extremely sensitive to this concentration, formal measurement of the minimum inhibitory concentration and minimum bactericidal concentration was not performed. Streptomycin was stopped. Twenty-four hours after the start of this regimen the rigors had stopped and the temperature returned to normal. Intravenous penicillin was continued for two weeks and she was discharged home taking oral penicillin 1 g four times a day for a further six weeks. One month after discharge she was well, without any changes in the cardiovascular examination or echocardiographic findings.

Comment

While the dangers of endocarditis in susceptible patients undergoing oral or genitourinary manipulation are well recognised,^{3,4} scant attention has been given to bacteraemia that may follow compression of affected foci such as boils or epithelial damage of many other kinds.⁵ In Western society electrolysis is a common method of depilation and no consideration is given to any possible serious complications. This report emphasises that the damaged heart valve is at risk from any procedure that may produce bacteraemia. Susceptible patients should be alerted to this possible hazard of electrolysis and consideration should be given to appropriate prophylaxis.

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Oral or parenteral iron treatment in chronic ulcerative colitis?

Iron-deficiency anaemia is a common complication of ulcerative colitis, but studies of iron absorption in this disease¹⁻³ have yielded conflicting results and hence differing guidelines on the best route for iron replacement. The aims of our study were therefore to establish whether iron-deficient patients with chronic colitis have an enhanced absorption of inorganic iron comparable with that found in other iron-deficient patients, and to assess the haematological response to, and the acceptability of, oral iron treatment in patients with colitis.

Subjects, methods, and results

We studied three groups of subjects: seven normal male volunteers, 10 iron-deficient controls without inflammatory bowel disease, and 13 iron-deficient patients with chronic active ulcerative colitis. Iron deficiency was diagnosed on the basis of hypochromic microcytic anaemia with low transferrin saturation, low serum ferritin concentration, and reduced or absent bone-marrow iron stores.

The absorption of inorganic iron-59 (⁵⁹Fe) was determined in all subjects using a whole-body counter⁴ and expressed as the percentage of ⁵⁹Fe given by mouth retained at 10-14 days. The normal volunteers retained 12.3 ± 9.3% (mean ± 1SD), the iron-deficient controls 66.7 ± 30.9%, and the iron-deficient patients with colitis 66.0 ± 26.5%. There was no significant difference in the mean absorption of iron between the two groups of patients, while both groups absorbed significantly more than the controls (p < 0.001; Student's unpaired t test).

Ten of the 13 patients with colitis were subsequently treated with oral iron supplements for up to eight months. Of the remaining three, one, the only inpatient in the study, underwent elective proctocolectomy, one defaulted from follow-up, and one was only mildly iron deficient. Of the 10 treated patients, eight responded completely, their mean haemoglobin concentration rising from 10.4 to 13.4 g/dl and mean corpuscular volume from 73 to 83

fl. One patient had no response despite enhanced absorption of ⁵⁹Fe, but he had persistent active disease with bloody diarrhoea. The remaining patient showed only a partial response, her haemoglobin concentration rising from 5.9 to 9.5 g/dl, until after a hysterectomy for coexistent menorrhagia.

Seven patients tolerated oral iron preparations without ill effects. Two patients attributed mild constipation or diarrhoea to their tablets, and a further patient stopped ferrous gluconate after six weeks because of nausea but subsequently took slow-release ferrous sulphate without symptoms.

Comment

Beal *et al*¹ measured the absorption of ⁵⁹ferrous sulphate in five iron-deficient patients with colitis including four inpatients: in three absorption was above the normal range. Subsequently Ormerod,² measuring ⁵⁹Fe concentrations in blood after oral administration of ⁵⁹ferrous ascorbate, showed impairment of iron absorption in 11 patients with colitis who were sufficiently ill to require inpatient treatment. He therefore proposed that iron should be replaced parenterally in such patients, although in practice they usually receive transfusions. In a third study of iron absorption in inflammatory bowel diseases Bartels *et al*³ using external whole-body counting, found normal or increased absorption of inorganic iron in nine iron-deficient patients with ulcerative colitis. Paradoxically their patients responded poorly to oral iron treatment, and these authors also recommended parenteral iron replacement.

