

Case Report

Dying for milk: A neonate with severe hypernatremia associated with inadequate breast feeding

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ABSTRACT

Inadequate breastfeeding may result in malnutrition, hypernatremic dehydration and catastrophic outcomes. We describe a case of severe breast feeding associated hypernatremia which was complicated by acute seizures and severe hyperglycemia. The baby's condition was initially confused with neonatal diabetes mellitus.

Keywords:

Breast feeding; Hypernatremia; Hyperglycemia; Milk.

INTRODUCTION

The benefits of breast feeding are well established and include decreased incidence of many acute infections and chronic diseases as well as improved neurodevelopmental outcomes [1]. However, insufficient breast feeding has serious complications, the most important of them is severe hypernatremic dehydration [2]. Hypernatremia is assumed to be a rare complication of breast feeding [3], but recent reports have suggested that the incidence is rising [4]. The failure to diagnose hypernatremic dehydration can have serious consequences, including seizures, intra-cerebral haemorrhage, vascular thrombosis and death [5,6]. In this case report, we describe a case of severe hypernatremic dehydration associated with severe hyperglycaemia in a neonate who is exclusively breast fed, with a review of the literature.

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CASE REPORT

A 12-day-old Saudi male infant was admitted to the Neonatal Intensive Care Unit (NICU) of Al Rass General Hospital, Qassim, Saudi Arabia, due to fever, poor feeding, lethargy and decreased urine output for the previous 24 hours. No history of diarrhoea or vomiting. The patient was born to a 21-year-old primigravida healthy mother via spontaneous vaginal delivery at full term. Apgar scores were 9 and 10 at 1 and 5 minutes, respectively. The pregnancy was normal and mother had prolonged rupture of membranes (PROM) for more than 18 hours. The baby was admitted to the special care baby unit and started on intravenous ampicillin and cefotaxime after blood samples were taken for blood culture. Antibiotics were given and stopped after 3 days when the blood culture was negative and the patient was discharged on exclusive breast feeding 12 – 14 times per day and was reported as feeding well. On

admission at 12 days of age, he was ill, lethargic and had signs of severe dehydration. He had no cyanosis or jaundice. His vital signs were: Temperature of 36 degree centigrade, heart rate 120/minute, respiratory rate 50/minute, blood pressure 80/50 mmHg and an oxygen saturation of 98% at room air. His body weight on admission was 2.3kg, which indicates 33% weight loss compared with the birth weight of 3.4Kg. The skin was doughy and capillary refill time was more than 2 seconds. Examination of the heart, lung and abdomen was unremarkable. On neurological examination he was lethargic but with no focal neurological sign and his primitive reflexes were normal. Laboratory findings on admission showed severe hypernatremia (serum sodium of 191 mmol/L), severe hyperglycaemia (blood glucose level of 54 mmol/L), pre-renal azotemia and thrombocytopenia (Table I).

Table 1 - Laboratory results at presentation

Investigation	Value	Normal range
Sodium	191 mmol/L	133–142 mmol/L
Potassium	6.4 mmol/L	3.7–5.2 mmol/L
Blood urea	60.8 mmol/L	8–28 mmol/L
Serum creatinine	752 mcmmol/L	17.7–88.4 mcmmol/L
Serum chloride	144 mmol/L	95–105 mmol/L
PH	7.25	7.32–7.43
Bicarbonate	13.2 mmol/L	17.2–23.6 mmol/L
Blood sugar	54.36 mmol/L	3.8 – 6.1 mmol/l
Uric acid	1320 umol/L	119–416 umol/L
Calcium	2.08 mmol/L	0.90–1.45 mmol/l
Hemoglobin	17.6 gram/dL	10.5–14.0 gram/dl
White blood cell count	15.800/mm ³	9.1–34.0/ mm ³
Platelets	82.000/mm ³	150,000–450,000/ mm ³
International normalized ratio (INR)	0.58	0.8–1.2

He was diagnosed as severe hypernatremic dehydration, pre-renal failure associated with severe hyperglycaemia due to insufficient breast milk feeding. He was given a bolus of isotonic saline (Normal Saline) 20ml per kg over 60 minutes, then the

free water deficit and sodium excess were managed by gradual and slow correction over 72 hours. The calculations used to correct the severe dehydration are shown in (Table 2).

