically may produce a disease that is indistinguishable. Three tests are available to differentiate the two organisms—namely, the demonstration of diaminopimelic acid in the cell wall of *A propionica*, specific fluorescence antibody staining, and the demonstration of the production of propionic acid by gas-liquid chromatography from glucose fermentation by *A propionica*.[2] As the first two tests are beyond the scope of most clinical laboratories, the importance of carrying out gas-liquid chromatography analysis of a glucose broth for all organisms resembling *Actinomyces* cannot be overemphasised.

Both organisms isolated from the abscess were sensitive to penicillin. Hence an important question is raised regarding the use of prophylactic antibiotics, in this case phenoxymethylpenicillin, in patients with cardiac anomalies, either congenital or acquired. Should the patient take subtherapeutic dosages of prophylactic antibiotics for long periods and risk acquiring resistant oral microflora or stop prophylaxis and risk infection with commensal micro-organisms?

In our patient oral prophylaxis with phenoxymethylpenicillin was justified as we assume that a bacteraemic episode had occurred a few weeks before presentation while he was not taking antibiotics. Although it is recommended that prophylaxis is indicated only when a patient is subjected to some manipulative procedure known to cause bacteraemia,[4] phenoxymethylpenicillin had been continued in our patient, albeit at subtherapeutic dosages, after he was discharged, in an attempt to prevent a further bacteraemic episode.

We thank Dr M Lee for permission to report this case, and Dr K T Karthigasu for helpful discussion in preparing the manuscript.

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Legionnaires' disease and abscess of appendix

While gastrointestinal symptoms, particularly diarrhoea and abdominal distension, are common in legionnaires' disease,[1] a surgical "acute abdomen" has not previously been reported in this condition. I report on a patient with legionnaires' disease complicated by an abscess of the appendix.

Case report

A 48-year-old engineer was admitted to this hospital with a seven-day history of headache, rigors, and fever. Three days before admission he had developed an unproductive cough without chest pain or dyspnoea; treatment with co-trimoxazole had been started. The next day he had developed central abdominal pain, which had worsened and localised in the right iliac fossa. He had been anorexic and was constipated. He had become ill on the day he returned from a 10-day holiday in Benidorm.

On examination he was seen to be feverish and toxic. He was tachypnoeic with clinical signs of a left basal pneumonia. There was a tender, 3-cm-diameter mass palpable in the right iliac fossa, with guarding and rebound tenderness.

Haemoglobin concentration was 15·9 g/dl and white cell count 10×10^9/l