

Neonatal Hypernatremic Dehydration Secondary to Lactation Failure

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Dehydration in the neonatal period has many causes. Dehydration is classified as isotonic, hypotonic, or hypertonic, based on the serum osmolality that in most cases is reflected in the serum sodium level. Hypernatremic dehydration is usually caused by gastroenteritis, but it can also be caused by salt poisoning or diabetes insipidus. This type of dehydration can be especially dangerous. The hyperosmolar state can lead to brain shrinkage, venous thrombosis, and subdural capillary hemorrhage. In addition, rehydration can cause cerebral edema and subsequent seizures. Seizures can frequently occur, even with the best rehydration regimens. Breast-feeding can commonly cause mild dehydration, but it is usually quite well tolerated. No prospective studies have been done to investigate the incidence or prevalence of hypernatremic dehydration secondary to lactation failure. At least one author believes that the condition is more common now because of early infant hospital discharge without adequate follow-up.¹ We report a case report and literature review of hypernatremic dehydration caused by lactation failure.

Case Report

A 7-day-old female infant was the product of a normal spontaneous vaginal delivery at 36 weeks, 6 days' gestation. The pregnancy was complicated by preterm labor treated with bedrest and terbutaline from 18 weeks' gestation. Terbutaline was stopped 10 days before delivery. Her course in the nursery was normal, and she was discharged at 30 hours of life, breast-feeding, voiding, and passing stools nor-

mally. She was seen through the emergency department the evening of admission for complaints of poor feeding for the previous 12 hours. Her parents believed the infant was doing well until the day of admission, when the mother had noted decreased milk production and decreased activity in the infant. The infant appeared to have had adequate urine output throughout the day, and no fevers were noted. There had been no vomiting or diarrhea. Family history was notable for a sister who had had jaundice and difficulties with breast-feed early in life.

At admission the infant was jaundiced and dehydrated but responsive with good tone and a good suck reflex. Admission weight was 2.30 kg, 680 g (23%) below birth weight. Her sclera were icteric, the anterior fontanel was sunken, and her skin was jaundiced with poor turgor. Findings on heart and lung examinations were unremarkable. Her reflexes were brisk and symmetric. Findings during the remainder of the physical examination were unremarkable.

Laboratory studies disclosed the following values: white cell count, 4800/ μ L; hemoglobin, 17.9 g/dL; hematocrit, 52.5%; platelets, 433,000/ μ L; serum sodium, 167 mEq/L; potassium, 6.8 mEq/L; chloride, 132 mEq/L; bicarbonate, 16 mEq/L; blood urea nitrogen, 40 mg/dL; creatinine, 1.1 mg/dL; and serum glucose, 53 mg/dL. Total bilirubin was 29.6 mg/dL with a direct fraction of 0.9 mg/dL. A urinalysis disclosed a large amount of blood and proteinuria (2+); specific gravity was 1.025. A diagnosis of hypernatremic dehydration was made, and she was admitted for therapy.

There appeared to be no clear cause of the child's marked hypernatremic dehydration, so on the first hospital day, the mother's breast milk was tested. Her breast milk sodium content was 82 mEq/L. At 1-week post partum, her sodium level was approximately 10 times the expected level for mature breast milk. The volume of breast milk was not measured, but the mother believed it was adequate until the day of admission.

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Birth weight 2.98 kg
 Admission weight 2.30 kg
 Weight loss 680 g
 Percent dehydration = $680/2980 = 0.228 = 23\%$

Fluid deficit = weight loss = 680 mL

Fluid Therapy

Step 1: Emergency phase

Restore vascular volume with bolus of 10–20 mL/kg of lactated Ringer's solution

Patient was given 15 mL/kg, or 40 mL, of lactated Ringer's solution as a bolus for 20 min

Step 2: Rehydration phase

Aim to correct water deficit and sodium excess within 48 hours. The fluid should be administered evenly for 48 hours

$100 \text{ mL/kg/d} \times 2 \text{ d} \times 2.9 \text{ kg} = 580 \text{ mL maintenance}$

$\text{Maintenance} + \text{deficit} = 580 \text{ mL} + 680 \text{ mL} = 1,260 \text{ mL}$

$1,260 \text{ mL}/48 \text{ h} = 26 \text{ mL/h}$

Fluid composition should be 5% dextrose in 25% or 20% normal saline with 30–40 mEq/L of potassium. Choose lactate or acetate anions if serious hyperchloremia or metabolic acidosis exists. Monitor serum electrolytes every 8–12 hours. Expect a linear regression to a sodium concentration of 140 mEq/L to take place during 48 hours.

Figure 1. Fluid deficit calculation.

The child was treated with an initial fluid bolus of 15 mL/kg of lactated Ringer solution to restore intravascular volume, then free water deficit and sodium excess were corrected during the next 48 hours by calculating maintenance requirements and fluid deficits. She required fluid at a rate of 26 mL/h of one-third normal saline with 5% dextrose. Hypernatremic dehydration requires careful, slow rehydration to prevent cerebral edema and neurologic sequelae. Figure 1 illustrates the calculations used to correct the hypernatremia and dehydration. The goal is to return the sodium level to normal within a period of 48 hours. The jaundice responded to rehydration and phototherapy, and the infant was discharged from the hospital bottle-feeding well on the third hospital day.