Table 2 - Fluid deficit calculations

<ul style="list-style-type: none"> <input type="checkbox"/> Birth weight = 3.4 kg, admission weight = 2.30kg <input type="checkbox"/> Weight loss 1.1kg, percent dehydration = $1.1/3.4 = 0.32 = 32\%$ <input type="checkbox"/> Free water deficit = weight loss = 1100ml
<p>Fluid therapy:</p> <p>- Step I: Emergency phase:</p> <ul style="list-style-type: none"> <input type="checkbox"/> Restore vascular volume with a bolus of 20ml/kg of 0.9% saline $20\text{ml} \times 3.4\text{kg} = 68\text{ml}$ over 1 hour <p>- Step II: Rehydration phase:</p> <p>Aim to correct water deficit and sodium excess within 48-72 hours</p> <ul style="list-style-type: none"> <input type="checkbox"/> Maintenance during 3 days: $100\text{ml /kg/day} \times 3.4\text{kg} \times 3 = 1020 \text{ ml}$ <input type="checkbox"/> Maintenance + deficit = $1020 + 1100 = 2120\text{ml}$ minus initial bolus = $2120 - 68 = 2052 \text{ ml}$ <input type="checkbox"/> $2052\text{ml}/71 \text{ hours} = 29 \text{ ml /hour}$ of 5% dextrose/0.45% normal saline

With respect to his ill appearance and the thrombocytopenia, sepsis was suspected and intravenous (IV) antibiotics were administered until the blood and urine cultures were reported as negative. Likewise, due to the severe hyperglycaemia, he was suspected to be suffering from neonatal diabetes mellitus and so he was started on regular insulin infusion 0.1 units per kg per hour. After the normal saline (NS) bolus, he passed a 25 ml of clear urine, which was analysed and showed PH of 5.0 specific gravity of 1.025, urine osmolality of 786 mosm/kg and no pus or red blood cells or proteins. Four hours

after the initiation of rehydration he developed a generalized tonic-clonic (GTC) convulsion and he was loaded with intravenous (IV) phenobarbitone; and a repeat chemistry revealed rapid drop of sodium and glucose levels (sodium 173 mmol/L and glucose 28 mmol/L, respectively) with mild hypocalcaemia. Insulin was discontinued and IV fluids changed to dextrose 5% + 0.45% normal saline ((D5 + ½NS). However, 6 hours later, he again developed a generalized tonic-clonic convulsion and became more lethargic, so he was given 10 ml of 3% sodium chloride and mechanically ventilated. Cranial computed

tomography (CT) showed mild cerebral edema but no bleeding. With this IV fluid regimen, the sodium and glucose concentrations decreased gradually over 48 hours; sodium 147 mmol/L, blood urea nitrogen

(BUN) 19 mmol/L, creatinine 61 mmol/L and glucose 8 mmol/L. After 72 hours his sodium, renal parameters and glucose levels were back to normal (Figure I).

