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Case Report

Hypernatremic dehydration in newborns

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ABSTRACT

Hypernatremic dehydration is an uncommon problem in newborns. Infants who fail to breast-feed may develop hypernatremic dehydration, a condition that can be fatal or cause major morbidity. Early discharge, lack of timely follow-up, or poor information during pregnancy or after delivery play important roles. Hypernatremic dehydration can lead to neonatal morbidity and mortality. We are reporting two cases of exclusively breastfed neonates with hypernatremic dehydration.

Keywords: Hypernatremia, Dehydration, Weight loss, Breastfeeding

INTRODUCTION

Hypernatremic Dehydration an uncommon problem in newborns due to inadequate breastfeeding which can be managed by rehydration.

CASE REPORT

1) A term male neonate born to a 25-year-old primigravida delivered by vaginal delivery with birth weight of 3.0 kg and normal APGAR scores. The baby was brought on day 12 of life with history of poor feeding and lethargy for 5 days. There was no history of diarrhea. Urine output was normal and the baby was on exclusive breast feeds. The mother felt that the baby was having decreased feeding well prior to the illness. Baby had fever on day 3 for which he was shown to doctor and was given paracetamol and antibiotic drops.

On examination baby was sick and lethargic and had signs of moderate dehydration. His admission weight was 2522 g indicating a weight loss of 16% from the time of birth. RR was 30/min, HR was 110/min and pulses were well felt, Temperature was 35.5ºC, BP = 74/47 (54 mmHg). On Systemic examination baby was lethargic, cry was absent, tone and activity was decreased. Moro's was absent and sucking was intermittent, rest systems were within normal limits. A provisional diagnosis of dehydration was made and the child was admitted to the NICU.

Investigation revealed hypernatremia and azotemia (Table 1). The initial serum sodium was 167 mEq/L and potassium was 5.1 mEq/L. The blood urea and creatinine at admission were 186 mg/dl and 3.1 mg/dl, respectively. Serum calcium was 7.3. Blood sugar at the time of admission was 97 mg/dl. ABG at the time of admission showed metabolic acidosis: pH - 7.339, PCO_2 - 47.3, PO_2 - 70, HCO^{3-} - 24.8 and BE - 1.4. Initial sepsis screen was negative: Hb - 18.5 g/dl, TLC - 11500/cumm, P54 L43 and platelet count 3.13 lakhs/cumm. Blood and urine cultures were negative. On D1 of admission baby had convulsion and phenobarbitone was started. The baby was managed with IV fluids to correct dehydration and hypernatremia. Calcium gluconate was also given to treat hypocalcemia. The baby required 195 ml/kg/day (1.3

times maintenance) of fluids from day 1 to day 4 to correct hydration status. Initially dextrose 1/2NS was used for four days followed by isolyte P maintenance and Palladai feeds thereafter. The serum sodium levels and renal parameters returned to normal by day 5 of admission (Table 1). The baby was on complete oral feeds by day 6 of admission.

Table 1: Case 1) Investigation revealed hypernatremia and azotemia: parameters with day of admission.

Day of admission	D1	D2	D3	D4	D5
Weight (gm)	2522	2715	2840	3005	2930
Fluid input (ml/kg)	195	195	195	195	195
Urine output (ml/kg/h)	1.6	2.1	1.8	4.8	3.1
Na+ (mEq/L)	167	162	151	146	141
K+ (mEq/L)	5.1	4.1	3.8	3.3	3-5
Blood urea (mg/dl)	186	133	103	79	52
Serum creatinine (mg/dl)	3.1	1.7	1.7	1.5	1

In view of the high blood urea and creatinine a abdominopelvic scan was done to rule out renal abnormality which showed increased echogenicity at the tip of medullary pyramids most likely due to benign cause, probably dehydration. NCCT brain was done which showed mild confluent areas of white matter hypodensities in bilateral parietal lobes most likely post ictal ischaemic changes. The baby was discharged on day 12 of admission on breast feeds and infant formula. The discharge weight was 2950 g. The final diagnosis was hypernatremic dehydration with acute renal failure. The cause of hypernatremia was thought to be due to inadequate breast feeding. An attempt was made to determine the sodium concentration of the breast milk but this was not possible. On follow up there was no polyuria or dehydration. Electrolytes and renal parameters remained normal. Phenobarbitone was stopped at 3 mts of age after neurological assessment was normal. Growth and development of the baby are within normal limits.

