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Hypernatremia

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Introduction

Background

Sodium levels are tightly controlled in a healthy individual by regulation of urine concentration and production and regulation of the thirst response. In patients with an intact thirst response, hypernatremia (defined as a serum sodium level >145 mEq/L) is a rare entity. When hypernatremia does occur, it is associated with a high mortality rate (>50% in most studies).

Given this high mortality rate, the emergency physician must be able to recognize and treat this condition. Accordingly, this article discusses the patients in whom hypernatremia should be suspected and a treatment strategy for patients in whom the condition is discovered.

In general, hypernatremia can be caused by derangement of the thirst response or the behavioral response thereto (primarily in infants, psychiatric patients, and elderly patients who are institutionalized), by problems with the renal concentrating mechanism (diabetes insipidus [DI]) secondary to kidney pathology (nephrogenic DI) or difficulty with the neurohormonal control of this concentrating mechanism (central DI), or by losses of free water from other sources.

Assessment and treatment of a hypernatremic patient focuses on 2 important questions:

- What is the patient's volume status?
- Is the problem acute or chronic?

Pathophysiology

Water homeostasis results from the balance between water intake and the combined water loss from renal excretion, respiratory, skin, and GI sources. Under normal conditions, water intake and losses are matched. To maintain salt homeostasis, the kidneys similarly adjust urine concentration to match salt intake and loss.

Hypernatremia results from disequilibrium of one or both of these balances. Most commonly, the disorder is caused by a relative free water loss, although it can be caused by salt loading. The various ways in which these equilibria can be disturbed are discussed in Causes.

When hypernatremia (of any etiology) occurs, cells become dehydrated. Either the osmotic load of the increased sodium acts to extract water from the cells or a portion of the burden of the body's free water deficit is borne by the cell. (Sodium, primarily an extracellular ion, is actively pumped out of most cells and is the primary determinant of serum osmolarity.) Dehydrated cells shrink from water extraction.

Cells immediately respond to combat this shrinkage and osmotic force by transporting electrolytes across the cell membrane, thus altering rest potentials of electrically active membranes. After an hour of hypernatremia, intracellular organic solutes are generated in an effort to restore cell volume and to avoid structural damage. This protective mechanism is important to remember when treating a patient with hypernatremia. Cerebral edema ensues if water replacement proceeds at a rate that does not allow for excretion or metabolism of accumulated solutes.

The effects of cellular dehydration are seen principally in the CNS, where stretching of shrunken neurons and alteration of membrane potentials from electrolyte flux lead to ineffective functioning. If shrinkage is severe enough, stretching and rupture of bridging veins may cause intracranial hemorrhage.

Frequency

United States

Hypernatremia occurs in approximately 1% of hospitalized patients. The condition usually develops after hospital admission. An incidence closer to 2% has been reported in debilitated elderly persons and in breastfed infants.[1,2]

International

Pediatric patients in developing nations may be at increased risk for hypernatremia because infant feeding may be complicated by poor maternal milk production (secondary to nutritional status) and errors in reconstitution of powdered formula.

Mortality/Morbidity

The mortality rate from hypernatremia is high, especially among elderly patients. Mortality rates of 42-75% have been reported for acute changes and 10-60% for chronic hypernatremia. Because patients with hypernatremia often have other serious comorbidities, precisely evaluating the degree of mortality directly due to hypernatremia is difficult. Morbidity in survivors is high, with many patients experiencing permanent neurologic deficits.

- Most deaths are due to an underlying disease process, rather than the hypernatremia itself. Delays in treatment (or inadequate treatment) of hypernatremia increase mortality.
- In hospitalized patients, persistent hypernatremia and protracted hypotension have been associated with a very poor prognosis.

Sex

Hypernatremia is diagnosed in males and females in equal numbers.

Age

Patients who present to the hospital with hypernatremia tend to be at the extremes of age. Breastfed infants occasionally present with hypernatremia in the first weeks of life, and elderly patients who are institutionalized are especially heavily represented.

