no laboratory tests for hepatitis A virus markers were available, is strongly supported by this case.

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Steatorrhoea induced by allopurinol

Adverse reactions to allopurinol include rashes, renal failure, hepatic damage, hypersensitivity angitis, peripheral neuritis, bone-marrow depression, and cataract formation,¹ but to our knowledge steatorrhoea has not been reported. Neither the manufacturer of the drug nor the Committee on Safety of Medicines has been notified of such an occurrence. Drug-induced steatorrhoea is usually of mild to moderate severity.² We describe a case of severe steatorrhoea induced by allopurinol.

Case report

An 80-year-old farmer was admitted to hospital in June 1981 with diarrhoea and weight loss of three weeks' duration. He had suffered from gout, which had been intermittently treated with allopurinol for the past three years.³ At the age of 78 he had been admitted to hospital for investigation of diarrhoea and anaemia. Gastrointestinal radiography (small bowel not included) had shown no abnormality, and the anaemia had been ascribed to bleeding haemorrhoids. He had noticed that there was a close association between his symptoms and ingestion of allopurinol and stopped the medication himself. He restarted it, however, after a few attacks of gouty arthritis. Colchicine was not prescribed.

Examination showed wasting and pitting ankle oedema. Laboratory results were compatible with malabsorption. Haemoglobin concentration was 10 g/dl. Serum iron, albumin, cholesterol, carotene, vitamin B₁₂, and folic acid concentrations were low. Results of a xylose tolerance test (25 g), Schilling test, and iron-loading test were abnormal. Protein electrophoresis showed a normal pattern. Adrenal and thyroid functions were normal. Serum and urine amylase activity and urinary 5-hydroxyindole-acetic acid concentrations were normal. Faecal fat content was 75 g/24 hours. Barium meal follow-through examination of the small intestine showed dilated loops with mucosal damage, and small-bowel biopsy showed effacement of normal mucosal architecture compatible with adult coeliac disease.

Allopurinol was stopped on admission. A few weeks later, after the biopsy

Clinical course showing association between steatorrhoea, allopurinol, and gluten-free diet.

results were known, a gluten-free diet was prescribed. A noticeable abatement of symptoms occurred during the third week of admission, and bowel movements were reduced to 3 to 4 a day. Severe gouty arthritis necessitated reinstitution of allopurinol, which produced a striking worsening of the diarrhoea, though he obediently adhered to his gluten-free diet. Allopurinol was again discontinued. This was followed by a steady decline in the number of bowel movements. Four weeks later he showed no clinical or laboratory evidence of steatorrhoea. After challenge with allopurinol the diarrhoea recurred accompanied by a generalised rash: both subsided shortly after withdrawal of the drug. He gradually introduced gluten into his diet and the diarrhoea did not return. In April 1982 he was taking a normal diet with large amounts of bread and had one bowel movement a day. Repeat radiography of the small intestine was normal with no laboratory evidence of malabsorption. The figure shows the patient's clinical course.

Comment

The association between allopurinol and the diarrhoea in this case, suggested by the patient himself, was proved unequivocally when on two separate occasions challenge with allopurinol renewed the diarrhoea, which abated shortly after withdrawal of the drug. A gluten-free diet was ineffective whenever allopurinol was resumed, and reintroduction of a normal diet had no deleterious effect on the patient's bowel habits as long as he did not consume allopurinol.

Although the histological findings are compatible with adult coeliac disease, this unique history and follow-up strongly suggest a true case of allopurinol-induced malabsorption.

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Postoperative infection in shunts for hydrocephalus: are prophylactic antibiotics necessary?

Infections are a troublesome complication of cerebrospinal fluid shunt operations for hydrocephalus and can usually be eradicated only by removing the shunt. In this report we regard infection arising within six weeks of an operation for primary insertion or revision of a shunt as a postoperative infection.¹ The reported incidence of postoperative shunt infection varies from 2% to 39%, with an average of 10% to 15%.² Most reports do not state the time relation of the infections to the operations; those reports which do distinguish between early infections, arising within one month of operation, and later infections that arise after one month. About 70% of all shunt infections arise within one month of surgery. Perioperative factors are much more important with early than late shunt infections. We report an incidence of 1.5% postoperative infection in 200 consecutive shunt operations. No patient was given prophylactic antibiotics.

Methods and results

We reviewed 200 consecutive cases of primary shunt insertions or revisions performed between December 1979 and 24 November 1981. No patient was given prophylactic antibiotics. A total of 132 shunts were ventriculo-peritoneal, 55 were ventriculoatrial, 12 were theco-peritoneal, and one was ventriculovesical. Pudenz-Heyer shunts accounted for 198 of the total. The remaining two shunts were Spitz-Holter. Registrars carried out 115 procedures, and senior registrars or consultants the remaining 85. Three postoperative infections occurred, all in operations performed by registrars. The

first was a *staphylococcus albus* infection which arose in a primary ventriculo-atrial shunt inserted in a 60-year-old patient who had developed hydrocephalus in association with a cystic tumour of the posterior fossa. The second was an *Escherichia coli* infection which occurred after a revision of the upper end of a ventriculoperitoneal shunt in a 2-year-old child who had hydrocephalus caused by a Dandy-Walker cyst. The third was a *Staph albus* infection which occurred after revision of the lower end of a ventriculoperitoneal shunt in a 1-year-old child who had presented with non-communicating hydrocephalus at the age of 3 months. The age range of the patients and the number of shunts carried out were as follows: under 1 year, 34; 1 to 10 years, 64; 10 to 20 years, 43; and over 20 years, 59.

