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Case Reports

Hypernatraemic Dehydration in Exclusively Breast-fed Infants

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Abstract We report two recent cases of hypernatraemic dehydration associated with insufficient breast milk intake in the neonatal period. Both mothers failed to recognise inadequate breast milk intake and signs of dehydration in their babies. The infants had massive weight loss (23% and 22% of birth weight) and sodium levels of 185 and 174 mmol/l respectively. They required slow intravenous rehydration with isotonic fluid and recovered without long-term sequelae. This uncommon problem should be preventable by good education and close supervision of nursing mothers. It is very important not to rehydrate too rapidly to avoid further neurological damage.

Key words Infant; Mothers; Breast-feeding; Hypernatraemia aetiology; Dehydration; Weight loss

Introduction

The long and short-term benefits of breast milk for infant health are well described.[1] The most frequent reason for stopping breast-feeding is "insufficient breast milk production". Often it is due to false perception or low suckling frequency on the breast.[2] Genuine breast milk insufficiency is uncommon. Its association with hypernatraemic dehydration in exclusively breast-fed neonates has been sporadically reported largely in the United States of America.[3-6] We report two recent British cases of hypernatraemic dehydration in babies who were exclusively breast-fed.

Case Reports

Case 1

An 11-day-old white male infant was admitted with weight loss and a 12-hour history of poor feeding. This was his mother's first pregnancy and the early antenatal course was uneventful but she had mildly elevated blood pressure during the last few weeks of pregnancy. Labour was induced at 42 weeks gestation with prostaglandins and syntocinon. The baby was delivered by vacuum extraction for foetal distress. The birth weight was 3.83 kg. He was well after the delivery and exclusively breast-fed. When discharged at four days of life, he was feeding six times daily and for 10 - 30 minutes per feed. The mother was re-admitted the next day for abdominal pain and treated with oral co-amoxiclav. The breast-feeding was continued. She improved the day after and was discharged. A day prior to the baby's admission, he was lethargic and not interested in breast-feeding. There had been no over-heating, diarrhoea, vomiting, or fever.

Examination on admission revealed a weight of 2.95 kg (23% loss in birth weight) and head circumference 34.3 cm. The baby was thin, lethargic, and his peripheries cool. The skin turgor was poor and the anterior fontanelle was depressed. He was judged to be more than 10% dehydrated. The admission blood biochemistry was as follows: sodium 185 mmol/l, potassium 4.8 mmol/l, creatinine 342 umol/l, urea 93.8 mmol/l, glucose 5.8 mmol/l, arterial pH 7.34, bicarbonate 20 mmol/l, base deficit 5 mmol/l and serum osmolality 447 mosmo/l. The urine osmolality was 546 mosmo/l and microscopy showed 20 white cells/ml and 65 red cells/ml. Blood and urine cultures were negative. Stool culture and microscopy showed no pathogen.

The baby was slowly rehydrated with intravenous 5% dextrose and 0.45% sodium chloride solution and he started passing urine 13 hours after admission. His plasma sodium was normalised over 3 days. His post-rehydration weight was 3.7 kg. Oral formula feeding was started and

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the baby was discharged 7 days after admission. His blood biochemistry, physical and neurological examinations were normal at one year of age.

Case 2

A 7-day-old white male infant was admitted with drowsiness and poor feeding. This was his mother's first pregnancy. The pregnancy was uneventful and he was born normally at term, weighing 3.2 kg. The baby was exclusively breast-fed and was discharged on the third day of life. Two days before admission, he was feeding poorly (taking 3-4 feeds a day) and lethargic. He had not fed at all for 16 hours before the admission. There had been no vomiting and he had no bowel motions for 3 days.

Examination revealed that the baby was drowsy, jaundiced, tachypnoeic, grunting and peripherally cyanosed. His fontanelle was depressed, mucous membranes dry and his weight was 2.5 kg (22% loss in birth weight). Clinically, he was more than 10% dehydrated. The admission laboratory results were as follows: sodium 174 mmol/l, urea 41.7 mmol/l, potassium 4.8 mmol/l, glucose 3.9 mmol/l, serum osmolality 463 mosmo/l, arterial pH 6.9, bicarbonate 5.2 mmol/l, base deficit 20.5 mmol/l, C-reactive protein 14.7 mg/l, total bilirubin 200 mmol/l, haemoglobin 16.3 g/l, white cell count 23.8 x 10^9/l, platelet count 331 x 10^9/l and urine osmolality 574 mosmo/l. No pathogen was identified in blood, urine, cerebrospinal fluid and stool.

He was resuscitated intravenously with 5% human albumin, 0.45% sodium chloride/ 5% dextrose and 8.4% sodium bicarbonate solutions. Twelve hours after admission, he developed tonic clonic seizures which were controlled with phenobarbitone. The sodium level immediately after the seizure was 170 mmol/l and the arterial pH was 7. He started passing urine fourteen hours after admission. The electrolyte disturbances were normalised over the ensuing three days and his post-rehydration weight was 3.1 kg. Oral formula feeding was gradually established and phenobarbitone was gradually stopped. Renal and cerebral ultrasound scans showed normal findings. The baby was discharged at the age of 14 days. His physical and neurological examinations were normal at 6 months of age.