Clinical

History

The history in the hypernatremic patient often points to the etiology of the syndrome. Search for any cause of extrarenal fluid losses (eg, burns, vomiting, diarrhea, fevers). Investigate the patient's perception of his or her fluid status and corrective measures he or she has taken. Does the patient complain of polyuria or polydipsia (ie, signs of DI or osmotic diuresis)? Does the patient have an intact thirst response? (This often is impaired in elderly persons.) A diminished thirst response is an indication to investigate the hypothalamus for a lesion in the thirst centers. For unclear reasons, patients with DI often crave ice-cold water.

In infants, seek sources of extrarenal losses, and investigate the patient's dietary habits. Hypernatremia in infants is often caused by improper preparation of formula or poor maternal milk production.

In patients who are hospitalized, reviewing the medicines and feedings the patient has received in search of an iatrogenic sodium load is imperative. Commonly identified sources include the administration of sodium bicarbonate during an arrest, high-sodium tube feedings, or overaggressive infusion of 3% isotonic sodium chloride solution. Pharmaceutical causes of nephrogenic DI should also be considered (see Causes).

Symptoms of hypernatremia tend to be nonspecific. Anorexia, restlessness, nausea, and vomiting occur early. These symptoms are followed by altered mental status, lethargy or irritability, and, eventually, stupor or coma. Musculoskeletal symptoms may include twitching, hyperreflexia, ataxia, or tremor. Neurologic symptoms are generally nonfocal (eg, mental status changes, ataxia, seizure), but focal deficits such as hemiparesis have been reported.

Physical

Physical examination findings in hypernatremia are nonspecific.

- Assessment of overall fluid status is important when determining the cause of the hypernatremia. Note signs of volume status, including mucous membranes, skin turgor, orthostatic vital signs, and neck veins.
- Because neurologic deficits are common in hypernatremia, perform a thorough neurologic examination.
- Significant hypovolemia can result when hypotonic fluid losses cause hypernatremia. The physical findings are those of dehydration or even hypovolemic shock, with tachycardia, orthostasis, and hypotension.

Causes

Hypernatremia is due to too little water, too much salt, or a combination thereof. The alteration can be in administration (too much salt or too little water) or output (too much dilute urine or extrarenal free water losses).

The most common cause of hypernatremia in elderly or institutionalized patients is lack of free water intake adequate to meet losses. Thirst is the body's main defense against increased serum tonicity. The thirst drive is activated through 2 pathways, one responsive to decreased intravascular volume and the other responsive to even slight increases in serum osmolarity. Most patients with an intact thirst mechanism and access to water can prevent the development of hypernatremia. Even patients with a defective renal concentrating mechanism (eg, patients with DI who may produce up to 20 L of urine a day) generally can keep up with water losses if they are allowed free access to water.

Some patients, however, cannot respond to their thirst drive. Infants and elderly patients who are debilitated depend on caregivers to provide fluids. Similarly, institutionalized patients may have limited access to water secondary to either external or internal constraints (eg, no access to water in their room, they believe the water is poisoned and refuse to drink it). Intrinsic water losses cannot be avoided, and some urine must be produced, even if it is maximally concentrated. Without access to water, these patients encounter a free water deficit, and their serum sodium level increases.

In some instances, the difficulty stems from an inability of the kidneys to concentrate the urine. This is known as diabetes insipidus (DI). DI can be due to a lack of a central stimulus to concentrate the urine (ie, lack of antidiuretic hormone [ADH] production [central DI]) or to a lack of renal response to such stimulus (ie, nephrogenic DI). The kidneys can fail to respond secondary to resistance to vasopressin or due to loss of the medullary-concentrating gradient for urine.

The differential diagnosis is most easily managed if the physician considers the patient's volume status.

