bacterial overgrowth in the intestinal lumen. After relief of the small-bowel obstruction by removal of his tumour, one would assume that bacterial overgrowth would not be a feature, and certainly three months after his operation there were no clinical or biochemical features of malabsorption. This may be relevant to the disappearance of our patient's clubbing.

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Primary Salmonella typhi meningitis in an adult

Meningitis due to *Salmonella typhi* is rare and usually occurs as a complication during typhoid fever. We report a case of *S typhi* meningitis that occurred as a primary illness in an otherwise healthy adult.

Case report

A 24-year-old woman dental surgeon gave a two-day history of fever, vomiting, and headache of acute onset, with no associated bowel symptoms. She had been in good health and had been vaccinated against typhoid two years previously. Her history was otherwise insignificant. Examination showed a febrile patient of average build. The cardiovascular and respiratory systems were clinically normal. The abdomen was soft, with no distension, tenderness, or hepatosplenomegaly. Although conscious and rational, the patient was listless and uninterested in her surroundings. Neck rigidity was severe, and Kernig's sign was positive. The rest of the neurological examination, including funduscopy, gave normal results.

The white cell count was $17{\cdot}2 \times 10^9$/l (17 200/mm^3), with 92% neutrophils, 6% lymphocytes, and 2% monocytes. Results of urine analysis and serum electrolyte and blood urea tests were normal. The cerebrospinal fluid (CSF) was turbid and under tension, with proteins 5 g/l and glucose 3·7 mmol/l (67 mg/100 ml), simultaneous blood glucose being 7·6 mmol/l (137 mg/100 ml). The CSF contained 870×10^6 neutrophils/l (870/mm^3) and 16×10^6 lymphocytes/l. The stained CSF deposit showed many Gram-negative bacilli. *S typhi* was isolated on culture of the CSF. The results of blood and stool cultures taken during the first and second weeks of the illness were negative for salmonellae. The standard agglutination tests (Widal) done on the fourth, 12th, 20th, and 50th days from the onset of the illness showed persistent, non-rising titres against O and H antigens of *S typhi* and H antigen of *S paratyphi A* consistent with previous antityphoid vaccination.

Treatment was started with parenteral penicillin, chloramphenicol, and oral sulphadiazine. When the culture report was available, penicillin and sulphadiazine were replaced by parenteral ampicillin and co-trimoxazole. Pronounced clinical improvement was seen from the third day in hospital. Chemotherapy was continued for 14 days, and the patient was discharged on the 16th hospital day. Over a follow-up of 18 months she has remained in good health.

Comment

Salmonella meningitis chiefly affects babies and children,[1 2] and in the former birth trauma is thought to predispose to infection.[2] Although several species of salmonellae such as *S paratyphi B*, *S typhimurium*, *S enteritidis*, *S choleraesuis*, and *S panama* have been isolated from the cerebrospinal fluid, *S typhi* has only rarely and sporadically been incriminated as causing purulent meningitis.[1–3] In most cases *S typhi* meningitis has occurred as a complication during a typhoidal illness, where fever and bowel symptoms were the presenting features.[4]

Watson[5] suggested that *S typhi* bacteraemia was much more common than was thought, and described 12 cases where *S typhi* was isolated from routine blood or clot culture in patients in whom neither the clinical features, the agglutination titres, nor the necropsy findings indicated a typhoidal illness. Seeding of the organisms in the meninges and their subsequent multiplication may explain the pathogenesis of isolated typhoid meningitis without preceding typhoidal illness. This case draws attention to *S typhi* as a cause of Gram-negative bacillary meningitis, particularly in typhoid endemic areas.

We thank Professor T E D Chapman for identifying the organism and Miss M Dullewe and Miss V S B C de Mel for secretarial help.

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Transplacental hyponatraemia due to oxytocin

The antidiuretic properties of oxytocin have been described,[1] and several reports have emphasised the dangers of water intoxication when oxytocin and large amounts of electrolyte-free fluid are administered. We describe a case of maternal water intoxication which occurred after the use of oxytocin in 5% dextrose for induction of labour, and which resulted in severe hyponatraemia and convulsions in the newborn infant.

