Dexamethasone in the treatment of hypernatraemic dehydration

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SUMMARY Ninety infants with severe hypernatraemic dehydration (plasma sodium > 150 mmol/l) were studied. Most had had a convulsion before admission. They were allocated to two treatment groups. Both groups received intravenous plasma followed by slow intravenous rehydration and correction of acidosis. In addition, one group received intramuscular phenobarbitone, the other group received dexamethasone 0.3 mg by intramuscular injection every 6 hours for 48 hours. Fewer infants receiving dexamethasone had convulsions during treatment (18% compared with 52%), and fewer (18%) of them died than in the group who did not receive dexamethasone (40%). Dexamethasone may have a role in the management of hypernatraemic dehydration in infants.

Since the introduction of low-solute milks the incidence of hypernatraemic dehydration has fallen in western countries; however in developing countries ignorance and the increasing use of dried cows' milk has resulted in a large number of cases with high morbidity and mortality. Generally a child is brought to hospital having had at least one convulsion before arrival. As the risk of further convulsions was not being reduced by our standard treatment,¹ it was decided to use dexamethasone in an attempt to reduce cerebral oedema and prevent further convulsions.

Patients and methods

During an 11-month period, 90 infants suffering from hypernatraemic dehydration (plasma sodium > 150 mmol/l and a calculated plasma osmolality > 350 mmol/kg) were studied. None of the infants was known to have a history of cerebral or renal abnormality; each had been admitted with gastroenteritis and had moderate or severe dehydration—that is 10 or 15%. Each infant was allotted alternately to one of two treatment groups (Table 1). Each had a lumbar puncture to exclude meningitis (2 cases were excluded), 4 infants had pneumonia in addition to gastroenteritis, and are included in the study. Infants were weighed on admission and then daily. Plasma urea, electrolytes, calcium, and glucose were measured on admission and then daily, or more frequently if necessary. Neurological state was graded according to Banister—that is 0 = nothing unusual; 1 = irritable, or poorly responsive and

Table 1 *Treatment regimen*

All infants
If circulatory failure, intravenous plasma (20–30 ml/kg) was given promptly
Intravenous 0.18% saline in 5% dextrose at 100 ml/kg per 24 hours corrected weight. (If oral intake introduced then the infusion rate was reduced accordingly but the total amount of fluid given over 24 hours remained the same)
Acidosis half corrected by 8.4% sodium bicarbonate if base deficit > 10 mmol/l or if standard bicarbonate < 13 mmol/l
Potassium chloride 26 mmol/l added when urine passed by the patient unless level of plasma potassium was high
Group 1
Phenobarbitone 15 mg intramuscularly every 12 hours
Group 2
Dexamethasone 0.3 mg intramuscularly every 6 hours

irritable when disturbed; 2 = increased tone with or without neck stiffness; 3 = jittery movements, with or without nystagmoid eye movements; 4 = convulsing.

Results

Details of patients and their investigations are shown in Table 2. There were 45 infants in each group, all of them were 10% or more dehydrated, thus all required initial plasma resuscitation. 84% had convulsed before admission. After admission 18% of infants in group 2 (who had dexamethasone) had one or more convulsions compared with 52% in group 1 (Table 3).

Hypocalcaemia. In only 2 infants did the plasma calcium fall below 7 mg/100 ml (1.75 mmol/l); neither infant showed any sign of hypocalcaemia

Table 2 Details of the two groups of infants

	Group 1 (n=45)	Group 2 (n=45)
Mean age (weeks)	9±4	11±2.4
Plasma sodium (mmol/l)	178±12	174±14
Plasma osmolality*	388±32	390±20.2
Blood urea (mg/100 ml)	124±32	142±42.6
Dehydration 10% or more	45	45
Plasma resuscitation necessary	45	45
Convulsion before admission	39 (87%)	37 (82%)
Mean neurological grade on admission	3.1	3.6

Conversion: traditional to SI units—blood urea: 1 mg/100 ml ≈ 0.166 mmol/l.

*Osmolality was calculated⁸ as follows:

Na (mmol/l) × 1.9 + K (mmol/l) × 1.9 + urea (mg/100 ml)/60 × 10 + glucose (mg/100 ml)/180 × 10.

Table 3 Progress during treatment

	Group 1 (n=45)		Group 2 (n=45)	
	No	(%)	No	(%)
Average percentage weight increase in first 24 hours	7.5	±2.5	6.3	±2.7
No of infants who had convulsions during treatment	23	(52)	8	(18)
No of infants who died	18	(40)	8	(18)
			P<0.001	

but each was given intravenous calcium. Both infants were in group 2 receiving phenobarbitone.

Hyperglycaemia. 13% of infants had high levels of blood sugar (>200 mg/100 ml; >11.1 mmol/l) none was given insulin, and the level of blood glucose fell rapidly during rehydration.

Anaemia. Most of the infants in this hospital are anaemic, and these 90 infants had a mean Hb of 8.6 (range 5.6–12.8) g/dl. 22 infants were each given a blood transfusion during their stay in hospital.

Discussion

Neurological complications dominate the clinical picture of hypernatraemic dehydration. The risk of permanent damage is greater in the young infant² and in patients with serum sodium of 160 mmol/l or more.³ Permanent neurological deficit is said to occur in 11 to 15% of such infants.⁴ Convulsions in hypernatraemia are thought to be due to cerebral oedema.^{5–7}

Our trial showed that dexamethasone used in conjunction with slow rehydration (0.18% saline in 5% dextrose) was associated with fewer convulsions and a lower mortality rate. It seems that the mortality rate may be related to reduction of the cerebral oedema. Our mortality rates are still unacceptably high but this is due mainly to the delay in the infants coming to the hospital; 84% had convulsed before coming to the hospital. Nevertheless, the 18% mortality rate of the dexamethasone group is similar to that reported 16 years ago in the UK.² This is remarkable since most of our infants arrived in moribund condition or were malnourished.

Although we feel that dexamethasone may have a role in the management of hypernatraemic dehydration, the best answer lies in prevention and in trying to undo the damage which bottle feeding has caused in developing countries. Further education of the mothers is needed together with restriction of high-solute milk products.

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