Successful Treatment of Neonatal Extreme Breastfeeding-Associated Hypernatremia

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Abstract

Exclusive breastfeeding is a leading cause of hypernatremia and dehydration. We describe successful treatment in a case of extreme breastfeeding hypernatremia in a neonate, presenting with serum sodium up to 232 mmol/L, acute renal failure and a rare complication of sinus vein thrombosis. With careful treatment, there were both clinical and laboratory improvement, with no further short-term complications proving that with attention, even extreme hypernatremia is treatable.

Keywords

Breastfeeding associated hypernatremia, Hypernatremia, Exclusive breastfeeding

Abbreviations

BFAH: Breastfeeding Associated Hypernatremia; PICU: Pediatric Intensive Care Unit

Introduction

Hypernatremia defined as serum sodium > 145 mmol/liter (neonatal hypernatremia is defined as serum sodium > 150) is one of the most common electrolyte disorders and is often due to net water loss. Since sodium is a functionally impermeable solute, high levels of extracellular sodium induce the movement of water across cell membranes resulting in cellular dehydration [1].

In the past, diarrheal dehydration was the most common cause for hypernatremia in infants [2]. Recent studies show that inadequate fluid intake and an iatrogenic cause are the leading causes for hypernatremia in infants today and is often accompanied by comorbidities [3]. Breastfeeding-associated hypernatremia (BFAH) is caused by insufficient breastfeeding due to maternal or infantile reasons and is aggravated when less milk is produced thereby resulting in higher sodium content [4]. Though different studies [4-6] place the incidence of BFAH at 0.1%-2.7% of term babies, some estimate that up to 10% of exclusively breastfeed babies suffer from BFAH [4]. During the last decades both exclusive breastfeeding and the incidence of BFAH has risen [4,7]. Morbidity is significant but mortality is very low when appropriate treatment is promptly administered [4,7].

Case Report

MN, an eight day-old male, presented at our emergency room with extreme hypernatremia and was admitted to the pediatric intensive care unit (PICU) for treatment and monitoring. The pregnancy was unremarkable and the patient was born during the 39th week of gestation following normal vaginal delivery, and a large for gestational age (LGA) birth weight of 4160 grams. He was the first child to young non-consanguineous parents and was exclusively breastfed at home.

During the three days prior to his admission, his parents reported poor appetite and weak sucking. On the day before presentation, he developed fever of up to 38.5 °C with cyanosis, but according to the family he had a fair amount of urine. There were no gastrointestinal...
symptoms. Additionally, the baby began grunting on the
day of presentation and felt very cold to his parents. He
was subsequently taken to an outpatient clinic of a local
general hospital.

At first assessment, he was found to be tachypneic,
cyanotic, stiff and irritable, with a prolonged capillary
refill time of 6 seconds. His vital signs were a heart rate
of 155 bpm, blood pressure was unmeasurable and his
core temperature was 33.4 °C. He weighed 2900 g (a 30% decrease from birth weight). His skin was dry, doughy
with a decreased turgor and his mucous membranes
were extremely dry. The examination of his heart, lungs
and abdomen was unremarkable.

Neurologically, his fontanelles were flat, he was irri-
table, with hyperflexion of his upper limbs, and though
his Moro reflex was normal, he had a decreased sucking
reflex.

Laboratory tests were performed including basic
chemistry, complete blood count, arterial blood gases,
and blood cultures. He was immediately started on
normal saline fluid resuscitation, cefotaxime, ampicillin
and oxygen, and was placed in an incubator for controlled
elevation of body temperature. After fluid expansion with
40 cc/kg, his blood pressure could be measured (73/40
mmHg) and his core body temperature improved.

Initial laboratory results showed a sodium level of 200
mmol/liter, potassium of 5.4 mmol/liter, chloride of 152
mmol/liter, creatinine of 528 micromol/liter, BUN of
147 mg/dL and albumin 42 gram/L. He had a metabolic
acidosis, with a pH of 7.17.

After calculation of water and sodium deficit, he was
continued on normal saline at a rate of 38.5 cc/hr and was
transferred to our hospital’s PICU. The parents reported
no use of milk formula, additional salt in the patient’s
diet, or any other additives.

On presentation at the PICU, he was alert, with vital
signs as follows: heart rate 130 bpm, blood pressure 70/40
mmHg, 36.5 °C core body temperature and O₂ saturation
in the 90 s, with a weight of 3100 g. He was treated
according to the standard protocols for the treatment of
hypernatremia. His water deficit was calculated around
700 cc and intravenous normal saline-half his deficit plus
maintenance-was administered over 24 hours at a rate of
30 cc/hr. Electrolytes were sampled every two hours, and
sodium concentration was normalized over the course of
six days (Figure 1). The infant’s kidney function returned
to normal five days after presenting to our hospital. After
correction of the severe dehydration, albumin levels
dropped to 28 gram/liter, most likely because the first
measured value was due to hemoconcentration, and did
not accurately represent albumin levels.