2) A term female neonate born to a 20 year-old primigravida by emergency LSCS in view of thick meconium with birth weight was 2.6 kg and normal APGAR scores at 1 and 5minutes. The baby brought on day 23 of life with history of poor feeding and lethargy of 5 days duration. There was no history of diarrhea. Urine output was normal and the baby was on exclusive breast feeds. The mother felt that the baby was having decreased feeding well prior to the illness and Baby was shown to local doctor in view of not gaining weight on D20 of life and Ayurvedic medications were given. As baby was not improving she was brought to our hospital.

On examination baby was sick and having emaciated look with signs of moderate dehydration. His present weight was 1.545 g indicating a weight loss of 40% from the time of birth. RR was 68/min. HR was 104/min and pulses were feeble, Temperature was 35ºC, BP = not recordable and CRT >2 sec. On Systemic examination baby was lethargic, cry was absent, tone and activity was decreased. Moro's was incomplete and sucking was weak, rest systems were within normal limits. A provisional diagnosis of dehydration was made and the child was admitted to the NICU.

Investigation revealed hypernatremia and azotemia. Sepsis screen was positive. The initial serum sodium was 198 mEq/L and potassium was 5.8 mEq/L. The blood urea and creatinine at admission were 323 mg/dl and 3.8 mg/dl, respectively. Serum calcium was 10.7. Blood sugar at the time of admission was 97 mg/dl. Initial sepsis screen was Positive: Hb - 21.6 g/dl, TLC - 4500/cumm, P40 L50 E4 M6 and platelet count 24000/ cu mm. Blood culture showed Klebsiella. The baby was managed with IV antibiotics and fluids to correct dehydration and hypernatremia. The baby required 195 ml/kg/day (1.3 times maintenance) of fluids from day 1 to day 5 to correct hydration status. Initially dextrose 1/2NS was used for four days followed by isolyte P maintenance and Palladai feeds thereafter. The serum sodium levels and renal parameters returned to normal by day 5 of admission (Table 2). The baby was on complete oral feeds by day 7 of admission.

Table 2: Case 2) Investigation revealed hypernatremia and azotemia: parameters with day of admission.

Day of admission	D1	D2	D3	D4	D5	D6
Weight (gm)	1545	1646	1896	2019	2101	2079
Fluid input (ml/kg)	195	195	195	195	195	195
Urine output (ml/kg/h)	1.6	1.2	2.3	2.7	3.5	2.45
Na+ (mEq/L)	198	164	156	150	141	136
K+ (mEq/L)	5.8	5.3	5.1	5.1	4.7	4.7
Blood urea (mg/dl)	323	220	153	100	68	37
Serum creatinine (mg/dl)	3.8	2.0	1.6	0.7	0.8	0.7

In view of the high blood urea and creatinine an abdomino-pelvic scan was done which was normal. The baby was discharged on day 12 of admission on breast feeds and infant formula. The discharge weight was 2102 g. The final diagnosis of hypernatremic dehydration with acute renal failure with sepsis was made. The cause of hypernatremia was thought to be due to inadequate breast feeding. Antibiotics were given for 21days. An attempt was made to determine the sodium concentration of the breast milk but this was not possible. On follow up electrolytes and renal parameters remained normal. Growth and development of the baby are within normal limits.

DISCUSSION

Hypernatremic dehydration is not a common problem in the neonates. Early postpartum hospital discharge may leave mothers poorly prepared for breast-feeding and may contribute to increased neonatal morbidity.[1] Some breastfeeding mothers perceive their milk supply is inadequate or recognize their baby is failing to thrive and rapidly resort to bottle feeding.[2] In contrast, other mothers are aware of the unique characteristics of breast milk and persist with exclusive breastfeeding, reluctant to give additional formula despite excessive neonatal weight loss. These babies are at risk of developing malnutrition and hypernatremic dehydration, with the potential for seizures and permanent neurological and vascular damage if their condition is not recognized and treated early.[3]

Hypernatraemic dehydration is notoriously difficult to diagnose on clinical examination alone, as skin turgor is preserved; the anterior fontanelle can retain its normal fullness, and urine output, although reduced, is maintained even in the face of severe dehydration. The clinical features are a spectrum, from an alert and hungry child who appears relatively well to a child who is lethargic, irritable and even moribund.[4,5]

CONCLUSION

Neonates with hypernatremic dehydration can be managed with fluid therapy alone. Rehydration of the neonate must be accomplished first by fluid resuscitation then slow steady rehydration with fluids more than maintenance. Women should be educated about the signs and symptoms of dehydration during prenatal visits and again before discharge after delivery. Those with feeding difficulties should be identified and given supportive care and lactation counselling. Neonates discharged early should be followed up to detect breastfeeding problems before the onset of serious and sometimes life-threatening dehydration.

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