Laing IA / Acta Pharmacol Sin 2002 Oct; 23 Supplement: 48-51

ISSN 1671-4083
Shanghai Institute of Materia Medica
Chinese Academy of Sciences
http://www.ChinaPhar.com

Hypernatraemic dehydration in newborn infants

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INTRODUCTION

Hypernatraemic dehydration is a potentially lethal condition and is associated with cerebral oedema, intracranial haemorrhage, hydrocephalus and gangrene[1]. The infant's plasma sodium concentration is elevated due predominantly to loss of extracellular water. In the past, hypernatraemia occurred most frequently when artificial feeds of too high a sodium concentration were fed to babies[2]. This was more common in infants who were fed powdered milk, especially if the mother added extra scoops of powder and failed to provide enough water in the mixture. The resultant hypernatraemia may have stimulated the child's thirst, and further feeds high in sodium would exacerbate the problem.

EPIDEMIOLOGY

Hypernatraemia was previously thought to be unusual in breast-fed babies. Nevertheless, from 1979 to 1989 there were sporadic reports of hypernatraemic dehydration occurring in breast-fed babies[3-8]. In the 1990s there has been an increase in the number of breast-fed infants reported to have hypernatraemic dehydration[9-15].

Over a period of 18 months in Edinburgh, 13 of almost 9000 infants born were admitted to the Neonatal Unit at less than three weeks of age with hypernatraemic dehydration. All were breast-fed. In our study the plasma sodium concentrations of these infants ranged from 150 to 173 mmol/L. Seven infants were readmitted having already been discharged home but six were diagnosed on the postnatal wards prior to discharge (unpublished data). Few cases have previously been described to occur in hospital[9,16].

AETOLOGY

Hypernatraemia may be associated with decreased fluid intake[17], excessive fluid loss[18] or excessive sodium intake[19,20]. The last of these was most dramatically established during a mass accidental salt poisoning of infants in March 1962 when there was an error in making up proprietary milk[20]. It is therefore not surprising that there have been attempts to establish that the aetiology of hypernatraemic dehydration in breast-fed babies is an unusually high sodium content of maternal breast milk. The sodium content of breast milk at birth is high and declines rapidly over the subsequent days. In 1949 Macy[21] established that the sodium content of colostrum in the first five days is (22±12) mmol/L, and of transitional milk from day five to ten is (13±3) mmol/L, and of mature milk after 15 d is (7±2) mmol/L. Morton[22] studied the breast milk of 130 women as they began to breast-feed. Women who failed to establish good breast-feeding did not experience the normal physiological drop in breast milk sodium concentration compared to those who had little difficulty in establishing a good milk flow.

Today the evidence points to the commonest cause of hypernatraemic dehydration being low volume intake of breast milk. The infant becomes dehydrated while the kidneys are mature enough to retain sodium ions. Water loss occurs predominantly through the skin and from the lungs.

CLINICAL PRESENTATION

The clinical presentation of hypernatraemic dehydration is usually around ten days with a range quoted

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in the literature from 3 to 21 d. The parents may have failed to identify that the infant is ill, and professionals may also be falsely reassured by the infant's apparent well-being[23]. Signs may be non-specific, including lethargy[3,4,24] and irritability[25]. Occasionally there is an acute deterioration which precipitates the infant's emergency admission to hospital.

During acute isonatraemic or hyponatraemic dehydration clinicians may rely on sunken eyes and depressed anterior fontanelle as signs of total body water loss. In hypernatraemic dehydration however there may be changes in brain cell osmolality and cerebral oedema, and the resultant fullness of the anterior fontanelle may disguise the underlying dehydration. Clinical examination of these infants at presentation is very variable. Some present with lethargy and have an unremarkable examination. Others may be alert and hungry and are clinically dehydrated. Still others may be moribund by the time they arrive at the emergency room.

MORBIDITY AND MORTALITY

The condition carries an acute morbidity and mortality. Arboit[26] described an infant whose serum sodium peaked at 180 mmol/L who developed a temporary right-sided facial palsy. Rowland[6] reported a child with a serum sodium of 174 mmol/L who had apnoeas and bradycardias, and another child whose sodium concentration was 206 mmol/L who displayed seizures, hypertension and disseminated intravascular coagulation. Clarke[27] described a 12 day old child who presented with a serum sodium of 176 mmol/L who evinced no neurological problems, but after establishing full oral feeds was diagnosed as having necrotising enterocolitis. More dramatic still, Cooper[14] reported five infants with hypernatraemic dehydration of whom two were apparently normal, one had amputation of the left leg secondary to an iliac artery thrombus, one had decreased facial movement, and one had 'EEG slowing,' multiple cerebral infarctions and seizures. The brain damage may be caused by cerebral oedema, intracranial haemorrhages, haemorrhagic infarcts and thromboses. These can be identified in babies by using magnetic resonance imaging, MRI[28]. Kaplan's paper[1] outlines the course of two infants who died. The first presented at nine days of age with a plasma sodium of 191 mmol/L who suffered seizures, massive intraventricular haemorrhage, multiple dural thromboses and who died on day 12. The second presented on day 13 with a plasma sodium concentration of 180 mmol/L and showed on CT scanning multiple areas of intraventricular, periventricular, and cortical haemorrhage. This second child died on day 16.

