Hypernatraemic dehydration

A prospective study in children with diarrhoeal disease

I. D. HILL, M. D. MANN, M. D. BOWIE

Summary

Hypernatraemia occurs in a significant number of infants with dehydrating diarrhoea. There are some diagnostic clinical features, but these are not specific, and without routine electrolyte estimations many with hypernatraemia would go undetected. The standard fluid therapy schedule used at the Red Cross War Memorial Children's Hospital gave satisfactory results in both hypernatraemic and non-hypernatraemic patients. It is suitable for use in situations when electrolyte estimations on all patients are not possible.

Hypernatraemic dehydration with its associated high mortality and morbidity is a serious complication of diarrhoeal disease in infants and young children. It is reported to account for between 11% and 30% of hospital admissions for diarrhoea and to occur predominantly during winter. Improper therapy with fluids may be associated with convulsions or aggravate existing signs of central nervous system (CNS) dysfunction. Both the composition of the fluid and the rate of administration appear to be important in the pathogenesis of seizures.

Previous reports from the Red Cross War Memorial Children's Hospital, Cape Town, have been of a selected group of patients or of a limited number. Both groups of authors noted the difficulty of clinical diagnosis, contrasting with the experience of others.

This study was designed to determine the incidence of hypernatraemia in a large number of unselected patients admitted to this hospital with diarrhoeal disease. The clinical features were noted and an effort was made to define those useful in distinguishing hypernatraemic from non-hypernatraemic dehydration. The mortality, short-term morbidity and prognostic significance of convulsions were documented and assessed.

Patients

Between 1 March 1978 and 28 February 1979 serum sodium concentrations were determined on admission in all children with diarrhoea admitted to the drip room at this hospital. Those with an initial serum sodium concentration of greater than 150 mmol/l were referred to one of us (I.D.H.) for supervision of further management.

The age, sex and height were recorded. The infants were weighed on admission and at least daily thereafter until discharge. In the history, note was made of anorexia or previous excess salt or solute intake. Thereafter the patients underwent a full clinical examination, with particular attention to the degree of dehydration. This was estimated and recorded as 'not dehydrated', '5% dehydrated' or '10% dehydrated'. Those suspected of having a chest infection underwent chest radiography. Features of CNS dysfunction were categorized as drowsiness, coma, increased tone, hyperreflexia and convulsions. The rehydrated weight (the stable weight while not receiving intravenous therapy) was used to assess the nutritional status by means of the Boston percentiles of weight for age. The actual percentage dehydration at the time of admission was calculated in retrospect from the weight on admission and after rehydration.

Fifty consecutive children admitted between 09h00 and 17h00 from Mondays to Fridays, with an initial serum sodium concentration of less than 150 mmol/l, comprised the non-hypernatraemic group. They were assessed and managed in the same way as the hypernatraemic group.

Management

Acid-base status, serum electrolyte concentrations and urea levels were determined on admission and as often as necessary to monitor progress. Other investigations were performed when clinically indicated.

Both groups of patients received the same fluid therapy. An inadequate circulating blood volume, detected clinically by poor peripheral capillary perfusion, was restored with initial, rapid intravenous infusion of an isonatraemic plasma volume expander (Haemaccel). Thereafter these patients were managed in the same way as those not initially shocked.

Severe metabolic acidosis (pH < 7.25) was partially corrected with 8% sodium bicarbonate. The amount calculated to correct half the base deficit was given intravenously over a period of 5 minutes. After correction of shock all intravenous fluid given was half-strength Darrow's solution in 5% dextrose water. The calculated volume of fluid for rehydration was based on the clinical assessment of the degree of dehydration (i.e. 50 ml/kg if 5% dehydrated and 100 ml/kg if 10% dehydrated). This, together with a maintenance volume of 120 ml/kg body weight per day, was given at a constant rate over the next 24 hours. Oral half-strength Darrow's solution in 5% dextrose water was given at 3-hourly intervals starting 3-12 hours after admission, providing vomiting or abdominal distension did not occur. With the introduction of oral feeds the volume of intravenous fluid was decreased so that the total fluid intake was equal to the calculated daily requirement. The patients were reassessed every 3-4 hours and the fluid requirements were recalculated. On clinical and biochemical improvement oral feeds of a low-solute cow's milk formula (S26) were started about 24 hours after admission. Parenteral antibiotics were given only if clinically indicated.
Results

During the 12 months, 3889 children with diarrhoea were admitted to the drip room. Of these, 147 (3.8%) were hypernatraemic on admission. The monthly incidence of patients with hypernatraemia admitted is shown in Fig. 1.

