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In Brief

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Author Disclosure

Drs Goff, Higinio, and Serwint have disclosed no financial relationships relevant to this article. This commentary does not contain a discussion of an unapproved/investigative use of a commercial product/device.

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Hypernatremia is defined as a serum sodium concentration of greater than 145 mEq/L (145 mmol/L). This state constitutes an important electrolyte abnormality that requires rapid clinical assessment and intervention to prevent deterioration and complications. Serum sodium values greater than 160 mEq/L (160 mmol/L) require immediate attention.

Hypernatremia most commonly indicates a deficiency of total body water relative to total body solute. This imbalance can be caused by three basic mechanisms: 1) water losses, including gastrointestinal, insensible, or renal; 2) inadequate water intake; and 3) excessive sodium intake, either oral or parenteral, or mineralocorticoid excess.

Hypernatremia historically has been associated with dehydration caused by diarrhea, usually found in children younger than 12 months of age. Several studies have shown that ineffective breastfeeding, particularly by first-time mothers, can lead to hypernatremic dehydration. A recent study suggests that in developed countries, where severe infectious diarrhea is much less common, hypernatremia has become primarily a hospital-acquired disease. In these cases, infusions of hypertonic fluids or hypertonic enteral feedings in conjunction with the patient's inability to take adequate amounts of water are common causes of hypernatremia. Inadequate free water intake due to a depressed sensorium may occur postoperatively in patients taking medications that cause central nervous system (CNS) depression.

Children who have CNS disorders, particularly severe static encephalopathies, are at particular risk for developing sodium homeostasis problems, although cerebral salt wasting with hyponatremia is more common than hypernatremia. If a neurologically impaired child has a hyperthermic response to stress or infection as well as impaired intake of fluids, the risk of hypernatremia is increased. When these circumstances do not exist, the clinician should consider disruption of antidiuretic hormone secretion causing excessive free water losses from diabetes insipidus or mineralocorticoid excess.

Patients who have hypernatremia can present with irritability and agitation and can progress to lethargy, depressed sensorium, and coma. Clinically, the patient who has hypernatremia and dehydration does not look as hypovolemic as in other states of dehydration because the shift of fluids from the intracellular to the extracellular space protects intravascular volume status longer. Physical examination findings may show increased tone in the extremities with brisk reflexes, "doughy" skin turgor, decreased capillary refill, nuchal rigidity, myoclonus, asterixis, chorea, and seizures.

Treatment involves: 1) determining the underlying disorder, 2) assessing and correcting any vascular volume deficit, 3) replacing the free water deficit, and 4) providing maintenance fluids to match continuing ongoing losses, both sensible and insensible. In practice, these issues are dealt with concomitantly. Adequate intravenous fluid boluses should be administered to stabilize the circulation with normal saline or another crystalloid (without glucose). Subsequently, the remaining volume deficit is calculated. Fifty percent of the remaining volume deficits can be replaced in the first 8 hours and the remainder over the next 16 hours.

Next, the free water deficits must be calculated with the following formula:

Free water deficit (mL) = 4 mL
× actual body weight (kg)
× [desired change in serum sodium mEq/L (mmol/L)]

Total volume deficit (along with ongoing fluid maintenance needs) and free water deficit must be calculated to determine the correct infusion rate and tonicity of fluids needed. For example, if you calculate a 200-mL free water deficit, infusing 400 mL of 5% dextrose in1/2 normal saline will provide 200 mL of free water.

The serum sodium concentration reduction should not exceed more than 1 mEq/L per hour and 15 mEq/L per 24 hours to prevent fluid shifts leading to cerebral edema. Because it takes 48 to 72 hours for idiogenic osmoles in the brain to adapt to changes in sodium concentration, the free water deficit should be replaced gradually, making sure that the reduction in serum sodium concentration does not exceed the previously cited guidelines. Accordingly, reaching the desired end-point might take longer than 48 hours, depending on clinical severity. Monitoring of serial sodium concentrations at 1- to 4-hour intervals may be necessary in the initial phases of sodium correction to ensure that serum sodium values fall slowly and gradually. Strict monitoring of intake and output is important to verify an adequate urine output of 1 mL/kg per hour and to be vigilant for the potential complication of acute tubular necrosis. The clinician should include volumes of fluid lost in watery diarrhea, vomiting, or nasogastric fluid when calculating fluid replacement.

Hypernatremia alone can cause significant complications. The acute stage of dehydration can be associated with mental status changes, seizures, or stroke. The acute intracellular dehydration causes volume loss in the brain, so the bridging veins of the brain can be sheared, leading to hemorrhage or venous thrombosis. Rapid lowering of serum sodium with rapid volume resuscitation can lead to acute brain swelling and central pontine myelinolysis. It is common for seizures to occur in this setting. Mortality rates in children being treated for hypernatremia can be as high as 15%. However, mortality is associated more commonly with uncorrected hypernatremia than with rapid correction.

In summary, hypernatremia usually is associated with dehydration in three groups of children: 1) infants; 2) children in hospital settings; and 3) neurologically compromised children who have major disabilities, including disorders of impaired thermoregulation and sodium homeostasis. A careful approach to fluid rehydration concomitant with lowering the serum sodium no more than 1 mEq/L per hour (or 15 mEq/L in 24 hours) is advised. Calculations of free water deficit and calculations of volume deficit are very important in correct management of hypernatremia. The clinician should be familiar with the complications that may occur during the acute phase of this illness as well as during the treatment.

Comment: The body's mechanism for maintaining electrolyte homeostasis is truly amazing. The ability of humans to concentrate their urine to retain fluids and the function of their thirst mechanism to increase fluid intake make hypernatremia unlikely in neurologically normal patients who have access to water and are able to take fluids by mouth. However, the disruption of these abilities or restricted water access makes certain patients vulnerable. Another high-risk group, as mentioned by Drs Goff and Higinio, are breastfed infants in the first few postnatal weeks. Hypernatremia develops due to a combination of the high human milk sodium concentration in the first few days of lactation and insufficient fluid intake, leading to dehydration. In a retrospective study of otherwise healthy neonates who were breastfed and admitted with serum sodium concentrations of 150 mEq/L (150 mmol/L) or greater, researchers found that the majority were infants born to primiparous women and presenting with jaundice. Their mean age at admission was 5 days (range, 2 to 16 days) and mean weight loss was 13.7%. Almost 75% of these babies had experienced greater than 10% weight loss compared with birthweight. This study serves as a reminder to pediatricians to consider hypernatremia in breastfed infants who exhibit significant weight loss and jaundice at their follow-up visits.

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