TREATMENT

Some complications, especially seizures, occur most frequently during treatment[9,18,29,30]. It is recognised that the mainstay of treatment is to rehydrate the child very slowly. If the staff caring for the child attempt to correct the high sodium concentration quickly, there is severe risk of osmotic changes in the brain which can exacerbate the cerebral oedema, thus adding to potential brain damage.

At presentation a full clinical assessment of the infant is made. If the infant appears well, then slow rehydration at a rate of 100 $mL \cdot kg^{-1} \cdot d^{-1}$ can be carried out using expressed breast milk or proprietory milk or a combination of both. Although many mothers of infants with hypernatraemic dehydration choose to bottle feed their infants thereafter, it is possible to establish or re-establish successful breast-feeding once the child has been rehydrated[5].

If the child is unwell then rehydration should be carried out intravenously. Little has been published on recommended regimens for intravenous rehydration of such a child. In 1975 Banister *et al*[18] reported on the intravenous treatment of 38 infants with severe hyperosmolar dehydration and hypernatraemia. Infants rehydrated at a rate of 150 $mL \cdot kg^{-1} \cdot d^{-1}$ were more likely to develop convulsions and peripheral oedema than the infants whose fluid intake was restricted to 100 $mL \cdot kg^{-1} \cdot d^{-1}$.

A collapsed child may require to be resuscitated initially with 20 mL/kg of colloid or 0.9 % saline infused over half an hour. If the child is not in shock, then rehydration may be commenced intravenously using 0.9 % saline at 100 $mL \cdot kg^{-1} \cdot d^{-1}$ provided the plasma glucose concentration remains greater than 2.5 mmol/L. Plasma urea and electrolyte concentrations are measured 6-hourly. In our experience it is not uncommon to see the plasma urea concentrations fall quickly in the first 24 h but little change is seen in the plasma sodium concentration. After 24 h our regimen recommends continuing rehydration at the same rate, but using 0.45 % saline in 5 %-10 % dextrose. Thereafter oral rehydration with breast milk or artificial milk should be possible, with cautious increases in volume rates as the

plasma sodium concentration decreases to normal levels in the subsequent days.

DISCUSSION AND RECOMMENDATIONS

It is possible that hypernatraemic dehydration has always been a problem in breast-fed infants and has had inadequate exposure in the medical literature. It is also possible that this condition has become much more common in the last decade. Why should this be? Does it merely reflect an increased prevalence of breast-feeding in our community? This may be a component, but it is important to note that the medical literature on this subject does not come predominantly from Scandinavia or Australia where breast-feeding is much more widespread than it is in many parts of the world.

Although the increasing prevalence of breast-feeding in some areas is a probable contributory factor, shorter postpartum stays in hospital and under-resourced community supports may be the combination which is currently failing our newborn infants.

In the past, weighing infants in the community has received bad press from some who feel that the mother is put under more pressure to perform successfully, which in turn can raise her anxieties and impede her success in breast-feeding. Nevertheless, with discharge home occurring earlier, an adequate supply of weighing scales should be made available to community midwives so that they can monitor carefully the progress of any infants in the community who are small, preterm, or who have been identified as having some feeding difficulties. Risk charts may be drawn up as an educational tool.

We advocate that all breast-fed infants, irrespective of whether they are regarded as high or low risk for developing hypernatraemic dehydration, should be weighed at least once several days after discharge. Doctors, midwives, community nurses and health visitors should be trained to identify the ‘ at risk’ child, whose feeding is not progressing normally. We should be meticulous in taking a careful history of feeding frequency, length of time at the breast, adequacy of maternal milk flow, frequency of urinary passage, and the timing of changing stool from meconium to a transitional milk stool. Any infant who appears to have some difficulties in establishing successful breast-feeding should be identified and details entered on a risk chart. The hospital and community should then be committed to providing the resources to support the mother and child, including the provision of expert advice on breast-feeding, and weighing the child as often as is considered necessary by the lead professional. If there is a weight loss of greater than 10 %, the infant should be weighed on a daily basis until a normal growth velocity can be recorded.

CONCLUSION

Breast-feeding undoubtedly produces health advantages for infant and mother. We are right to promote expansion of breast-feeding in the developed and developing worlds. On the other hand, it is not acceptable to gloss over individual breast-feeding tragedies lest the resultant publicity discourages mothers from choosing to breast-feed their babies. Rather we must address the underlying problems which may arise during breast-feeding, identify resources of finance and expertise to eliminate these, and continue to recommend breast-feeding as the best method of nourishing healthy infants. Given the numbers of children involved, this topic must receive a very high priority in our health strategies.