- Hypovolemic hypernatremia (ie, water deficit >sodium deficit)
  - Extrarenal losses - Diarrhea, vomiting, fistulas, significant burns
  - Renal losses - Osmotic diuretics, diuretics, postobstructive diuresis, intrinsic renal disease
  - Adipsic hypernatremia is secondary to decreased thirst. This can be behavioral or, rarely, secondary to damage to the hypothalamic thirst centers.
- Hypervolemic hypernatremia (ie, sodium gains >water gains)
  - Hypertonic saline
  - Sodium bicarbonate administration
  - Accidental salt ingestion (eg, error in preparation of infant formula)
  - Mineralocorticoid excess (Cushing syndrome)
- Euvolemic hypernatremia
  - Extrarenal losses - Increased insensible loss (eg, hyperventilation)
  - Renal losses - Central DI, nephrogenic DI
    - These patients appear euvolemic because most of the free water loss is from intracellular and interstitial spaces, with less than 10% occurring from the intravascular space.
    - Typically, symptoms result if serum sodium level is more than 160-170 mEq/L.
- Central DI differential diagnosis
  - Head trauma
  - Suprasellar or intrasellar tumors

- Granulomas (sarcoidosis, Wegener granulomatosis, tuberculosis, syphilis)
- Histocytosis (eosinophilic granuloma)
- Infectious (encephalitis, meningitis, Guillain-Barré syndrome)
- Vascular (cerebral aneurysm, thrombosis, hemorrhage, Sheehan syndrome)
- Congenital
- Transient DI of pregnancy

- Nephrogenic DI (deficient renal response to ADH) differential diagnosis
  - Advanced renal disease (interstitial disease)
  - Electrolyte disturbances - Hypokalemia, hypercalcemia
  - Systemic diseases - Sickle cell disease, Sjögren syndrome, amyloidosis, Fanconi syndrome, sarcoidosis, renal tubular acidosis, light-chain nephropathy
  - Dietary disturbances - Excessive water intake, decreased salt intake, decreased protein intake
  - Drugs - Lithium, demeclocycline, colchicine, vinblastine, amphotericin B, gentamicin, furosemide, angiographic dyes, osmotic diuretics
  - Miscellaneous - Postobstructive diuresis, diuretic phase of acute renal failure, osmotic diuresis, paroxysmal hypertension

Differential Diagnoses

Hyperosmolar Hyperglycemic Nonketotic Coma

Other Problems to Be Considered

DI
Salt ingestion
Hypertonic dehydration
Delirium

Workup

Laboratory Studies

- Basic workup: When hyponatremia is discovered in a patient, obtain urine osmolality and sodium levels. Check serum glucose level to ensure that osmotic diuresis has not occurred.
  - The kidneys' normal response to hypernatremia is excretion of a minimal amount of maximally concentrated urine. If urine osmolarity is high, suspect extrarenal hypotonic fluid losses (eg, vomiting, low sodium diarrhea, sweat, evaporation from burns, low sodium ostomy output). The urine also is concentrated in salt overload states, although the total volume should increase.
  - Isotonic urine osmolality can be observed with diuretics, osmotic diuresis (mannitol, glucose, urea), or salt wasting.
  - Hypotonic urine and polyuria are characteristic of DI. Note, however, that partial DI can occur in which some concentrating ability remains, especially in the absence of a water load.
  - Serum sodium levels of more than 190 mEq/L usually indicate long-term salt ingestion.
  - Serum sodium levels of more than 170 mEq/L usually indicate DI.
  - Serum sodium levels of 150-170 mEq/L usually indicate dehydration.

Imaging Studies

- Head CT scan or MRI is suggested in all patients with severe hypernatremia.
  - Traction on dural bridging veins and sinuses caused by movement of water from the brain and brain shrinkage can lead to intracranial hemorrhage, most often in the subdural space.

- Hemoconcentration from total body water loss may lead to dural sinus thrombosis.
- Imaging studies may indicate a central cause for hypernatremia.

Other Tests

- Water deprivation test: With DI, water deprivation induces serum hyperosmolality and hypernatremia, but urine osmolality does not increase appropriately.
- ADH stimulation: With nephrogenic DI, urine osmolality does not increase after ADH or desmopressin acetate administration.

Treatment

Prehospital Care

Standard supportive attention to the ABCs is appropriate. Hypovolemic patients with signs of hemodynamic compromise (eg, tachycardia, hypotension) should receive volume resuscitation with isotonic sodium chloride solution.