Comment

Recent reports suggest that prophylactic antibiotics may prevent shunt infection.^{1,3} Malis⁴ reported a series of 1732 consecutive major neurosurgical operations in which intraoperative prophylactic tobramycin, vancomycin, and streptomycin were given and in which no postoperative infections occurred.⁴ Prophylactic antibiotics are not used in our unit, since they are thought to be of minor prophylactic value and give rise to resistant bacteria and consequently infections which may prove difficult to treat. We consider that careful surgical technique is the single most important factor in the prevention of shunt infection.⁵

Scrupulous surgical technique includes careful planning of incisions to ensure maximum blood supply to the healing tissues, gentle handling of tissue to avoid tissue necrosis, avoiding the overuse of unabsorbable sutures in the wound, and careful prevention of wound haematomas. No shunt infection occurred after operations performed by the experienced members of the neurosurgical team. Furthermore, two of the infections arose after operations performed by one registrar during the first 10 weeks of training. This registrar has since carried out 57 shunt procedures over 12 months with no postoperative shunt infections. We therefore suggest that scrupulous surgical technique alone should be enough to prevent infection after cerebrospinal fluid shunt operations.

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Remission from polymyositis after total body irradiation

Cellular immune mechanisms have been implicated in the pathogenesis of polymyositis. The modulation of these mechanisms by ionising radiation is clinically effective in the treatment of animal models of autoimmune disease and in some patients with rheumatoid arthritis.¹

Case report

A 44-year-old man presented in November 1980 with fever, malaise, and proximal muscle weakness. Polymyositis was diagnosed. Activities of all muscle enzymes were greatly increased and creatinine kinase activity was 2175 IU/l. Muscle biopsy and electromyography confirmed the diagnosis. Respiratory function values were normal. Prednisolone, 60 mg/day, and azathioprine 125 mg/day were instituted.

The patient responded well; within three months he had no weakness and muscle enzyme activities were normal. Steroids were progressively

reduced while maintaining azathioprine. He relapsed for the first time in May 1981 with severe proximal weakness and a creatine kinase activity of 2350 IU/l. Prednisolone was increased to 60 mg/day but on this occasion the response was poor. After three months he was still symptomatic, though muscle enzyme values had fallen. In September he relapsed for the second time, again with severe proximal weakness and creatine kinase activities at their highest value (>5000 IU/l). A repeat muscle biopsy showed active inflammation. High-dose oral steroids and azathioprine failed to bring about any improvement. Pulse methylprednisolone (1 g intravenously daily for three days) was tried unsuccessfully, with no improvement in muscle enzyme activities after three weeks. His weakness continued to increase and he became unable to lift his head from the pillow.

In an attempt to control his condition, total body irradiation was administered (150 cGy (rads) over five weeks, fractionated as two 15 cGy treatments each week; an 8 MeV linear accelerator was used, delivering a midplane dose rate of 12 to 14 cGy/min). The effect of this treatment was monitored with serial muscle enzyme estimations, lymphocyte counts, and strain-gauge myometry of major muscle groups. Within 10 days of starting treatment his muscle strength had improved and muscle enzyme values began falling. After four weeks he was ambulant, and three months after the course of treatment returned to work. Absolute lymphocyte counts fell rapidly and remained low. A muscle biopsy 10 weeks after total body irradiation showed resolution of the inflammatory process. Six months after treatment the patient showed signs of relapse with increasing weakness and muscle enzyme activities.

Reduction in blood lymphocyte count (normal $1.5-3.5 \times 10^9/l$) and serum creatine kinase activity (normal $<200 IU/l$) after total body irradiation (TBI), with strain-gauge myometric recordings of left quadriceps. Arrows denote muscle biopsies.

Comment

Irradiation depresses peripheral lymphocyte counts and has diverse effects on lymphocyte function. These effects may persist for many years.² There is one reported case of a patient with polymyositis treated successfully with total body irradiation after failure of conventional treatment.³ In our patient the dose of 150 cGy given as total body irradiation produced mild dyspepsia and transient granulocytopenia as the only side effects. In assessing the effect of treatment we employed serial strain-gauge myometric estimations of major muscle groups, a method which in our experience produced reliable and reproducible measurements of muscle strength.⁴ These data supported the clinical, biochemical, and histological evidence of improvement in muscle inflammation after total body irradiation. Throughout this period there was no change in the doses of oral immunosuppressive drugs.

Radiation-induced immunosuppression, given as total lymphoid irradiation, has recently been used in the treatment of intractable rheumatoid arthritis. Total body irradiation offers advantages over this method in the ease of administration, the low incidence of side