Radiological evidence of consolidation was present in 21% of the hypernatraemic group, whereas this was so in only 2% of the non-hypernatraemic group \((P < 0.01)\).

Symptoms of CNS dysfunction are detailed in Table IV. Abnormal findings were noted in 38% of the hypernatraemic patients, but in only 4% of the non-hypernatraemic children \((P < 0.001)\).

The age distribution is shown in Table I. A significantly greater proportion of the patients in the hypernatraemic group was below 6 months of age \((P < 0.01)\). No sex predilection was evident and no statistically significant difference in nutritional status was demonstrated between hypernatraemic and non-hypernatraemic patients (Tables II and III).

TABLE IV. PATIENTS PRESENTING WITH CNS DYSFUNCTION IN HYPER- AND NON-HYPERNATRAEMIC DEHYDRATION

<table>
<thead>
<tr>
<th></th>
<th>Hypernatraemia</th>
<th>Non-hypernatraemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drowsy, but rouseable</td>
<td>32</td>
<td>2</td>
</tr>
<tr>
<td>Jittery, hypertonic or hyperreflexic</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td>Coma and/or convulsions</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>56</td>
<td>2</td>
</tr>
</tbody>
</table>

No. of cases assessed 147 50  
% positive 38 4

The clinical estimate of the dehydration was compared with the actual degree of dehydration calculated in retrospect after recovery. Underestimation of dehydration was recorded when the actual dehydration exceeded the clinical estimate by more than 2.5%. In the hypernatraemic group dehydration had been underestimated in 72.5% of cases. This differs significantly from the 36% in the non-hypernatraemic group \((P < 0.001)\). Dehydration in the hypernatraemic group was frequently grossly underestimated (Table V).

TABLE V. UNDERESTIMATION OF DEHYDRATION BY CLINICAL CRITERIA IN HYPER- AND NON-HYPERNATRAEMIC PATIENTS

<table>
<thead>
<tr>
<th>Degree of underestimation</th>
<th>Hypernatraemic group (138*)</th>
<th>Non-hypernatraemic group (50*)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5% - 4.9%</td>
<td>40</td>
<td>11</td>
</tr>
<tr>
<td>5.0% - 9.9%</td>
<td>53</td>
<td>7</td>
</tr>
<tr>
<td>&gt;10%</td>
<td>7</td>
<td>0</td>
</tr>
</tbody>
</table>

Number of patients assessed in each group.

Eight deaths occurred in the hypernatraemic group, a mortality of 5.4%. In 2 patients, neither of whom had had convulsions or was in coma before admission, there was extensive suppurative pneumonia post mortem, and death could not be attributed directly to the hypernatraemia. The other 6 infants had had convulsions or were in coma before admission. Postmortem examination in 4 showed gross cerebral oedema with associated cerebral vein thrombosis and intracranial haemorrhage. Consent for postmortem examination was refused in the remaining 2.

There were no deaths in the non-hypernatraemic group studied. Among all the non-hypernatraemic children admitted to the drip room (3742) during the year there were 10 deaths, a mortality of 0.3%. This is significantly lower than that for the hypernatraemic infants \((P < 0.001)\).

