Stroke in infant: A rare presentation of severe hypernatremic dehydration

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Abstract

Hypernatremic dehydration in infancy is a medical emergency, which constitutes a potential threat to life. Herein, we report an 8-month-old baby with severe hypovolemic hypernatremic dehydration and seizures secondary to acute gastroenteritis, and a history of formula milk with a bottle and improperly diluted oral rehydration solution. The infant was ventilated in view of low GCS and successfully rehydrated with slow sodium correction. Child also developed intracranial complications i.e. cerebral sinusvenous thrombosis which was treated and discharged with anticoagulation therapy.

Introduction:

Cerebral sinovenous thrombosis (CSVT) is rare in the pediatric age group accounting for (0.67/1,00,000 children) cases[1]. Hypernatremia (Na+ >145 milliequivalent / liter) is more common in bottle-fed babies particularly in those erroneously using concentrated formula feeds and undiluted cow milk. The vast majority of cases of hypernatremic dehydration (90%) are reported in children less than two years of age with the worst outcome in infants less than 6 months of age. The case fatality rate in this age group is as high as 60% with conventional treatment[2]. Here in, we report a case of an 8-month-old infant who presented with seizures and stroke secondary to severe hypernatremic dehydration.

Case Report :

An 8-month-old female child was brought with complaints of vomiting, loose stools since 8 days, and fever for 2 days, with a history of formula feeding with a bottle. At admission, the child had altered sensorium, Jitteriness, poor feeding, and seizures.

On examination, revealed decerebrate posturing, pulsatile anterior fontanelle, sunken eyes, absence of tears with parched mucous membrane, poor skin turgor, low volume pulses, a heart rate of 150/ beats per minute, respiratory rate of 52 /cycles per minute, and blood pressure of 70/46 mmHg. Central nervous system examination revealed a poor Glasgow coma scale (GCS) of 8/15, left abducent nerve palsy, generalized hypertonia with exaggerated deep tendon reflexes, right-sided hemiparesis with Kernig's sign, and Brudzinski's sign positive.

Blood investigations (Table1) showed hypernatremia with acute kidney injury, severe metabolic acidosis (pH 7.05, PCO2 24.4, HCO3 6.83), and severe anemia. Cerebrospinal fluid analysis was normal.

An MRI of the brain (figure 1) showed acute infarct with a hemorrhagic transformation involving bilateral frontoparietal and left occipital lobes and abnormal leptomeningeal enhancement present in the left parietal and frontal lobe, collective findings likely suggestive of hemorrhagic meningoencephalitis and evidence of a filling defect in the posterior aspect of the superior sagittal sinus, suggestive of cerebral sinus venous thrombosis.

Table 1. Investigations

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
Hemoglobin	4.8			5.8		8.3		8.5
White blood cells	18,190			13,000		12,320		15,500
Platelet count	2,33,000			66,000		67,000		2,63,000
Sodium	167	172	173	179	173	162	154	144
Potasssium	3.8	3.2	3.6	3.2	3.3	3.8	3.8	4.4
Chloride	145	134	141	137	146	129	121	121
Creatinine	1.5	1.3	1.2	0.9	0.7	0.7		0.5

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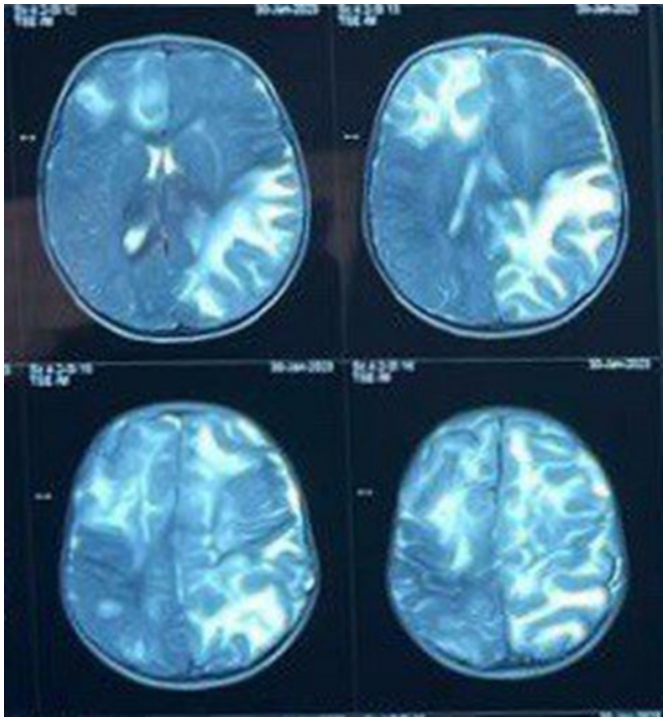

Figure 1. Ill-defined T2 hyperintensities in the left frontoparietal region and right frontal region, effacement of the occipital horn of the left ventricle.