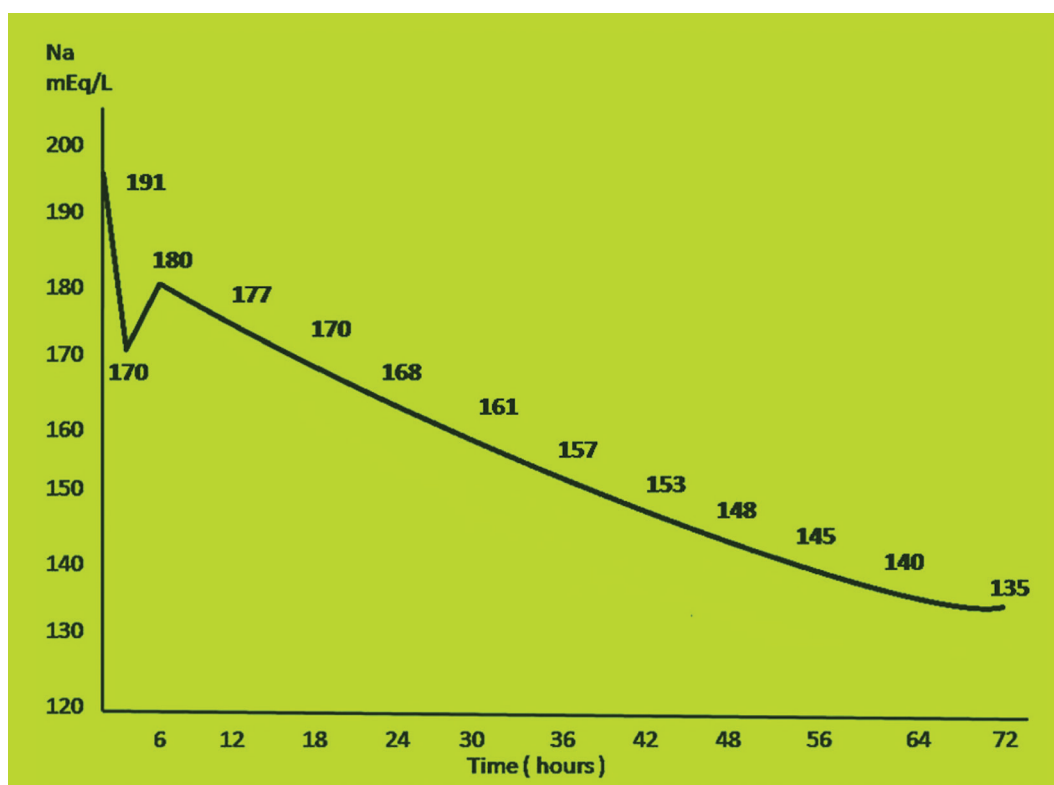


Figure 1 - The changes in serum sodium concentrations during fluid therapy

On the 3rd day he was started on naso-gastric tube feeding and he was extubated on the 5th day. He developed hypo-albuminic edema that was managed with albumin and frusemide. He was discharged after 18 days of admission with normal neurological examination, feeding on an infant formula and with a discharge weight of 3.2kg. He was readmitted at the age of 2.5 months with an acute viral gastroenteritis with mild dehydration, his body weight was then 4kg and he had normal sodium, glucose, calcium and renal parameters. Currently he is 8 years old with normal growth and development.

DISCUSSION

Hyponatremia was thought to be unusual in breast fed babies. During the decade 1979-1989 sporadic reports of breast feeding associated hyponatremia (BFAHN) appeared in the literature [7]. In the 1990s there was an increase in the reported cases. Cases were reported from Ohio, the Netherlands, Hong Kong, India and England [8-12]. Harding et al [13] estimated that in Bristol they saw one case per month. In the United States, reports were not confined to the medical literature. The Wall Street, The Times and various American television programs highlighted the new

epidemic [14]. During the period 1979-1990s, BFAHN was reported in 65 infants. Serious complications were reported such as seizures, disseminated intravascular coagulation (DIC), cerebrovascular accidents (CVA) and even deaths. In many cases, the mothers were of higher than average educations and were primiparus with no experience of breast feeding but a strong desire to breast feed. Four cases occurred in the hospital. Parents characteristically had little appreciation of their infant illness, many presented after a routine review, unrelated consultation or with an acute deterioration. The clinical features were striking, with marked weight loss and an appearance that varies from alert and hungry to moribund. Complications, especially convulsions, occurred during treatment rather than at presentations [15]. Our patient had the same scenario of presentation. A recent population based study found an incidence of 2.5/1000 births which equals to 7.1/1000 breast fed babies, equals to 23.3/1000 of breast fed, fist time mothers [16]. Moritz et al [17] in the largest report over 5 years in 3718 hospitalized neonates, found BFAHN in 70 (1.9%) which is significantly higher than the reported incidence of hypernatremia due to all causes among hospitalized children and adults (1.1%). In a more recent population based study of severe neonatal hypernatremia, Oddie et al [18] found an incidence of 7 per 100,000 over a one year period in the UK and the Republic of Ireland. Despite of the common occurrence of the BFAHN, only very few cases were reported from the Gulf countries [19,20]. In general, hypernatremia may be caused by decreased fluid intake, increased fluid loss or increased salt intake. In some cases of BFAHN, high concentration of the sodium content of the breast milk was noted and considered causal [21], however volumes of milk when reported are strikingly low, and the poor satiety, coupled with poor urine and stool outputs, suggest water deprivation with secondary accumulation of sodium to maintain circulation volume as the cause of

