CASE REPORTS

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Hypernatremia in Breast-Fed Newborns

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BREAST-FEEDING OFFERS several advantages to newborn infants.¹ Estimation of intake of breast milk in these infants, however, is difficult. This report describes an infant who had hypernatremia associated with inadequate breast-feeding and failure to thrive; four other cases with similar clinical courses have been reported.²⁻⁴ To facilitate prevention and early recognition of this condition, the common features of these cases are reviewed.

Report of a Case

The infant, a 3,240-gram girl, was delivered at home by a midwife. It was the mother's first pregnancy. The infant was exclusively breast-fed every two to four hours and frequently fell asleep after five minutes of feeding. She cried very little when hungry and at 2 weeks of age had lost 855 grams (weight 2,385 grams). At 20 days she was first seen by a physician and admitted to hospital,

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weighing 2,200 grams. There was no history of vomiting, diarrhea or intake of other foods or medicines. At physical examination, the infant was emaciated but alert, sucked well and had signs of moderate dehydration (decreased skin turgor, depressed anterior fontanelle, sunken eyes. absence of tears and dry mucous membranes). Her length was 49.5 cm, occipital-frontal circumference 33.6 cm, pulse 108, blood pressure 90/50 mm of mercury, respiratory rate 24. Laboratory data included the following: serum sodium concentration 182, potassium 4.0, chloride 146 and bicarbonate 20 mEq per liter; blood urea nitrogen 94, serum creatinine 1.3 and serum glucose 108 mg per dl; hematocrit 69 percent. Urine had a specific gravity of 1.032, an osmolarity of 843 mOsm per liter and 2+ protein. Analysis of the mother's milk showed a sodium concentration of 39 mEq per liter on admission and 12 mEq per liter on discharge. There was no evidence of mastitis. The infant's hyperosmolarity was slowly corrected. When nursed she obtain only 20 to 30 ml of breast milk; thus, she was discharged on a schedule of breast-feeding with formula supplements averaging 145 ml per kg of body weight per day. Four days after discharge, her weight had increased 130 grams and the serum sodium concentration was 138 mEg per liter. A neurological examination showed no abnormalities.

Discussion

Hypernatremia in infancy can arise in a variety of clinical settings including central or nephrogenic diabetes insipidus, gastroenteritis, respiratory infections, salt intoxication and diminished fluid intake. The pathophysiological mechanisms are

TABLE 1.—Features of	Five Cases of Hypernatremic	Dehydration Associated With				
Inadequate Breast-Feeding						

	.Clarke²	Anand ³	Anand³	Arboit4	Jaffe
Age at presentation (days)	14	15	18	15	20
Supplemental feedings	None	None	None	None	None
Personality	"Sleepy"	"Sleepy"	NR	NR	"Sleepy"
Failure to thrive	+	+ 11	+	+	+
Parity	1	1	NR	NR	1
Diminished milk production	NR	+	+	NR	+
	(fed only				
	$3 \times /day$				
Breast milk sodium concentration (mEq/1)	NR	31	74	104	39
Serum sodium concentration (mEq/1)	178	192	202	180	182

excessive free water loss, excessive sodium intake or diminished free water intake. The last mechanism seems the explanation in this and the four other reported cases.

All five infants shared common features: they were emaciated, less than 1 month of age and exclusively breast-fed-that is, with no water or formula supplementation (Table 1). Three of the infants were "sleepy," difficult to keep awake for feeding and content to starve uncomplainingly, a problem to which attention has recently been drawn.⁵ One mother was noted to be upset by her difficulty in arousing her baby, but was reassured by family members that this was normal.² In the three cases where maternal parity was described, all mothers were primiparous. This suggests a lack of familiarity with normal newborn eating and sleeping patterns, and may explain why these mothers delayed seeking medical attention.

Although three of the mothers appeared to have diminished milk production, an expected finding in the face of diminished demand, only one was reported as perceiving her milk production to be inadequate.³ Each had an unexplained increase in milk sodium concentration in comparison to a mean of 7±2 mEq per liter reported by Macy⁶ for women 15 days postpartum. In fact, these values were even high for women during the first five days postpartum when the mean sodium concentration is significantly higher (21 ± 5 mEq per liter).7 This last association is an intriguing but unlikely explanation for the hypernatremia. The infants' diminished intake, suggested by the emaciation and degree of dehydration, makes it improbable that they ingested a high amount of sodium. The cause of the increased milk sodium concentration remains uncertain.

Without any evidence of excessive free water loss, the explanation for the hypernatremia in these five infants is diminished free water intake. The combination of large insensible water losses diminished renal concentrating capacity makes newborn infants susceptible to water depletion. Even nursing infants who have low obligatory urinary water loss (because of breast milk's low solute content) can become water depleted when intake of breast milk decreases to the extent that the renal concentrating capacity is exceeded.

Comment

The five cases of hypernatremia in breast-fed newborns emphasize the importance of frequent follow-up of newborn infants. Careful attention must be paid to the adequacy of their weight gain. Although most babies cry when they are hungry, health professionals who care for newborns must be aware that otherwise normal babies may be content to nurse infrequently, and thereby fail to thrive and, in this context, become hypernatremic. With adequate instruction of mothers and proper care of their breast-fed infants, this potentially damaging condition⁸ can be prevented.

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Refer to: Smith CI, Juttner HU, Redeker AG: Bile duct stricture shown during transhepatic cholangiography by cholecystokinin. West J Med 135:55-57, Jul 1981

Bile Duct Stricture Shown **During Transhepatic** Cholangiography by Use of Cholecystokinin

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THE HORMONE cholecystokinin (CCK) has been used to show gallbladder contraction during cholecystography (following oral ingestion of contrast material). We report the case of a patient with a stricture of the common bile duct in whom CCK was used during transhepatic cholangiography

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