Several hours after admission, MN developed focal
tonic seizures. His fontanelles were normotensive at this
time and there were no changes in his primitive reflexes.
He was treated with a loading dose of phenobarbital.
Sodium at the time of the seizures was 180 mmol/L.
Glucose and calcium levels were in the normal range.
Brain computed tomography scan (without contrast)
completed an hour after the seizures began, showed
hyperdensity within the superior sagital sinus extending
from the frontal region through the posterior aspect of
the vertex and associated with hyperdensities within
the bilateral frontal vertex feeding veins suggesting a
sinus vein thrombosis which was later confirmed by
magnetic resonance imaging (MRI). No central pontine
myelinolysis or brain edema was noted and was then
started on a therapeutic dose of enoxaparin.

It should be noted that although the overall reduction
in sodium concentration proceeded as expected, there
were two abnormal sodium results, 230 mmol/L and 210
mmol/L at 2 and 2.5 hours respectively after admission
to PICU. Diabetes insipidus and hormonal causes for
hypernatremia were ruled out. It is important to note
that as his mother’s first-born child who was exclusively
breastfed, MN was at risk for hypernatremia.

A month after first presenting to our hospital MN was
discharged with no gross neurological deficit. The par-
ents were counseled as to proper feeding for their child,
and were guided to feed him with both breastfeeding and
milk formula. At follow up six months after his first ad-
mission, he was, again, grossly neurologically intact.

Discussion

The advantages of breastfeeding are many and include
reduced risk for infections, allergic diseases, diabetes,
obesity, malignancy, sudden infant death and necrotizing
enterocolitis. Further, there is lower overall mortality in
breastfed infants as well as short- and long-term maternal
benefits [8,9].

A 2012 Cochrane review [9] determined that the op-
timal duration of exclusive breastfeeding is six months.
Breastfeeding-associated hypernatremia is caused by
insufficient breastfeeding due to maternal or infantile
reasons [4] though studies have shown an incidence of
up to 2.7%—this is probably an underestimation. During
the last decades both exclusive breastfeeding and the in-
cidence of BFAH has risen, in all likelihood due to the
public’s response to the strong recommendations sup-
porting exclusive breastfeeding for nearly all newborns
[4,7,8].

Incidence is higher with primiparous mothers, uned-
ucated mothers or when there are difficulties with breast-
feeding, excessive room heating, oliguria, and inade-
**Figure 1A:** Serum biochemistry during hospitalization in the PICU - serum sodium (mmol/L) in hours.

**Figure 1B:** Serum biochemistry during hospitalization in the PICU - serum creatinine (mmol/L) in hours.

**Figure 1C:** Serum biochemistry during hospitalization in the PICU - serum urea (mmol/L) in hours.
Conclusions

We present a newborn infant, with normal prenatal and delivery history who suffered from extreme hypernatremia of up to 232 mmol/L and a rare complication of sinus vein thrombosis. He was diagnosed with BFAH due to his history of poor exclusive breast milk feeding and after ruling out alternative diagnoses. Despite the severity of his condition he gradually improved after undergoing judicious treatment according to established protocols. He was discharged from the hospital and on follow-up six months later displayed no neurological deficit.

This extreme case highlights several important points: (1) Pediatricians must be aware that BFAH is a common condition that requires appropriate parental guidance and close monitoring especially in high-risk cases; (2) Hypernatremia is a curable disorder when treated appropriately and expeditiously, and; (3) attention should be given to discrepancies in serum sodium ranges, especially concerning extreme values, when using ion-selective measuring instruments. The possibility of inaccurate measures reported by these instruments requires particular care and clinical evaluation on the part of the attending physician.

Conflict of Interest

All authors have indicated they have no potential conflicts of interest to disclose.

Funding

No external funding for this manuscript.

Financial Disclosure

All authors have indicated they have no financial relationships relevant to this article to disclose.

Contributors’ Statement

Dr. Braun and Dr. Zilkha conceptualized and designed the study and reviewed and revised the manuscript. Dr. Gross and Dr. Siedner-Weintraub collected data and drafted the initial manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

Dr. Gross wrote the first draft of the manuscript. No honorarium, grant, or other form of payment was given to anyone to produce the manuscript.

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