Hypernatraemic dehydration and breast feeding: a population study

S Oddie, S Richmond, M Coulthard

Abstract
As part of a population based regional review of all neonatal readmissions, the incidence of dehydration with hypernatraemia in exclusively breast fed infants was estimated. All readmissions to hospital in the first month of life during 1998 from a population of 32 015 live births were reviewed. Eight of 907 readmissions met the case definition, giving an incidence of at least 2.5 per 10 000 live births. Serum sodium at readmission varied from 150 to 175 mmol/l. One infant had convulsions. The sole explanation for hypernatraemia was unsuccessful breast feeding in all cases. The eight cases are compared with the 65 cases published in the literature since 1979. Presentation, incidence, risk factors, pathophysiology, treatment, and prevention are discussed. (Arch Dis Child 2001;85:318–320)

Keywords: hypernatraemia; dehydration; breast feeding; newborn; readmissions

Hypernatraemic dehydration has been reported to complicate exclusive breast feeding in 65 cases in the literature.1–26 Fits occurred in 17 cases, and substantial lasting morbidity2,7 11 21 and mortality have been reported.22 Following a population based review of readmission to hospital in the first 28 days of life, we report on infants presenting with dehydration and hypernatraemia and provide an estimate of the incidence of this condition in the UK.

Methods
All babies readmitted to any of the 14 hospitals in the former Northern Region before the age of 29 days during the calendar year 1998 were identified by interrogating hospital information departments. Babies reviewed in outpatient clinics, emergency departments, paediatric wards, or day units were not included unless they stayed overnight. Readmissions starting after midnight and before 0900 were included, even if the child was discharged on the day of admission. There were 32 015 live births in the Northern Region in 1998.

Information was collected from original hospital notes on the first readmission and the birth episode by a single auditor (SO). The infant’s day of birth was considered as day 1 if born before midday and as day zero if born later. A case was included if there was a history of poor feeding, with weight loss that was corrected by fluid replacement, and serum sodium was measured at 150 or greater.

Results
From a population of 32 015 live births, 1897 (5.9%) possible readmissions were identified. Notes were traced and examined for 1716 (90.5%) of these and 907 were true readmissions. Notes on 181 cases from the original list could not be located. If the notes found were representative of those missing, we retrieved about 90% of all true readmissions. If all the 181 missing notes were true readmissions, then we have reviewed the notes of 83% of readmissions, and if none were true readmissions we have reviewed 100%. It is likely that the true figure lies between these two extremes, and that it is near to 90%.

Thirty four readmissions had lost more than 10% of their birth weight, and sodium concentrations were recorded in all but four. Only eight had a serum sodium of 150 or greater. All eight were breast fed. These babies are the subject of this report. They presented at a median day 7 (range 6–10, see table 1). All were the first babies managed at home by their mothers and all were born at term (median 39.6, range 37–41 weeks). Two were below 3000 g at birth. A concerned midwife referred four infants for admission. It was unclear who initiated readmission in the remaining four cases. There was concern about jaundice in one case, vomiting in two cases, and poor feeding or poor weight gain in four. None had diarrhoea.

All lost at least 15% of their birth weight. One may have had fits. She had a serum sodium of 175 mmol/l at presentation, having lost more than 25% of her birth weight. She experienced a reduction in serum sodium from 175 to 170 mmol/l within eight hours of readmission to hospital. Cycling movements were noted which settled spontaneously six hours after readmission. Electroencephalography was not performed.

By 1 year, no baby was found to have a disorder that might have explained the hypernatraemia and none had died. In only four cases did the notes suggest that hypernatraemia had been recognised as a significant concern. Only

Table 1 Cases presenting with hypernatraemia on readmission

<table>
<thead>
<tr>
<th>Body wt (g)</th>
<th>Day of postnatal discharge</th>
<th>Day readmitted</th>
<th>% Wt loss (%)</th>
<th>Max [Na] (mmol/l)</th>
<th>Fully breast fed on second discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>4220</td>
<td>7</td>
<td>10</td>
<td>15</td>
<td>151</td>
<td>No</td>
</tr>
<tr>
<td>3100</td>
<td>Home delivery</td>
<td>6</td>
<td>16</td>
<td>160</td>
<td>Yes</td>
</tr>
<tr>
<td>3720</td>
<td>2</td>
<td>7</td>
<td>17</td>
<td>150</td>
<td>No</td>
</tr>
<tr>
<td>3495</td>
<td>3</td>
<td>7</td>
<td>18</td>
<td>152</td>
<td>No</td>
</tr>
<tr>
<td>2670</td>
<td>2</td>
<td>6</td>
<td>19</td>
<td>156</td>
<td>No</td>
</tr>
<tr>
<td>2780</td>
<td>3</td>
<td>6</td>
<td>20</td>
<td>163</td>
<td>?</td>
</tr>
<tr>
<td>3590</td>
<td>2</td>
<td>8</td>
<td>26</td>
<td>157</td>
<td>No</td>
</tr>
<tr>
<td>2350</td>
<td>4</td>
<td>10</td>
<td>27</td>
<td>175</td>
<td>No</td>
</tr>
</tbody>
</table>

Arch Dis Child 2001;85:318–320
one baby was breast fed at subsequent discharge.