REFERENCES

1 Kaplan JA, Siegler RW, Schmunk GA. Fatal hypernatremic dehydration in exclusively breast-fed newborn infants due to maternal lactation failure. Am J Forensic Med Pathol 1998; 19: 19-22.
2 Chambers TL, Steel AE. Concentrated milk feeds and their relation to hypernatraemic dehydration in infants. Arch Dis Child 1975; 50: 610-5.
3 Clarke TA, Markarian M, Griswold W, Mendoza S. Hypernatremic dehydration resulting from inadequate breast-feeding. Pediatrics 1979; 63: 931-2.
4 Rowland TW, Zori RT, Lafleur WR, Reiter EO. Malnutrition and hypernatremic dehydration in breast-fed infants. JAMA 1982; 247: 1016-7.
5 Thullen JD. Management of hypernatremic dehydration due to insufficient lactation. Clin Pediatr 1988; 27: 370-2.
6 Jaffe KM, Kraemer MJ, Robison MC. Hypernatremia in breast-fed newborns. West J Med 1981; 135: 54-5.
7 Roddey OF Jr, Martin ES, Swetenburg RL. Critical weight loss and malnutrition in breast-fed infants. Am J Dis Child 1981; 135: 597-9.
8 Marino R, Gourji S, Rosenfeld W. Neonatal metabolic casebook. Hypernatremia and breast feeding. J Perinatol 1989; 9: 451-3.
9 Ng PC, Chan HB, Fok TF, Lee CH, Chan KM, Wong W, *et al*. Early onset of hypernatraemic dehydration and fever in exclusively breast-fed infants. J Paediatr Child Health 1999; 35: 585-7.
10 Oddie S, Richmond S, Coulthard M. Hypernatraemic dehydration and breast feeding: a population study. Arch Dis Child 2001; 85: 318-20.

11 Livingstone VH, Willis CE, Abdel-Wareth LO, Thiessen P, Lockitch G. Neonatal hypernatremic dehydration associated with breast-feeding malnutrition: a retrospective survey. Can Med Assoc J 2000; 162: 647-52.
12 Cooper WO, Atherton HD, Kahana M, Kotagal UR. Increased incidence of severe breastfeeding malnutrition and hypernatremia in a metropolitan area. Pediatrics 1995; 96: 957-60.
13 van der Heide PA, Toet MC, Diemen-Steenvoorde JA, Renardel de Lavalette PA, de Jonge GA. Hypertonic dehydration in "silent" malnutrition of breast-fed infants. Ned Tijdschr Geneeskd 1998; 142: 993-5.
14 Paul AC, Ranjini K, Muthulakshmi, Roy A, Kirubakaran C. Malnutrition and hypernatraemia in breastfed babies. Ann Trop Paediatr 2000; 20: 179-83.
15 Harding D, Cairns P, Gupta S, Cowan F. Hypernatraemia: why bother weighing breast fed babies? Arch Dis Child Fetal Neonatal Ed 2001; 85: F145.
16 Chaudhary R, Twaddle S, Levi R, Haque K. Hypernatremic dehydration in breast-fed infants. Paediatr Today 1998; 6: 85-7.
17 Pickel S, Anderson C, Holliday MA. Thirsting and hypernatremic dehydration —a form of child abuse. Pediatrics 1970; 45: 54-9.
18 Banister A, Matin-Siddiqi SA, Hatcher GW. Treatment of hypernatraemic dehydration in infancy. Arch Dis Child 1975; 50: 179-86.
19 Colle E, Ayoube E, Raile R. Hypertonic dehydration (hypernatraemia): the role of feeding high in solutes. Pediatrics 1958; 22: 5.
20 Finberg I, Kiley J, Luttrell CN. Mass accidental poisoning in infancy. JAMA 1963; 184: 187-90.
21 Macy IG. Composition of human colostrum and milk. Am J Dis Child 1949; 78: 589-603.
22 Morton JA. The clinical usefulness of breast milk sodium in the assessment of lactogenesis. Pediatrics 1994; 93: 802-6.
23 Chilton LA. Prevention and management of hypernatremic dehydration in breast-fed infants . West J Med 1995; 163: 74-6.
24 Anand SK, Sandborg C, Robinson RG, Lieberman E. Neonatal hypernatremia associated with elevated sodium concentration of breast milk. J Pediatri 1980; 96: 66-8.
25 Peters JM. Hypernatremia in breast-fed infants due to elevated breast milk sodium. J Am Osteopath Assoc 1989; 89: 1165-70.
26 Arboit JM, Gildengers E. Breast-feeding and hypernatremia. J Pediatri 1980; 97: 335-6.
27 Clarke AJ, Sibert JR. Hypernatraemic dehydration and necrotizing enterocolitis. Postgrad Med J 1985; 61: 65-6.
28 Korkmaz A, Yigit S, Firat M, Oran O. Cranial MRI in neonatal hypernatraemic dehydration. Pediatr Radiol 2000; 30: 323-5.
29 Ernst JA, Wynn RJ, Schreiner RL. Starvation with hypernatremic dehydration in two breast-fed infants. J Am Dietet Assoc 1981; 79: 126-30.
30 Ghishan FK, Roloff JS. Malnutrition and hypernatremic dehydration in two breast-fed infants. Clin Pediatr 1983; 22: 592-4.