Discussion

The cause of dehydration in the first baby was likely to be due to a combination of inadequate feeding frequency and poor maternal milk supply. The reasons for the poor milk supply in the mother were not apparent, but the stress of the brief illness might have been a contributing factor. Breast milk sodium levels were not measured in our cases. However, in the 22 reported cases where breast milk sodium levels were measured, the range of sodium levels was 13 to 104 mmol/l with a mean of 50.7 mmol/l. The elevated sodium levels had thought to be a contributing factor. Physiologically, the breast milk sodium level falls from around 65-70 mmol/l at the first postpartum day to below 20 mmol/l by the third postpartum day.[7] The reason why some mothers produce high sodium, low lactose and low volume milk is not known. Many factors (e.g. hormonal, neuro-psychological) are likely to be involved. A high breast milk sodium level has been proposed to be an indicator of poor lactogenesis. As milk production increases the sodium level tends to fall accordingly.[8] In practical terms, measuring breast milk sodium may be a simple way of assessing the adequacy of milk production.[8] The hypernatraemic dehydration in these babies is therefore caused by the combined effects of the inadequacy of milk volume (i.e. water deprivation), reduced ability for neonatal kidneys to conserve water and raised breast milk sodium.[9]

Our two cases were fortunate to have suffered no apparent sequelae. The seizures suffered by the second baby were likely to be due to the effects of severe acidosis and hyperosmolality on the brain cells. There have been forty reported cases of hypernatraemic dehydration due to inadequate breast milk feeding in the past twenty years. Massive (14-37%) weight loss is the rule. Most are associated with serious complications or sequelae and they include acute renal failure, renal vein thrombosis, coagulopathy, leg amputation, seizures, cerebrovascular accident, cerebral palsy, delayed development, perforated duodenal ulcer, hypertension, and necrotizing enterocolitis.[3-6] Slow intravenous rehydration and very gradual restoration of electrolytes are the keys to prevent further complications due to over-zealous treatment.[10] Clarke et al rehydrated their patient by the oral route, but the patient developed subsequent necrotising enterocolitis. [4] This suggests that oral rehydration may be unwise in babies with such extreme degree of dehydration as the splanchnic circulation is likely to be severely compromised. The basic principles of fluid therapy in hypernatraemic dehydration are: establishment of haemodynamic stability with boluses of isotonic intravenous fluid (e.g. 10-20 ml/kg of normal saline solution) over 10-15 minutes, replacement of water and electrolytes deficit over 48 hours to allow a reduction of sodium level to be less than 10-15 mmol/ day, and replacement of insensible and ongoing fluid and electrolytes loss.[3]

In the majority of reports, the mothers are usually primigravida and highly motivated for breast-feeding.

Thus, they are inexperienced and very insistent on breast-feeding. The pregnancy and delivery are usually uncomplicated. The babies are generally contented, quiet and fail to demand for feeds even when dehydrated. The infants usually present at 10 to 20 days of age with severe dehydration and profound hypernatraemia. They thrive with adequate nutritional support. Additional risk factors include: inappropriate feeding schedule, poor sucking, inverted nipples and reluctance to give supplement feeds. In the second case, the baby was obvious not fed adequately for a long period of time. The mother had taken "feeding on demand" to its extremes. Similar to previous described cases, the baby sucked poorly and slept a great deal. The mother thought all was well when the baby appeared contented and not hungry. As illustrated by the present two cases, the primigravida or inexperienced mothers may not recognise that their babies are not getting enough breast milk and falling ill until late stages.

The recurrent factors in previous cases are poor or no supervision, early discharge after confinement (1-2 days), and inexperienced mothers. In our two cases, there were breaks in supervision by their community midwife. The first case was because of hospitalisation of the mother but the reason for the second case was not apparent. British nursing mothers generally have regular contact with their community midwives in the first two postnatal weeks. They also have ready access to advice from their health visitors and family doctors. Such close supervision may be the reason why hypernatraemic dehydration due to breast milk insufficiency appears to be rare in Britain (only one case was reported in the past[4]), but the low breast-feeding rate (65% at one month) may also be a factor. There are no statistics of this problem available in Britain. However further reviewing of the biochemical database for the previous four years in our hospital (the only Children's hospital in Liverpool) revealed no other cases.

Breast-feeding is largely a learned skill. Successful breast-feeding requires determination, help, support and encouragement starting from the antenatal period. Apart from the actual procedure of breast-feeding, mothers should be taught to recognise signs of her babies' well-being. For example, the newborn should have smooth healthy skin, good colour, frequent (e.g. at least 5 a day) wet diapers with pale odourless urine, two to five bowel movements and at least 8-10 feeds in 24 hours. The baby should also be alert, responsive, sucking actively and contented but not lethargic.[11] Medical advice or reassurance should be sought if these criteria are not met especially if there is a weight loss of more than 12 percent.[12] Mothers may recognise her milk flow is adequate by the feeling of breast fullness before feeds, and seeing milk dribbling or squirting from the breasts. She may feel a "let-down" (tingling and fullness in the breasts associated with milk flow and there may also be a sensation of thirst) when feeding her baby. Expressing a bit of milk with hands or pump will also help to see the breast is forming milk. When milk production is genuinely not sufficient and not improved by frequent suckling, supplementary feeds would be required.

This case report highlights the uncommon but hidden serious problem of hypernatraemic dehydration that could be associated with breast-feeding. Factors affecting the mother and the baby may operate together to cause this problem. It should be preventable by good education and close supervision of nursing mothers.

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