[2] Komp DM, Selden Jr RF. Coagulation abnormalities in cystic fibrosis. *Chest* 1970;**58**:501–3.
[3] Corrigan Jr JJ, Taussig LM, Beckerman R, Wagener JS. Factor 11 (prothrombin) coagulant activity and immunoreactive protein: detection of vitamin K deficiency and liver disease in patients with cystic fibrosis. *J Pediatr* 1981;**99**:254–7.
[4] Hart JP, Schearer MJ, McCarthy PT, Rahim S. Voltammetric behaviour of phylloquinone (vitamin K_1) at a glassy carbon electrode and determination of the vitamin in plasma using high performance liquid chromatography with electrochemical detection. *Analyst* 1984;**109**:477–81.
[5] Shearer MJ, Rahim S, Barkhan P, Stimmler L. Plasma vitamin K_1 in mothers and their newborn babies. *Lancet* 1982;**ii**:460–3.
[6] Shearer MJ, Crampton OE, McCarthy PT, Mattock MB. Vitamin K_1 in plasma: relationship to vitamin K status, age, pregnancy, diet and disease. *Haemostasis* 1986;**suppl 5**:83.

Correspondence to Dr IA Choonara, Division of Clinical Pharmacology, University Hospital, S–751 85 Uppsala, Sweden.

Accepted 25 October 1988

Water intoxication and hyponatraemic convulsions in neonates

V VANAPRUKS AND K PRAPAITRAKUL

Department of Paediatrics, Pramongkutkalo Medical College, Bangkok, Thailand

SUMMARY We studied two neonates fed diluted formula and excessive water who developed hyponatraemic convulsions; treatment included intravenous hypertonic saline and water restriction. Educating mothers is important to stop recurrences.

From 1967 to 1983 there were 24 cases of infants with water intoxication reported in the literature; their ages ranged from 3 weeks to 11 months.[1–4] We report two neonates with seizures secondary to water intoxication.

Case reports

CASE 1

A 20 day old girl, the product of a full term, normal delivery, was the first child born to a healthy 20 year old mother. The baby had been well since birth. On the day of admission she was taken to hospital because of irritability and slight abdominal distension not associated with vomiting or diarrhoea. The physical examination was unremarkable and after reassuring the mother the child was sent home. One hour later the baby had a tonic-clonic convulsion and was readmitted to hospital where she was treated with intravenous diazepam. Blood was taken for biochemical and microbiological investigations. The child was given 5% dextrose in one fifth normal saline intravenously.

Physical examination on admission (after intravenous diazepam) showed an unconscious child with a rectal temperature of 36·5°C, pulse rate 120/minute, and respiratory rate 36/minute; there was no anaemia, jaundice, or oedema. The anterior fontanelle was open and soft, the abdomen was slightly distended with normal bowel sounds, and there was no hepatosplenomegaly. The patient responded to deep pain by moving her extremities; her pupils were equal and reactive to light.

The white cell count was $5{\cdot}37\times10^9$/l with 60% polymorphonuclear cells and 40% lymphocytes, and the haemoglobin concentration was 107 g/l. A microscopic examination of the urine was normal with a specific gravity of 1·005. The blood urea nitrogen was 3·2 mmol/l and the blood glucose concentration 7·8 mmol/l. The concentration of sodium in the serum was 117 mmol/l, potassium 4·8 mmol/l, chloride 92 mmol/l, and carbon dioxide 15 mmol/l. The osmolality of the serum was 240 mmol/kg and of the urine 83 mmol/kg. The cerebrospinal fluid was normal; culture of blood, urine, and cerebrospinal fluid gave negative results.

Because of the severe hyponatraemia in an otherwise healthy infant a detailed history of feeding was taken from the mother. On the day before admission the baby had been given 2 oz of diluted formula consisting of one tablespoon of S26 powdered milk (Wyeth (Ireland) Ltd) in 3 oz of water every four hours; in between each feed the mother gave the infant 4 oz of water. Throughout this period she passed a large quantity of urine. The diagnosis of hyponatraemia caused by excess feeding of water was made and the baby was managed by restricting fluid intake. No further convulsion occurred and the serum electrolytes became normal on the following day.