The child was started with Plan C dehydration management i.e. intravenous fluid normal saline (30 mL/kg over 1 hour), followed by ringer lactate (70 mL/kg over 5 hours). The child was assessed again and reclassified as having some dehydration, and a Plan B correction of 500 ml of ORS was given for 4 hours through the nasogastric tube. Hypernatremia dehydration was managed with intravenous fluids of 5% glucose and half normal saline for 72 hours, later free water correction was given,with serial sodium level monitoring(figure 2).

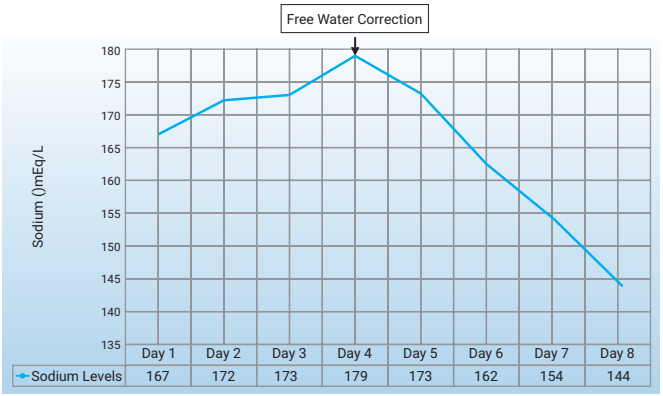

Figure 2: trends of serum sodium levels

Raised intracranial pressure was managed with mannitol, elective intubation, and mechanical ventilation. As the child had anemia, it was corrected with 2 settings of packed red cells (10 ml/kg). The child was started on subcutaneous low molecular weight heparin (LMWH) at 1 mg/kg every 12 hours in view of CSVT. The child showed improvement and was discharged with LMWH. Later, the child lost follow-up.

Discussion:

Hypernatremia is a state of relative water deficiency and excessive solute concentration in body fluids. It is said to be present when plasma sodium level is more than 145mEq/l. Based on the amount of deficit in total body fluids, hypernatremia is described as either hypovolemic, euvolemic, or hypervolemic. Hypovolemic hypernatremia is a common problem in pediatric practice. The majority of the affected children are below the age of 2 years and the worst outcome is observed in infancy[3]. Symptomatic hypernatremia is seen when serum sodium levels are >160 mEq/L. Hypernatremic dehydration in infants is usually due to inadequate breastfeeding, inappropriately prepared formula milk, fever, and acute gastroenteritis where the water loss is far greater than the salt loss[4].

There may be fever, tachycardia with poor perfusion, and hypotension with hypovolemia. The skin will be thick, doughy with dry mucous membrane, and depressed anterior fontanel with sunken eyes. An important observation is intense thirst and craving for water. Plasma hypertonicity and the subsequent intracellular water loss causes the brain cells to shrink, leading to the rupture of bridging vessels with hemorrhages (subarachnoid and parenchymal) and thrombosis[5]. The brain responds, by acquiring new intracellular solutes known as “idiogenic osmoles” to protect the intracranial volume[6]. During rapid rehydration with relatively hypotonic intravenous fluids, excess water enters the cerebral cells leading to rebound cerebral edema, permanent cognitive impairment, cerebral dysfunction, spastic paralysis, and seizure disorders have been described, Extensive lateral and central pontine and extrapontine myelinolysis have also been reported[7].

Hypernatremic dehydration being a hypercoagulable state predisposes to thrombosis of both cerebral and systemic circulation. Some studies reported CSVT in infants secondary to hypernatremic dehydration in the literature[8-13].

In our case, improper preparation of formula feeding with a bottle and gastroenteritis has led to hypernatremic dehydration and CSVT. Clinical features of CSVT in infants include jitteriness, seizures, lethargy, poor feeding, and focal neurological deficits. An MRI brain with MR venography is considered the gold standard for diagnosis.

Anticoagulation is the recommended treatment with LMWH for 6 weeks to 3 months[14,15]. Supportive

care in the form of adequate hydration, anti-seizure medications, and antibiotics for infection is also necessary. Associated venous infarction and refractory seizures can worsen the outcome, with long-term neurological sequelae. Long-term neurological outcome is unclear in infantile CSVT and up to 20-30% of infants can have abnormal neurodevelopment.

Conclusion:

Hypernatremic dehydration in infancy is a medical emergency with high rates of morbidity and mortality. Early diagnosis and prompt and appropriate treatment are crucial for survival and prognosis. However, diagnosis is often difficult and dehydration is underestimated. One should suspect intracranial complications of hypernatremia or its over treatment, including CSVT, when there is no improvement in neurological status even after the correction of hypernatremia.

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Conflict of interest: None
Source of funding: None

Date received: May 29, 2023
Date accepted: Nov 12, 2023