take extra salt, and warned that dehydration in saunas might be dangerous. He went to a health farm, where his diet consisted solely of grapefruit juice (supplemented with celery salt). He had a 10- to 15-minute sauna each morning. During the first three days he lost 4.5 kg. On the fourth day he became confused, ataxic, and tremulous; his diet was immediately increased and saunas were stopped. Next day, after no further lithium, he was still obviously ill and his GP was called. His serum lithium concentration was 1.6 mmol/l. The patient's wife then restarted the lithium at the reduced dosage of 800 mg a day.

I saw him five days later. Although improving, he had slurred speech, mild ataxia, and mild impairment of memory. The serum lithium concentration was now 1.1 mmol/l. One week later he still had difficulty standing unaided on one leg but this minimal impairment went a few days later.

Comment

This episode shows that precipitating lithium intoxication by dieting and salt depletion is not only a theoretical possibility but a practical danger. The dehydration and extra salt depletion resulting from sauna bathing¹ probably exacerbated the effects of the diet. The serum lithium concentration of 1.6 mmol/l some 36 hours after the last dose suggest that at one stage the concentration was probably around 3.2 mmol/l as the serum half life of lithium is one to two days.¹

Neurological signs continued for two weeks after serum lithium concentrations were within the therapeutic range. Equilibration of lithium is much slower within the central nervous system than in serum.³

This case also shows once again that patients may not heed or even hear the warnings of their doctors. It would seem sensible to give all patients taking lithium written instructions, such as those of Shaw,² that warn them against having special diets without consulting their doctor. This patient was also asked at the health farm if he was taking drugs. He said "no" because he felt that mentioning psychiatric medication would brand him. Perhaps it would be better if the staff at such establishments specifically mentioned lithium as a contra-indication to the regimen.

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² von Hartitzsch, B, et al, *British Medical Journal*, 1972, **4**, 757.

³ Schou, M, Amdisen, A, and Bastrup, P C, *British Journal of Hospital Medicine*, 1971, **6**, 53.

⁴ Dean, S, Green, D J, and Melnick, S C, *British Medical Journal*, 1977, **1**, 1449.

⁵ Shaw, D M, *British Journal of Psychiatry*, 1977, **130**, 432.

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Chronic myeloid leukaemia of adult type in 5-month-old infant

Of the two types of chronic myeloid leukaemia (CML) that occur in children,^{1,2} juvenile CML is found in children aged under 2 while adult CML occurs in children mainly between the ages of 7 and 16. We report here a case of adult type CML in a 5-month-old infant.

Case report

A 5-month-old boy of Kenyan Asian parents presented in January 1975 with a four-day history of crying and abdominal distension. He had previously had persistent rhinitis and some feeding problems. Apart from a right hydrocele the routine examination at 3 months had shown nothing abnormal.

On admission he was anaemic. The liver was enlarged 3 cm and the spleen 7 cm in the midclavicular line. Haemoglobin was 4.5 g/dl; 12.1%, was Hb F; white cell count was $501 \times 10^9/l$ ($501\,000/mm^3$) with 47% polymorphs, 33% immature granulocytes, 4% blasts, 12% eosinophils, and 3% lymphocytes. Platelets were $64 \times 10^9/l$. The leucocyte alkaline phosphatase score was 1. Marrow aspirate was hypocellular. Erythropoiesis was normoblastic but depressed. Myelopoiesis was hyperplastic with normal maturation but an excess of myelocytes. Megakaryocytes were reduced. Marrow culture was positive for the Philadelphia chromosome in all of the 10 cells examined. The cerebrospinal fluid contained 20 lymphocytes. The serum urate was

0.52 $\mu\text{mol/l}$ (8.7 $\mu\text{g}/100\text{ ml}$). Scattered lytic lesions were seen on skeletal survey.

In view of the prognosis we decided not to treat him aggressively and this was explained to the parents. Busulphan treatment (0.4–1.5 mg/day) was started. He was supported by blood transfusions as required. Throughout the initial illness his blood count was relatively stable (see chart) but he had intercurrent infections of the ears and respiratory tract. Abdominal discomfort was troublesome. Latterly arthralgia and bleeding into the mucous membrane and skin were problems.

A marrow aspirate taken in the ninth month of the illness showed 50% replacement of the marrow by myeloblasts, with depression of the other cell lines. This was subsequently reflected peripherally as a relative pancytopenia. He was treated with mercaptopurine (25 mg/day) and prednisolone (20 mg/day). His condition rapidly deteriorated, however, and he died at home as planned, aged 16 months.

Haematological findings and treatment.

Comment

Chronic myeloid leukaemia (CML) comprises 2–5% of childhood leukaemia.³ There are two types.^{1,2} Juvenile CML, which is found in children under 2 years, is characterised by anaemia, recurrent infections, lymphadenopathy, purpura, and eczematoid facial rash. Classically the white cell count is $20\text{--}80 \times 10^9/l$ with profound thrombocytopenia and raised Hb F. The Philadelphia chromosome is absent. In contrast, the adult type is of insidious onset, with anaemia and hepatosplenomegaly, and its greatest incidence in children is between the ages of 7 and 16 years. The white cell count is considerably higher at $150\text{--}400 \times 10^9/l$. The Philadelphia chromosome is present.

Because of the presence of the Philadelphia chromosome, an extremely high white cell count, and gross splenomegaly in the absence of lymphadenopathy, facial rash, and purpura, we considered this case to be a classic example of the adult type of CML. A search of published reports⁴ has shown no other reported case of the adult type in a 5-month-old infant.

Requests for reprints should be addressed to Dr R G Wilson.

¹ Reisman, L E, and Trujillo, J M, *Journal of Pediatrics*, 1963, **62**, 710.

² Hardisty, R M, et al, *British Journal of Haematology*, 1964, **10**, 551.

³ Lascari, A D, *Leukemia in Children*. Springfield, Illinois, Thomas, 1973.

⁴ Bloom, G E, et al, *Pediatrics*, 1966, **38**, 295.

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