Case report

A healthy 24-year-old primigravida underwent surgical induction of labour at term after a pregnancy complicated by mild hypertension. After rupture of the membranes an intravenous infusion of oxytocin in 5% dextrose in water was begun, increasing in concentration over four hours from ½ to 4 units of oxytocin per 500 ml 5% dextrose, and subsequently given at a rate of 32 milliunits oxytocin per minute. Eighteen hours later, having received a total of 6·5 litres of 5% dextrose and 36 units of oxytocin, she had a grand mal convulsion. This was controlled with intravenous diazepam, and a liveborn male infant, birthweight 3470 g, was delivered by forceps 15 minutes later. The urine output during labour was 2·7 litres, only 1·3 litres being passed during the 14 hours before delivery. Maternal blood taken after delivery showed plasma concentrations of sodium 117 mmol(mEq)/l, chloride 86 mmol(mEq)/l, osmolality 243 mmol(mOsm)/kg, potassium 3·5 mmol(mEq)/l, and urea 2·3 mmol/l (14 mg/100 ml). A diagnosis of water intoxication was made, fluids were withheld, and she had a spontaneous diuresis of 3 litres. Thirty-six hours later the plasma electrolyte concentrations and osmolality were normal and she appeared to have made a full neurological recovery. She returned home on the sixth postpartum day and was perfectly well when seen six weeks postnatally.

At birth the infant was limp and apnoeic, requiring endotracheal intubation and artificial ventilation until spontaneous respirations were established at 10 minutes of age. After transfer to the special care baby unit he developed a raised respiratory rate and was noted to have dystonic limb movements and short episodes of apnoea. From the age of 5 hours he had repeated generalised convulsions. Between fits he had increased extensor tone and further apnoea with cyanosis. Umbilical cord blood taken at delivery showed a plasma sodium concentration of 114 mmol/l and osmolality 243 mmol/kg, and venous blood taken immediately after the first convulsion showed plasma concentrations of sodium 110 mmol/l and chloride 81 mmol/l, osmolality 240 mmol/kg, potassium 5·2 mmol/l, calcium 2·1 mmol/l (8·4 mg/100 ml), magnesium 0·67 mmol/l (1·6 mg/100 ml), and urea 2·7 mmol/l (16·3 mg/100 ml). Lumbar puncture was normal.

His fits were treated with intramuscular phenobarbitone and paraldehyde, but resolved only after correction of his hyponatraemia with twice-isotonic saline given intravenously and fluid restriction. He also received injections of 9-fludrocortisone and magnesium sulphate. In the first 48 hours of life he passed 240 ml of urine, and by the age of 36 hours the plasma electrolyte concentrations and osmolality had returned to normal and his convulsions had

ceased; all medication was discontinued. He subsequently remained well and returned home with his mother on the sixth day. When seen at the age of 9 months by a consultant paediatric neurologist he had been free of fits and was considered to be completely normal.

Discussion

In this case severe neonatal illness resulted from transplacental hyponatraemia after the maternal administration of excessive quantities of 5% dextrose in combination with oxytocin at a rate which produces an 80% reduction in urinary flow.[1] Although 30 cases of maternal water intoxication due to oxytocin have been described, in only two of these was oxytocin used for inducing labour.[2,3] In neither of these cases was the clinical course of the newborn infant mentioned, although a cord plasma sodium concentration of 124 mmol/l, which returned to normal by 12 hours of age, was documented in one infant.

Maternal hyponatraemia, experimentally produced by hypotonic expansion of the extracellular fluid compartment with intravenous 5% dextrose, results in a parallel fall in fetal plasma sodium concentration.[4] This has been confirmed clinically in a report of transplacental hyponatraemia in four newborn infants whose mothers had become hyponatraemic either after the administration of 5% dextrose during labour or after the use of diuretics and a low-salt diet for toxaemia.[5] Three of these four infants were ill, their clinical features including apnoea at birth, hypotonia, poor suck, cyanosis, and convulsions. They thus resemble our case, and all appeared to make a full recovery.

The present report serves to emphasise the dangers of water intoxication in both mother and fetus that results from the antidiuretic properties of oxytocin when administered in combination with large quantities of electrolyte-free fluid. It also underlines the importance of recognising that fluid and electrolyte abnormalities in the mother may result in similar abnormalities, and consequent illness, in the newborn infant.