CASE 2

An 8 day old boy, the fifth child of an impoverished family, was born at term after a normal vaginal delivery, weighing 3020 g. The Apgar scores were 9 and 10 at one and five minutes, respectively. The baby had no problem during his nursery stay. At home he was fed 2 oz of a normal dilution of S26

every two hours followed by 2 oz plain water. On the day of admission the baby became lethargic and fed poorly. He developed intermittent tonic-clonic seizures which lasted for three hours. In the emergency room the baby was given 15 mg phenobarbitone intravenously. His convulsions stopped but they recurred several times and could not be controlled by phenobarbitone, diazepam, calcium gluconate, or glucose.

On admission his temperature was 36°C, pulse rate 112/minute, and respiratory rate 38/minute. He weighed 3140 g. He was unconscious and was having tonic-clonic convulsions; his anterior fontanelle was slightly tense; he was not oedematous. The rest of the physical examination was normal. Laboratory investigations showed a serum sodium concentration of 116 mmol/l, chloride 86·9 mmol/l, potassium 4·99 mmol/l, carbon dioxide 16·9 mmol/l, glucose 7·2 mmol/l, calcium 2·2 mmol/l, and phosphorus 2·02 mmol/l. Culture of blood, urine, and cerebrospinal fluid gave negative results.

The seizures stopped after 20 ml of 5% sodium chloride was given intravenously; the boy had no further seizures.

Discussion

These two infants had no history of any salt losses to account for the hyponatraemia and appeared clinically well hydrated with no evidence of oedema. From the history of excess water intake, hypothermia, low serum osmolality, low serum sodium concentration, normal serum blood urea nitrogen and creatinine we concluded that the babies had dilutional hyponatraemia after an increased water intake. Case 1 was the first child of an inexperienced mother. The second case was the fifth child of an impoverished family; water intoxication occurred because of feeding the child large amounts of water.

Most infants with water intoxication have hypothermia, are sleepy or irritable, and when seizures occur they are often multiple and recurrent. Some cases reported had oedema and hypertension; hyponatraemia with a sodium concentration of less than 125 mmol/l, a low chloride, and low carbon dioxide concentration was common. In one of our cases as well as cases reported by others,[1–4] the blood urea nitrogen was higher than the normal range for their age,[5] which might reflect a blood volume contraction due to shifting of water from the extracellular fluid to the cell when hyponatraemia occurred; the transient rise in blood glucose concentration was presumably caused by the seizures and stress.[1]

The mechanism of hyponatraemia is controversial: inappropriate secretion of antidiuretic hormone or inability of the kidney to excrete excess water have been suggested.[2] In young infants kidney function is immature, and the glomerular filtration rate is only 30% of that of a normal adult.[6] When infants ingest large amounts of water over a short period of time their kidneys are unable to excrete the large water load; this results in a dilution of their serum sodium, and salt depletion can further impair water excretion.

In the young infant with a strong sucking reflex, however, a baby might be satisfied with sucking and ingest excess water if it is continually offered by the mother. Crumpacker and Kriel theorised that infants continue to drink excess water because they are hungry.[3]

We are grateful to Dr Peter Echeverria from the United States Army Medical component Armed Forces Research Institute of Medical Sciences for revising this manuscript.

References

1 Nickman SL, Buckler JM, Weiner LB. Further experiences with water intoxication. *Pediatrics* 1968;**41**:149–51.
2 Bennett HJ, Wagner T. Acute hyponatremia and seizures in an infant after a swimming lesson. *Pediatrics* 1983;**72**:125–7.
3 Crumpacker RW, Kriel RL. Voluntary water intoxication in normal infants. *Neurology* 1973;**23**:1251–5.
4 Parrtridge JC, Payne ML, Leisgang JJ, *et al*. Water intoxication secondary to feeding mismanagement. *Am J Dis Child* 1981;**135**: 38–41.
5 Schwartz GJ, Haycock MB, Spitzer A. Plasma creatinine and urea concentration in children: normal values for age and sex. *J Pediatr* 1976;**88**:828–30.
6 Edelman CM Jr, Barnett ML. Role of the kidney in water metabolism in young infants. *J Pediatr* 1960;**56**:154–79.

Correspondence to Dr V Vanapruks, Department of Paediatrics, Pramongkutkalo Medical College, Bangkok 10400, Thailand.

Accepted 26 September 1988