BFAHN [22]. Sodium content of breast milk at birth is high and declines rapidly over the subsequent days. Morton [23], studied the breast milk of 130 women as they began to breast feed; women who failed to establish a good breast feeding did not show the normal decline in sodium content. Primary insufficient lactation is rare. Poor milk production is usually due to secondary insufficient lactation caused by poor milk removal from the breast [24]. Hypernatremia may be associated with hyperglycaemia and mild hypocalcaemia, the mechanism of which is not known [25]. Hyperglycaemia was noted in 13 out of 42 reported cases of BFAHN by Van Amerongen et al [26]. Our reported case had severe hyperglycaemia (54.36 mmol/L), and this was confused with neonatal diabetes mellitus and inappropriately treated with insulin, which contributed, to the rapid decline of osmolality and the development of the seizures. The severe hyperglycaemia associated with hypernatremia should be distinguished from the neonatal diabetes mellitus, the treatment should not include insulin, only rehydration will correct the hyperglycaemia [25]. Another factor led to the development of seizures in our case was the initial use of a hypotonic solution (D5 + ¼ NS), which led to rapid decline of serum sodium from 191 to 171 mmol/l over 2 hours. Actually, the main stay of treatment is slow hydration after a bolus of NS, followed by NS in the 1st 24 hours and this to be changed to ½ NS in the next 48 hours. Banister et al [27], reported on the IV treatment of 36 infants with hypernatremic dehydration, infants who were rehydrated at a rate of 150 ml per kg per 24 hour were more likely to develop convulsions and peripheral edema than those who were given 100 ml per kg per 24 hours. BFAHN is usually associated with acute morbidity and mortality including seizures, apnoea, bradycardia, facial palsy, necrotizing enterocolitis, limb amputation, cerebral haemorrhage and infarction [28]. Salih et al [29] reported 3 cases of stroke due to hypernatremic dehydration out of 104 case of

stroke due to different causes. Recent prospective data revealed that more than half of patients exhibited abnormal development with long-term follow up [30]. Our patient developed only acute seizures during the rehydration phase but he was developing normally at 8 years of age. In a 5-year study of neurodevelopmental outcome of 182 neonates born at or near term, hypernatremic dehydration was not associated with adverse neurodevelopmental outcomes [31]. BFAHN should be totally preventable. One strategy is the judicious use of supplemental feedings. Indications for considering supplemental feeding for breastfed neonates include weight loss more than 7%, jaundice approaching or at phototherapy levels, low urine output, decreased stool frequency, lethargy, agitation or inconsolable crying, feeding difficulties,

and evidence of delayed lactogenesis [32]. This approach has been used successfully in Switzerland, supplemental feedings were given to infants when weight loss was near 10% or there were breast feeding difficulties [33]

CONCLUSION

Breast feeding associated hypernatremia (BFAHN) can cause life-threatening complications in neonates, such as seizures and severe hyperglycaemia, which can be confused with neonatal diabetes mellitus as seen in our patient. These complications and those reported in the literature suggest close monitoring of weight and hydration status of exclusively breast fed babies and earlier intervention if weight loss exceeds 10% of birth weight.

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