Hypernatraemic dehydration and necrotizing enterocolitis

A.J. Clarke and J.R. Sibert

Department of Child Health, Llandough Hospital, Penarth, Wales.

Summary: Severe hypernatraemic dehydration developed over the first twelve days of life in a breast-fed infant girl. Upon oral rehydration with formula milk, no acute neurological problems arose, but she subsequently developed necrotizing enterocolitis. Intravenous rehydration may be preferred to the oral route in such infants.

Introduction

Hypernatraemic dehydration is known to occur as a consequence of inadequate fluid intake in purely breast-fed infants (Anand et al., 1980; Clarke et al., 1979; Roddey et al., 1981). An elevated sodium concentration in the mother’s milk may also contribute to the hypernatraemia (Anand et al., 1980). We report such a case where oral rehydration was successfully employed, but who later developed necrotizing enterocolitis.

Case report

An infant girl was born at term weighing 3.10 kg. When discharged at two days, feeding was not satisfactory and lactation was not established. A community midwife sent the child to our clinic because of weight loss at 12 days.

The infant weighed 2.30 kg and was clinically marasmic and dehydrated, but not shocked. There had been no diarrhoea or vomiting and the child was eager to feed. The mother had successfully breast-fed her previous child for several months, but expressed concern that her milk supply was inadequate on this occasion.

The serum sodium on admission was 176 mmol/l and the glucose was 1.1 mmol/l. Slow rehydration with formula milk, orally and by tube, was used to ensure a gradual correction of the hyperosmolar state and to provide calories. The blood glucose was corrected to 8 mmol/l by the first feed, and the serum sodium fell steadily to 146 mmol/l over the first 48 h. The osmolality fell from 370 to 292 mOsm/l and the haematocrit from 0.73 to 0.55 over a similar period.

After one week in hospital, by which time oral feeds in full volume were being taken well, frank blood appeared in the infant’s stools and she passed copious mucus. Her rectal mucosa was inflamed, and an abdominal X-ray revealed gastric distension, a marked paucity of bowel gas and a foamy appearance in the descending colon. A diagnosis of necrotizing enterocolitis was made, a full sepsis screen was taken, and conventional treatment with gastric drainage and intravenous fluids and antibiotics was begun. No bacterial or viral pathogen was found in the stool or elsewhere, except Staphylococcus aureus on two superficial swabs. Parenteral nutrition was continued for a full week before the gradual reintroduction of milk feeds, and the child was discharged once a good weight gain had been established. She has done well at home since then.

Discussion

Previously reported cases of hypernatraemic dehydration caused by inadequate fluid intake in purely breast-fed infants, have all been hydrated with intravenous fluids (Anand et al., 1980; Clarke et al., 1979; Roddey et al., 1981). Intravenous hydration has been associated with neurological problems such as hemiplegia and convulsions, in the acute hypernatraemic dehydration of gastroenteritis in infants given high-solute feeds (Habel & Simpson, 1976). Oral rehydration might seem safer, because changes in the serum osmolality should be more gradual and the cerebrospinal fluid serum osmolality gradient would then be lower. Our child had no neurological complications.

Polycythaemia and hypoglycaemia are both prime risk-factors for necrotizing enterocolitis, especially in infants of more than 2 kg (Wislon et al., 1983), and our patient suffered both of these conditions for an
unknown period prior to her hospital admission. She then developed necrotizing enterocolitis some time after her successful oral rehydration. If there is a high incidence of these risk factors in this group of fluid-depleted breast-fed babies, then intravenous rehydration may be safer than oral. Parenteral nutrition would be needed to provide calories until oral feeding was deemed safe, and very slow rehydration would still be needed to prevent the complications of cerebral oedema. This case also reminds us of the importance of close community supervision for those mothers and infants whose feeding is not well established by the time of discharge from hospital (Roddey et al., 1981).

References