Discussion

Mature human breast milk is normally low in sodium. This protects against the development of hypernatremia in breast-fed neonates.² Studies of the electrolyte composition of breast milk have shown a mean sodium value of 64.8 ± 4.4 mEq/L after delivery, dropping to a mean of 21.4 ± 2.3 mEq/L by the third postpartum day (colostrum), and leveling off to a value of 7 ± 2 mEq/L by 2 weeks (mature milk).³ Compared with cow's milk, mature human milk contains considerably less sodium, potassium, and chloride.⁴ Also, as the sodium concentration drops, the lactose level increases. Sodium chloride and lactose concentrations combine reciprocally to maintain the milk's osmolality at a level similar to that of blood. Any fall in lactose concentration could cause a rise in the sodium content of the milk.²

The drop in sodium concentration is not dilutional, as the major decline in sodium concentration occurs approximately 1 day before the major increase in milk volume. Sodium concentration does not vary from foremilk to hind milk, suggesting that there is no alteration during storage in ductal structures.⁵ Allen et al⁶ showed that the correlation between lactose levels and sodium, chloride, and potassium levels during pregnancy provides evidence that paracellular pathways between mammary alveolar cells are open during pregnancy and are at least partially closed during lactation. Failure of these paracellular pathways to close might be one mechanism involved in elevated breast milk sodium content and lactation failure, but this mechanism has not been studied.

High levels of sodium in breast milk are closely associated with lactation failure.⁷ One study showed that those who failed lactation had higher initial breast milk sodium concentrations, and the longer they stayed elevated, the lower the success rate. This association has subsequently been confirmed.⁸ Several possibilities have been suggested as to the cause of increased sodium levels in breast milk. Delayed maturation, perhaps caused by inadequate levels of lactose is one possibility.⁹ It has been shown that sodium values are not affected by the mother's diet by the method of milk expression.⁵ One study looked specifically at the effects of maternal sodium intake on postprandial sodium concentrations in breast milk and showed no meaningful impact.¹⁰

Reduction in feeding frequency is associated with a marked rise in milk sodium concentrations.¹¹ This association might be related to reduced production, which could in turn be secondary to neonatal factors, such as primary suckling deficiency or poor suckling as a result of infection, or to maternal factors, such as stress, mastitis, or sore or retracted nipples. A vicious circle can develop so that when breast milk production is reduced, the infant becomes weak and sucks poorly, and the drive for lactation drops further until dehydration occurs.^{8,12}

Breast milk sodium concentration is of particular physiologic importance in the feeding of neonates, and clinical problems arise if there is an excessive newborn intake of sodium. The kidney of the neonate has a limited capacity to concentrate solids, and the renal solute load, which is determined by sodium, potassium, chloride, phosphorous, and protein, exerts a major effect on water balance. This process can lead to a smaller margin of safety against dehydration, which is particularly a problem with diarrhea, fever, and low water intake.⁴ In hypernatremic dehydration, the extracellular fluid volume and plasma volumes are relatively preserved. The typical signs of dehydration, such as decreased skin turgor, tachycardia, and hypotension, are less pronounced than in hyponatremic or isonatremic dehydration. For these infants, the combination of inadequate fluid intake and hyperosmolarity is particularly devastating. Complications reported include convulsions, hyperglycemia, focal neurologic deficits, and disseminated intravascular coagulation.¹²

The infant with hypernatremic dehydration secondary to breast-feeding is typically encountered somewhere between the first and third weeks of life. The infants are often strikingly lethargic, malnourished, and dehydrated. Gestation, delivery, and initial neonatal course are usually uncomplicated. The mother is often primigravid, intelligent, and well motivated to breast-feed. The infant is often described as a sleepy or quiet baby who does not appear hungry, and he or she usually thrives with adequate nutritional support.^{2,4,9,12} A specific danger lies in the possible delayed recognition of this disorder, because most of the infants reported have nursed well and appear content. They therefore come to medical attention late, with severe dehydration, often weighing much less than 10% below birth weight.¹³

Prevention of hypernatremic dehydration secondary to lactation failure requires the physician to be alert to this possibility as well as adequate instruction to the parents regarding the signs and symptoms of feeding difficulties.³ Lactation counseling should begin at the hospital and continue in the physician's office for at least 2 weeks after discharge.¹³ Encouraging mothers to pump their breasts to facilitate maturation might help.⁹ Scheduling a weight check for the infant within the first week after discharge might also be prudent and could in the long run result in both cost savings and health care advantages.⁵

Conclusion

Hypernatremic dehydration in the breast-fed neonate should suggest an abnormal concentration of sodium in the mother's breast milk, a sign of lactation failure. Rehydration of the neonate must be accomplished first by fluid resuscitation, then slow steady rehydration for a period of 48 hours. Women should be educated about the signs and symptoms of dehydration during prenatal visits and again before discharge after delivery. Early discharge demands early follow-up to detect breast-feeding problems before the onset of serious and sometimes life-threatening dehydration.

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