Research Paper

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Severe hypernatremia and hyperglycemia in a breast-fed neonate.

Medical Science

KEYWORDS : Hypernatremia, hyperglycemia, neonate, breast fed, neurological outcome

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ABSTRACT

Hypernatremia and hyperglycemia are potentially devastating conditions which warrants adequate and immediate treatment. We report a case of severe hypernatremia with severe hyperglycemia in a neonate who had a normal neurodevelopmental outcome at one year. Severity of hypernatremia or hyperglycemia cannot predict the long term outcome. Appropriate and prompt treatment, with a gradual fall in serum sodium could lead to a good neurological outcome.

Introduction

Hypernatremia is a life threatening condition which needs adequate and immediate treatment. Lack of appropriate treatment may lead to severe neurological deficits and brain damage, including cerebral edema and intracranial hemorrhage in the neonatal period. [1] Incidence of hypernatremic dehydration secondary to breast feeding is as high as 7.1 per 10,000 breast fed newborns. [2]

Hyperglycemia in neonates is defined as serum glucose level greater than 125 mg/dl after a 4 hours fast. [3] Sepsis and stress are known to be associated with hyperglycemia. Neonatal diabetes mellitus, an uncommon cause of hyperglycemia in the newborn period, presents within the first four weeks of life and persists for more than two weeks. [4]

There have been very few reports of severe hypernatremia with hyperglycemia together in neonates with normal neurodevelopment outcome. We report one such case.

Case Report: A 15 day old neonate was admitted to NICU with decreased acceptance of breast feed, lethargy for 3 days and fever with reduced urine output for one day. The baby was born to a 34 year old, healthy mother by normal vaginal delivery at term gestation. The birth weight was 3.5 kg and APGAR score was normal. The neonate was exclusively breast fed since birth.

Prior to admission in NICU, the neonate was admitted to a nursing home with aforementioned complaints and treated with IV fluids (Isolyte P) for 4 hrs. Investigations done there revealed Hb – 18.3 g/dl, TLC – 7600/ Cu mm, P – 40%, L – 58% and platelet count 1 Lac/Cu mm. Serum Na - 215 meq/dl, K – 5.6 meq/dl, random blood sugar (RBS) was 599 mg/dl and C-reactive protein was positive (1:8).

The neonate was then transferred to our institute. On admission he had temperature 39.5ºC, heart rate - 166/min and respiratory rate – 64/min. Examination revealed a sick baby who was lethargic, had signs of severe dehydration and sclerema. His weight at admission was 2.43 kg, indicating a weight loss of 30% since birth. Systemic examination was otherwise normal. A provisional diagnosis of sepsis with hypernatremia and severe dehydration was made and the baby was given an initial normal saline bolus of 20 ml/kg.

Investigations on admission showed normal haemogram except a platelet count of 1 lac/cu mm, S. Na – 180 meq/dl, S. K – 6.5meq/dl, S. urea – 432mg/dl, Creatinine – 4.1mg/dl, RBS – 952 mg/dl. ABG at the time of admission showed metabolic acidosis: pH – 7.2, HCO_3- 14.5. Urine routine showed pH- 7, sugar – 3+, pus cells – 7-8/hpf, ketone- absent. Calculated serum osmolality was 529mosm/kg.

After normal saline bolus, the neonate was given fluids at 120ml/kg/day. Initially, half normal saline was used followed by 1/5th normal saline. The neonate was started on injection Cefepime. After 4 hours of admission the neurological condition of the baby deteriorated and he had a convulsion. The neonate was intubated, put on ventilator and was treated with phenobarbitone. Antibiotic was changed to meropenam. In view of high blood sugar, insulin drip was started. Serum sodium decreased to 176 meq/dl in next 6 hours, to 170 meq/dl over 20 hours and to162 meq/dl after 48 hours. At 48hours RBS was 551 mg/dl and ABG improved.

Blood sugar gradually decreased, so insulin drip was tapered and stopped on day three of admission. Serum sodium (131meq/dl) and serum creatinine (0.4mg/dl) normalized by day five of admission. The neonate was extubated on day eight of admission. The baby was discharged on breast feeds and discharge weight was 2.5kg. Ultrasonography of the brain and EEG done at discharge were normal.

On subsequent visits serum electrolytes and blood sugar were normal. At one year of age the child is walking with support. His developmental quotient for motor skills is 80 and for mental skills is 95 on Developmental Assessment Scale for Indian Infants (DASII).

Discussion: Hypernatremic dehydration carries significant morbidity and mortality. Hypernatremia was previously thought to be unusual in breast-fed babies. From 1979 to 1989 there were sporadic reports of hypernatremic dehydration occurring in breast fed babies. [5] Since 1990s, there has been an increase in incidence of hypernatremia in breast - fed babies. [6] Hypernatremic dehydration due to breast milk feeding usually presents between the first and third weeks of life. The clinical presentation may be non- specific, including lethargy and dehydration as in our patient. An important association of hypernatremic dehydration in breast fed babies is inadequate breast feeding technique in inexperienced mothers. [7]

Cornerstone of treatment is slow rehydration and gradual correction of hypernatremia. In 1975 Banister *et al* reported 38 infants with severe hyperosmolar dehydration and hypernatremia. Infants rehydrated at a rate of 150ml/kg/day were more likely to develop convulsions and peripheral edema than the infants whose fluid intake was restricted to 100ml/kg/day. [8] Our case was initially resuscitated with 120ml/kg and then restricted to 100ml/kg/day.

Hypernatremia causes a state of hyperosmolarity which leads to a modified stress reaction. [9] Our child had significant hyperglycemia which was possibly due to hypernatremia and septicemia both of which are known to give rise to hyperglycemic states. Septic babies and sick neonates may have hyperglycemia due to altered renal threshold with decreased utilization of glucose in the presence of increased circulating insulin. [10] It may be due to increased levels of steroid hormones and catecholamines.

This was not a case of transient neonatal diabetes mellitus because by definition in this condition hyperglycemia lasts at least for two weeks, but in our case hyperglycemia lasted only for two days.

Esad Koklu reviewed 116 neonates with hypernatremia and on long term follow up found that more than half of infants had abnormal development at one year of age. [11] Long term outcome of neonatal hyperglycemia at two years of age, in very preterm infants showed higher incidence of abnormal neurological and behavioral development in these babies. [12] Our patient had normal mental and motor development at 12months.

Therefore, one can infer that long term neurological outcome cannot be predicted from the severity of hypernatremia or hyperglycemia. Appropriate and prompt treatment, with a gradual fall in serum sodium can lead to a good neurological outcome.

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