We thank Professor J P M Tizard for help and encouragement.

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Vitamin E in treatment of Huntington's chorea

Vitamin supplements and special diets have become fashionable recently, particularly in chronic, incurable, and poorly understood diseases. A good example of this is the gluten-free diet and multiple sclerosis. This type of treatment is difficult to assess in diseases of variable course but this should not preclude trying to prevent people wasting time, money, and effort on such treatments. Patients and their families cannot be blamed for grasping at straws, for the professionals should not allow untested theories to become widely disseminated.

Huntington's chorea is chronic, incurable, and poorly understood. Present treatment is only symptomatic and palliative, and no treatment has been discovered that will affect the relentless downhill course. A claim, however, was made in Canada[1] and seconded in England[2] that high doses of vitamin E considerably benefited choreic patients when added to existing treatment, although in each case only one patient had been treated. Vitamin E has been used for many years for anything from ailing sexuality to intermittent claudication, but only in the latter is there any evidence of its efficacy.[3] In an attempt to avoid problems of the type associated with multiple sclerosis and the gluten-free diet we decided to undertake a double-blind crossover trial to assess these claims before they became too widely known.

Method

Ten patients suffering from Huntington's chorea were considered sufficient for a pilot study. The criteria laid down for inclusion were: (*a*) a genuine case of Huntington's chorea—that is, with a family history; (*b*) a patient who was looked after at home and not in an institution; (*c*) a patient who was known to and had been examined by us; and (*d*) a reliable family member to complete forms. We found 11 such patients (five women and six men) aged 30-70 who were prepared to participate, and permission to include them was sought from their general practitioners.

As patients with Huntington's chorea often present to an outsider as being fairly well, presumably being able to summon up enough energy for one interview, we decided that the person looking after the patient should complete the progress questionnaire weekly. The variables on the form were therefore subjective and kept as simple as possible, although they were pertinent to the problems of choreic patients. A check on the weight of the patient each week was originally requested but some families had no scales. No appreciable weight change, however, was noted in those that completed this section. The variables used were: energy, involuntary movements, walking, speech, mental alertness, mood, behaviour, sleep, appetite, and (weight). Possible scores were better, worse, or no change. Better scored +1, worse scored −1, and no change scored nil.

The trial lasted 24 weeks, divided into two. For the first 12 weeks the patient took a placebo or vitamin E (tocopheryl acetate (Ephynal), 200 mg tablets) and the reverse for the second 12 weeks. The dosage was 400 mg three times a day, which is slightly lower than that used in Canada but high enough to cause any improvement if improvement was the outcome.[2] The key to the trial was kept in a sealed envelope in case of emergency and not opened until the analysis of the trial.

During the trial one patient died after 13 weeks from a myocardial infarction, and a second had considerable problems with which his wife was unable to cope and he took the tablets only spasmodically. Of the remaining nine patients, only four completed the trial and assessment forms completely, the others omitting an occasional weekly return. These omissions were randomly distributed between the placebo and vitamin E and have been treated as containing no change—that is, zero score. No side effects were expected and none were complained of in the entire trial.

Response to vitamin E and to placebo of 11 choreic patients

Patient No	No of forms completed	Vitamin E	Placebo
1	24	0	+11
2	24	−14	−19
3	24	0	+27
4	17	−9	−2
5	20	+4	+3
6	24	−12	−20
7	22	+22	+30
8		(did not complete trial)	
9	20	−34	−39
10	20	−14	−41
11		(patient died)	
Total		−57	−50

Results and comment

The response to the placebo was −50 (the sum of the scores of all patients) and to the vitamin E −57 (see table). The negative scores obtained for both placebo and vitamin E reflect the progressive nature of the disease. The scores are not significantly different and the only conclusion that may be drawn is that empirical treatment with vitamin E has no place in the treatment of choreic patients. This study produced a negative result but because of the problems of the gluten-free diet in multiple sclerosis we thought it important to publish these negative findings.

Our grateful thanks to Dr Norman Pollitt for his help and advice and to Roche Products Ltd for supplying the Ephynal and placebo tablets.

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