If a thirsty patient's mental status allows, he or she does not need to be kept in a nothing-by-mouth status. In the debilitated nursing home patient, hypodermoclysis (subcutaneous fluid administration) may be considered as an alternative to transport to a hospital.

Emergency Department Care

The ED management of hypernatremia revolves around 2 tasks: restoration of normal serum tonicity, and diagnosis and treatment of the underlying etiology. When possible, providing free water to a patient orally is preferred.

Hypernatremia should not be corrected at a rate greater than 1 mEq/L per hour.

Carefully monitor all patients' inputs and outputs during treatment.

Consider CNS imaging to exclude a central cause or to identify CNS bleeding from stretching of veins.

Using isotonic sodium chloride solution, stabilize hypovolemic patients who have unstable vital signs before correcting free water deficits because hypotonic fluids quickly leave the intravascular space and do not help to correct hemodynamics. Once stabilization has occurred, free water deficits can be replaced either orally or intravenously.

Euvolemic patients can be treated with hypotonic fluids, either orally or intravenously (ie, dextrose 5% in water solution [D5W], quarter or half isotonic sodium chloride solution), to correct free fluid deficits.

Hypervolemic patients require removal of excess sodium, which can be accomplished by a combination of diuretics and D5W infusion. Patients with acute renal failure may require dialysis.

Traditionally, correction of hypernatremia begins with a calculation of the fluid deficit as shown below. Predicted insensible and other ongoing losses are added to this number and the total is administered over 48 hours. Recheck serum electrolyte levels frequently during therapy. To avoid cerebral edema and associated complications, the serum sodium level should be raised by no more than 1 mEq/L every hour. In patients with chronic hypernatremia, an even more gradual rate is preferred.

An alternative method to plan the correction of sodium imbalances has been proposed by Adrogue and Madias. They have devised a formula that can be used to calculate the change in serum sodium level after the administration of 1 L of a given infusate. This formula has the advantages of taking into consideration the tonicity of the infusate and encouraging reassessment of the treatment plan with each liter of solution or new set of electrolytes.

- Free Water Deficit = Body Weight (kg) X Percentage of Total Body Water (TBW) X ([Serum Na / 140] - 1)
  - Percentage of TBW should be as follows:
    - Young men - 0.6%

- Young women and elderly men - 0.5%
- Elderly women - 0.4%

- Example
  - A serum sodium level of 155 in a 60-kg young man represents a fluid deficit of

60 X 0.6 X ([155 / 140] - 1) or 3.9 L

  - With another 900 mL of insensible losses, the patient requires 4.8 L of fluid in the next 48 hours, resulting in an infusion rate of 100 mL/h.
- The Adrogue and Madias formula

Change in Serum Sodium = ([Na] Infused - [Na] serum) / (TBW + 1)

- The "1" in the denominator represents the extra liter of infusate added to TBW.
- When TBW is calculated as above, TBW = Body Weight (kg) X Percent Water

- Example
  - For the patient above, the expected change can be calculated with D5W or D5 half isotonic sodium chloride solution.
  - For D5W, Change = (0 - 155) / ([60 X 0.6] + 1) = -4.18 mEq/L
  - For half isotonic sodium chloride solution, Change = (77 - 155) / ([60 X 0.6] + 1) = -2.1 mEq/L
- If D5W is chosen to avoid fluid overload, an infusion rate of 250 mL/h results in a correction just over 1 mEq/h. (Note: This assumes the patient has no other losses during this time. Intrinsic losses make the correction slower [more conservative] than calculated.)

Consultations

- Patients with renal failure may require dialysis to help correct sodium and fluid balance.

Medication

Maintenance of adequate fluid intake is the most important therapy for all causes of DI that can result in hypernatremia. Hormonal and pharmacologic therapies must be tailored for the specific causes of DI (eg, central, nephrogenic). Central DI is treated with replacement therapy of ADH. The therapy for nephrogenic DI depends on reducing urine volume with combinations of salt restriction, thiazide diuretics, and prostaglandin synthetase inhibitors.

The other causes of hyponatremia do not require medications beyond hypotonic fluid for correction.