Breast feeding rates in the Northern Region are low, being approximately 35% at first hospital discharge. First time mothers represent 32% of births. The incidence of hypernatraemic dehydration secondary to breast feeding difficulty of 2.5 per 10 000 live births can therefore be estimated to represent rates of 7.1 per 10 000 breast fed newborns, or 22.3 per 10 000 breast feeding first time mothers. Cases may have been missed because they occurred in infants before initial discharge home, or because appropriate investigations were not done. These figures therefore represent a minimum incidence.

Discussion

Hypernatraemic dehydration of breast fed infants has been described in 65 infants in 26 reports since 1979. Serious complications are reported, including fits, disseminated intravascular coagulation, multiple cerebrovascular accidents, and need for amputations.

In many cases the mother has been of higher than average educational attainment, with no practical experience of breast feeding but a strong desire to breast feed. Almost all cases have been to primiparous mothers; in very few has the mother successfully breast fed a previous baby. Occasional infants have had predispositions to feeding problems, such as Down’s syndrome or a cleft palate. Some mothers have had inverted nipples. Four cases occurred in hospital. However, most were normal term babies cared for by first time mothers at home.

Presentation is usually at around 10 days of age (range 3–21 days). Parents characteristically have little appreciation of their infant’s illness and may present after routine review, apparently unrelated consultation, or with an acute deterioration. The typical history is of an infant who is either excessively passive or who cries unusually. Some parents have been reassured by family or professionals. Urine output is low but may not be appreciated as such. An absence of the expected increase in stool frequency in the first few days of life is sometimes noted.

Clinical examination is often striking, with significant weight loss and an appearance which varies from alert and hungry, to moribund. Complications, most notably fits, characteristically occur during treatment rather than at presentation. Death is caused by cerebral oedema or infarction.

High sodium concentrations in the breast milk have been noted and considered causal. However, this may just be a marker of poor interaction between baby and breast, as suggested by a report of a disparity between sodium measurements in milk from one woman’s left and right breasts. Volumes of breast milk, when reported, are strikingly low. The poor satiety achieved by some of these infants, their poor urine output, and poor stool output all suggest that the problem is water deprivation, with secondary accumulation of sodium in an attempt to maintain circulating volume. In support of this hypothesis, a baby presented to one of the authors (SR) at 6 days of age having lost 20% of her birth weight and with a serum sodium of 158 mmol/l. Though she was bottle fed, no milk ever reached her stomach because she had oesophageal atresia.

In the 1970s gastroenteritis with hypernatraemia of relatively acute onset was a significant cause of mortality and neurological sequelae in infants. Intake of excess sodium in inappropriately prepared formula milks was blamed at the time, but a more plausible explanation is that continued feeding with milk after the onset of diarrhoea resulted in the delivery of a protein rich solution to the colon which, after digestion by colonic bacteria, produced a considerable osmotic load. The mean duration of symptoms was typically 2.5 days or 4 days. Animal experiments and some human data show that brain damage with acute hypernatraemia occurs as a result of brain cell shrinkage as the plasma osmolality rises too quickly for the protective mechanisms to take effect.

Hypernatraemia in breast fed infants is crucially different from that associated with gastroenteritis as it develops over a longer period, allowing the central nervous system to adjust to the increasing osmolality. In our cases the median time of presentation was day 7; it was day 12 in the cases from the literature (fig 1).

Slow onset hypernatraemia should be corrected slowly. Central nervous system cells can maintain intracellular volume despite slowly increasing plasma osmolality by manufacturing intracellular osmotically active substances (amino acids, trimethylamines, myo-inositol). Rapid correction of plasma osmolality without allowing sufficient time for the brain cells to dismantle these defences causes cell swelling (cerebral oedema), disruption of fine neuronal connections, and consequent brain damage. The speed with which the situation can be safely corrected is proportional to the speed with which it arose.
Magnetic resonance imaging spectroscopy in an infant with hypernatremia found these “idiogenic osmolytes” producing a calculated excess intracellular osmotic pressure of 17 mOsm/kg. The concentration fell as serum osmolality declined, but did not return to normal until 36 days after electrolyte correction was started.

In the published literature 17 infants had seizures, of whom 14 had a serum sodium above 165; only one was below 175 (the sodium concentration was not given in the remaining three infants). In 12 the convulsions occurred after treatment started. This is commented on in some reports. Seizures may have been related at least as much to the treatment of the hypernatremia, as to the hypernatremia itself. In many cases the hypernatremia is not extreme, and therapy may be less critical.

When discussing this condition with midwives we encountered considerable resistance to routine weighing of breast fed babies for fear that documenting weight loss would lead to abandonment of breast feeding. Some weight loss during the first week of life is physiologically, the information should be easily communicated to mothers. Exactly how much is “normal” is uncertain. Ensuring practical support for primiparous breast feeding mothers, both in hospital and at home, should prevent this problem occurring. Weighing of their infants in the first week of life, as suggested by the American Academy of Pediatrics working group on breast feeding, should allow early identification of those where breast feeding is not progressing adequately.

Sam Oldie is a member of the Tyne and Wear Health Action Zone breast feeding subgroup. We are indebted to Dr EN Hey for advice and for the data on normal weight loss in term breast fed babies shown in fig 1.

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Arch Dis Child 2001 85: 318-320
doi: 10.1136/adc.85.4.318

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