Six of the hypernatraemic patients had convulsions for the first time after rehydration with intravenous fluids was started. A definite history of anorexia was obtained in 23.8% of the hypernatraemic group, and 19.9% had a history of excessive salt or solute intake before admission. The corresponding figures for the non-hypernatraemic group were 12% and 8% respectively. These differences were not statistically significant \((P > 0.05)\).
The time of onset of the seizures ranged from 4 to 36 hours after admission. Two of these infants had a cerebrospinal fluid pleocytosis compatible with an aseptic meningitis. In a third case the serum calcium level was very low and convulsions did not recur after correction of this. No obvious relationship between the amount of fluid already administered or the serum sodium level at the time of the convolution was noted. None of the non-hypernatraemic patients had convulsions during recovery from their diarrhoea.

Three children in the hypernatraemic group were assessed as having residual CNS dysfunction on discharge. One, who had had convulsions while on intravenous fluid therapy, developed a right hemiparesis. Seven months later there had been marked improvement, but slight right-sided weakness still remained. The other 2 had not had convulsions, but were generally hypotonic on discharge. At follow-up examination 1 month later they had improved considerably, but were not completely normal. There was no evidence of CNS dysfunction in the non-hypernatraemic group on discharge.

Discussion

The incidence of hypernatraemia is lower than that reported by others, but similar to that previously reported from this hospital. Recent reports suggest that there has been a decline in the incidence of hypernatraemia. There is some conflict of opinion as to whether the increased use of low-solute cow's milk formulas is responsible for the change. Much of the reported variation in incidence is due to the selection of patients studied, but this series was unselected and all patients presenting to the hospital with dehydrating diarrhoea were included. The seasonal variation described in other areas was not found.

Our experience that early recognition of the hypernatraemic child by clinical means remains difficult is shared by some authors. Comparison of the two groups shows that there are certain features which, if present in the infant with diarrhoea, should arouse the suspicion that the patient may be hypernatraemic.

The younger child is particularly at risk and the majority of patients are under 6 months of age. An associated pneumonia is important and may play a role in the pathogenesis of the condition. The hyperpnoea accompanying respiratory infection aggravates water loss from the lungs. The most useful signs are those of CNS dysfunction, drowsiness being the most common abnormal finding. This has been noted by other authors, but in our experience such features did not occur as frequently as reported.

Coma and convulsions before admission and initiation of treatment appear to be poor prognostic features. Six of the 9 infants who presented with one or the other subsequently died; a similar finding was recorded by Weil and Wallace. These were probably manifestations of irreversible structural brain damage; postmortem findings support this belief. Convulsions occurring during the course of treatment do not appear to have the same poor prognostic significance. They are usually of short duration and easily stopped with anticonvulsants. They have been attributed to rapid shifts of water, with the production of cerebral oedema. In this series no relationship to timing or stage of rehydration and serum electrolyte concentration was found. These late-onset convulsions may be associated with morbidity, as in the child with the residual right hemiparesis, and may not always be due to the hypernatraemia, since other possible causes were identified in 3.

Our findings indicate that a large proportion of hypernatraemic individuals will initially go undetected if the serum sodium concentration is not checked in all patients admitted with diarrhoea. This is often not possible as a routine. It is, therefore, imperative to have a fluid therapy regimen that meets the needs of the non-hypernatraemic child and also minimizes the risk of iatrogenic complications in the hypernatraemic infant.

A potential practical problem in management is the tendency to underestimate the degree of dehydration in hypernatraemia. The signs of dehydration are directly related to the volume of water lost from the extracellular fluid compartment. In the hypernatraemic patient the extracellular fluid volume is relatively spared at the expense of the intracellular fluid volume; thus masking the degree of dehydration. We believe that this underestimation may be to the patient's advantage. By aiming to correct the clinical degree of dehydration over a 24-hour period, one would in fact be correcting the actual dehydration over a period of up to 48 hours. This meets the need for unhurried correction of the fluid deficit, which has been amply emphasized.

The present fluid therapy regimen has proved suitable for infants with non-hypernatraemic dehydrating diarrhoea. It is also suitable for infants with hypernatraemic dehydration, since it is associated with a lower mortality rate than that reported by others and a relatively low risk of iatrogenically induced complications.

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REFERENCES