ADH replacement therapy

This therapy reduces free water loss and concentrates the urine.

Vasopressin (Pitressin)

Has vasopressor and ADH activity. Increases water resorption at the distal renal tubular epithelium (ADH effect) and promotes smooth muscle contraction throughout the vascular bed of the renal tubular epithelium (vasopressor effects). Vasoconstriction also is increased in splanchnic, portal, coronary, cerebral, peripheral, pulmonary, and intrahepatic vessels. Decreases portal pressure in patients with portal hypertension. A notable undesirable effect is coronary artery constriction, which may dispose patients with coronary artery disease to cardiac ischemia. This can be prevented with concurrent use of nitrates. Duration of action is approximately 3-6 h. Short half-life lessens the risk of acute water intoxication and makes it the ideal treatment of central DI in emergent situations.

Dosing

Adult

5-10 U IM/SC bid/qid prn

Pediatric

Not established

Interactions

Lithium, epinephrine, demeclocycline, heparin, and alcohol may decrease effects; chlorpropamide, urea, fludrocortisone, and carbamazepine may potentiate effects

Contraindications

Documented hypersensitivity

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Caution in cardiovascular disease, seizure disorders, nitrogen retention, asthma, or migraine; excessive doses may result in hyponatremia

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Desmopressin acetate (DDAVP)

Increases cellular permeability of collecting ducts, resulting in reabsorption of water by the kidneys. Duration of action is approximately 12-24 h. Has become the long-term treatment of choice for central DI.

Dosing

Adult

2-4 mcg/d IV/SC divided bid

Pediatric

3 months to 12 years: 5-30 mcg/d intranasally qd or divided bid

Interactions

Demeclocycline and lithium decrease effects; fludrocortisone and chlorpropamide increase effects

Contraindications

Documented hypersensitivity; platelet-type von Willebrand disease

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Avoid overhydration in patients when the drug is being used for its hemostatic effects

Follow-up

Further Inpatient Care

- Perform frequent reexaminations, especially neurologic examinations.
- Monitor electrolytes frequently (every 1-2 h during initial resuscitation, then every 4 h).

- Ensure adequate energy intake.
- Assess daily weights, intakes, and outputs.

Transfer

- Patients with hypernatremia who are fluid overloaded may require hemodialysis. If necessary, transfer these patients to a center with hemodialysis capabilities.

Deterrence/Prevention

- Prevention is directed at the underlying cause.
- Hypernatremia in infants is largely due to inappropriately reconstituted infant bottle formula. Avoid preparing homemade infant formulas, and never add salt to any commercial infant formula.

Complications

- Acute hypernatremia often results in significant brain shrinkage, thus causing mechanical traction of cerebral vasculature.
- Stretching of bridging veins can result in subdural hemorrhages.
- Venous congestion can lead to thrombosis of the intracranial venous sinuses.
- Arterial stretching can result in subcortical hemorrhages and cerebral infarctions.
- Seizures are possible.
- Hypernatremia of more than 2 days' duration is considered chronic hypernatremia and is associated with an increased mortality rate.
- Patients whose serum sodium level exceeds 180 mEq/L often have residual CNS damage.
- If hypernatremia is corrected too rapidly, brain edema and associated neurologic sequelae can occur. Patients with chronic hypernatremia are especially prone to this complication.

Prognosis

- Most patients survive, but residual neurologic deficits are common.
- Permanent neurologic sequelae have been reported in up to 30% of patients with acute hypernatremia.

Patient Education

- Because elderly patients often are affected, educating caretakers about dehydration avoidance measures is important.
- Patients with nephrogenic DI must be trained to avoid salt and to drink large amounts of water.

Miscellaneous

Medicolegal Pitfalls

- Legal liability can result if hypernatremic patients develop CNS deficits after rapid and aggressive hydration.
- In patients with hypernatremia who are debilitated, the possibility of neglect/abuse must always be considered.

Special Concerns

- When elderly, infant, or institutionalized patients are being treated for hypernatremia, ensure that social issues leading to the hypernatremic state (eg, requirement for more support with activities of daily living, improved access to water) are addressed.

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