The present study shows that iron-deficient patients with active colitis who are not acutely ill absorb inorganic iron as avidly as other iron-deficient subjects. Thus repletion should be as effective by mouth as parenterally and without any of the hazards and discomfort associated with intravenous or intramuscular administration. The good response to treatment in eight of our 10 patients with colitis confirms this and shows unimpaired use of iron by these patients. Moreover, they generally tolerated iron tablets well: side effects could as easily have been due to the colitis itself as to iron treatment.

We conclude that iron-deficiency anaemia in outpatients with chronic ulcerative colitis should be treated with oral iron, which is well absorbed, acceptable, safe, and effective.

We thank Miss Barbara Jones, principal physicist in the department of nuclear medicine, Charing Cross Hospital, and Dr Sheila Callender for their help and advice with this study.

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Liver and spleen candidiasis: imaging and verification by fine-needle aspiration biopsy

Patients receiving immunosuppressive agents are at risk of infection, and with improved treatment of leukaemia and lymphoma and widespread use of organ transplantation more such patients are being seen. Diagnosing disseminated fungal invasion is difficult before death,¹ but aspiration biopsy with microscopy and culture may be successful.^{1,2}

We report candidiasis of liver and spleen that occurred in a patient with acute leukaemia. The final diagnosis was established by sonographically controlled fine-needle aspiration biopsy of a liver lesion.

Case report

A 35-year-old woman was admitted for relapse of acute lymphoblastic leukaemia (FAB grade L 2). Full remission had been achieved after 28 months' treatment with OPAL (vincristine, prednisone, doxorubicin, and L-asparaginase (colaspase)). The relapse proved resistant to OPAL, and high-dose combination chemotherapy with TACC (cyclophosphamide 45 mg/kg/day intravenously, cytosine arabinoside (cytarabine) 100 mg/m²/day intravenously in divided dosage 12-hourly, and 6-thioguanine 100 mg/m² by mouth 12-hourly for four days, with lomustine 200 mg/m² by mouth on the second day)³ resulted in bone marrow aplasia. Autologous bone marrow transplantation was therefore performed. The period of total aplasia was complicated by septicaemia (*Klebsiella aerogenes*), which was treated with intravenous broad-spectrum antibiotics (gentamicin 240 mg and cefuroxime 6 g daily for three weeks and (because of poor response) carbenicillin 30 g/day for two weeks). Intercurrent gastrointestinal candidiasis was treated with antimycotic drugs (miconazole 1.5 g/day by mouth and 1.5 g/day intravenously for four days, and 5-fluorocytosine (flucytosine) 10.0 g/day as continuous intravenous therapy).

About three weeks after onset of the septicaemia nausea, vomiting, and right upper abdominal pain appeared. Serum alkaline phosphatase and aspartate and alanine aminotransferase activities remained normal. γ -Glutamyltransferase activity was 83 U/l (normal 3-30 U/l) and conjugated bilirubin concentration 8 μ mol/l (0.5 mg/100 ml) (normal 0-5 μ mol/l (0-0.3 mg/100 ml)). The gall bladder was not opacified on oral cholecystography. Computed tomography disclosed multiple, small (0.5-2.5 cm diameter) non-capsulated lesions of low attenuation (6 Hounsfield units) in liver and spleen (fig (a)). Sonography showed soft-tissue aggregates in some of the lesions in

(a) Tomogram of upper abdomen showing small multiple lesions in liver and spleen (arrowed). (b) Real-time sonogram of right lobe of liver showing cystic lesion containing soft-tissue aggregate (arrowed)—?candidoma.

the liver. Under guidance of real-time sonography the largest of these lesions was punctured with a 0.8 mm needle (fig (b)). Smears of the aspirate stained with May-Grünwald-Giemsa and dried in air contained fungal mycelia and spores. Culture of the main part of the aspirate for bacteria and fungi grew a strain of *Candida albicans*.

5-Fluorocytosine 10.0 g/day intravenously and amphotericin B up to 25 mg/day intravenously produced a poor response, and three months later the patient still had symptoms of disseminated candidiasis with abscesses evident on sonography and computed tomography.

Comment

The candida abscesses in this patient's liver and spleen had the sonographic and radiological appearances of multiple cysts. There was no capsule formation, and the lesions contained little debris. This may be explained by her lowered resistance to infection. In addition, sonography showed aggregates of fungus in some larger lesions. Such candida ball formation has been described in bile ducts.^{4,5} Invasive disseminated fungal infection should be diagnosed as soon as possible in a patient undergoing immunosuppression, since early antifungal treatment may be life saving. The diagnosis, however, is seldom achieved before death.¹

Our findings show that candida microabscesses in liver and spleen may be imaged by sonography and computed tomography. A liver lesion containing a soft-tissue aggregate is suggestive of a candida abscess (candidoma). Under sonographic control biopsy material may be obtained for microscopy and culture.

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Hypokalaemia due to salbutamol overdosage

Salbutamol, a beta₂-adrenergic agonist, is used mainly to treat bronchial asthma but has also been advocated for use in severe cardiac failure, congestive cardiomyopathy, and premature labour. We report a case of self-poisoning with salbutamol associated with pronounced hypokalaemia.

Case report

The patient, a 76-year-old widow, was admitted two hours after taking a total of 40 salbutamol (4 mg) and Distalgesic tablets. On admission she was conscious, agitated, sweating, and tremulous. There was regular tachycardia of 140 beats/min, blood pressure was 90/50 mm Hg, respiratory rate 40/min, and temperature 37.5°C. There was no evidence of muscular weakness. Gastric lavage was performed and debris of pink tablets obtained.

Investigations yielded the following results: plasma sodium 140 mmol(mEq)/l, potassium 2.3 mmol(mEq)/l, chloride 100 mmol(mEq)/l, bicarbonate 27 mmol(mEq)/l, urea 5.7 mmol/l (34 mg/100 ml), and glucose 7.7 mmol/l (139 mg/100 ml). Haemoglobin was 11.7 g/dl with a white cell count of 16.4 × 10⁹/l (neutrophils 94%). Serum paracetamol concentration on admission was 55 mg/l. Plasma renin activity was 0.15 mmol/l/h (0.2 ng/ml/h) (normal supine range 0.77-1.54 mmol/l/h (1.0-2.0 ng/ml/h)) and plasma aldosterone 386 pmol/l (13.9 ng/100 ml) (normal supine range 100-500 pmol/l (3.6-18.0 ng/100 ml)). An electrocardiogram showed sinus tachycardia without the characteristic changes of hypokalaemia.

She was treated with 80 mmol (6 g) potassium chloride in an intravenous infusion of 2 l physiological saline given over 24 hours. A 1.84 l urine collection over this period contained 197 mmol sodium and 59 mmol potassium. One day after admission the plasma potassium concentration had risen to 4.4 mmol/l, and it subsequently remained normal without further supplementation.

Comment

Agitation, tremor, tachycardia, and hypotension are typical features of salbutamol overdosage,¹ and neutrophil leucocytosis is consistent with adrenergic stimulation. The effect of salbutamol on the metabolism of potassium is complex. In common with catecholamines and other sympathomimetic agents, salbutamol directly stimulates transfer of potassium from extracellular to intracellular fluid by activating sodium-potassium-dependent adenosinetriphosphatase. In addition, secretion of insulin, which has a similar effect on the transfer of potassium, is stimulated by salbutamol.² The hypokalaemic effect of intravenous salbutamol in insulin-dependent diabetics and normal subjects, however, is comparable,³ suggesting that insulin may not contribute greatly to the induced hypokalaemia.

Intravenous salbutamol causes a fall in plasma potassium concentration in normal subjects, patients with asthma, and patients