Hypernatremia

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Case Presentation

A 68-year-old male with hypertension, coronary artery disease and central nervous system lymphoma presented with 1 week of dyspnea. On evaluation in the emergency room he was alert and complained of headaches, some gait instability, and progressive shortness of breath. His weight was stable at 82 kg. He did not report changes in his diet or urine volume, and he denied laxative use. His initial serum sodium was 150, urine Na 100, urine K 50. Imaging was concerning for a relapse of the central nervous system lymphoma and the patient was admitted for further management. His urine volume was 1.5 L per day. Over the next 7 days the patient decompensated neurologically and had marked hyperventilation with respiratory rates in the 40s. Serum sodium climbed from 150 to 174 despite addition of 1.8 L of intravenous D5W per day.

Question

What is the cause of the severe hypernatremia noted in this case and what is the approach to management of correction?

Answer The general approach to hypernatremia is to first look for a source of the water loss or a reason for limited access to water. In the ICU the most likely will be renal related water loss. Exogenous salt intake related hypernatremia is rare with the exception of usage of hypertonic saline. It is important to determine the free water deficit and the ongoing free water losses. The free water deficit = % total body water × kg weight × (current Na/desired Na – 1). % total body water depends on gender and age: Adult male 0.6, Elderly male 0.5, Adult female 0.5, Elderly female 0.45, Child 0.6. In our case, the free water deficit at admission is 0.6 × 82 × (150/145-1) = 1.7 L. Initial electrolyte free water clearance C_{H2O} = V × [1-(UNa + UK)/PNa] = 1.5 × [1-(75 + 25)/150] = 0.5 L where C_{H2O} is the electrolyte free water clearance, V is urine volume, UNa is urine sodium, UK is urine potassium and PNa is serum sodium. Thus the amount of free water lost in the urine is 500 mL which is negligible at hospital admission. But from the history of tachypnea, free water losses in this case may exceed the 1.8 L of intravenous D5W administered per day. Seven days later, the serum sodium was 174 and the electrolyte free water clearance was calculated again to be only 500 mL indicating that the free water losses were not renal related. The tachypnea observed was the source of the water losses. In normal conditions the amount of water lost in the skin and respiration is 600–800 mL. With central neurogenic hyperventilation, water losses can exceed 1 L per day. In our case, with an increase in serum sodium from 150 to 174, the free water deficit increased by 8 L in 7 days. At day 7, the free water deficit was 9.8 L despite receiving 1.8 L of D5W per day. The patient was treated with increased free water in the form of 2.8 L of IV D5W/ day for 5 days in order to correct the free water deficit and the ongoing losses from tachypnea. When the serum sodium was below 150 the amount of D5W was decreased to match ongoing losses at approximately 1.5 L of D5W per day depending on the direction of the daily serum sodium value. The cause of the severe hypernatremia in this case is free water loss via tachypnea related to a relapse of the central nervous system lymphoma. Central neurogenic hyperventilation is reported in central nervous system lymphoma.

Principles of Management

Risk Stratification

Hypernatremia (sodium concentration > 145 mEq/L) is not uncommon at ICU admission (2–6% of cases) and more commonly develops in the intensive care environment [1, 2].

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Table 49.1 Common causes of hypernatremia in the ICU

High free water losses	Low free water intake	Increased salt intake
Osmotic diuresis	Limited water access	IV 3% NaCl
Diabetes insipidus	Impaired thirst	
Insensible losses		
Osmotic diarrhea		

Several factors contribute to the risk of hypernatremia. With sedation and change in mentation, patients are not in control of the regulation of their water intake. Patients are dependent on healthcare professionals to estimate and deliver free water intake needs. Critically ill patients commonly have elevated losses of free water from renal (polyuria, post-ATN diuresis) or non-renal sources (fever, osmotic diarrhea) [3] (Table 49.1). Further, volume removal strategies and hypertonic fluid administration can contribute to the occurrence of hypernatremia.

Critically ill patients who have nutrition withheld have limited water intake from administration of medications, vasopressors or inotropic agents when mixed in D5W. Hypernatremia may present differently depending on the type of critically ill patient. Medical ICU patients in general are at higher risk of developing hypernatremia [1].Burn patients develop hypernatremia from insensible losses and sepsis [4]. Hypernatremia over 48 hours after surgery may be produced from unrecognized chronic diabetes insipidus which is normally managed by a patient's daily consumption of large amounts of fluid [5].

Evaluation

The primary step in evaluation of hypernatremia is the patient story, course, diet, past history, examination and medication usage history. If serum sodium in an ICU patient is above 145, the most important factor in diagnosis is determining if there is a source of water loss and if that loss is ongoing. We then can calculate the free water deficit which is indicative of the amount of free water that has been lost. If indeed the patient has polyuria then we can use the urine to determine the amount of ongoing free water losses.

Correcting Serum Sodium

The correction of serum sodium in hypernatremia is the combination of correcting the free water deficit and the ongoing free water losses and will depend on the serum sodium level. Treatment strategies include 5% Dextrose (D5W) for acute hypernatremia or half-normal saline for chronic hypernatremia if oral water cannot be tolerated. Literature in children demonstrated the risk of seizures and cerebral edema with correction >12 mEq/L day [6, 7] leading to the conservative therapeutic aim of correcting serum sodium levels <12 mEq/L day. Health care providers have more control of the correction of serum sodium as it is achieved by administration of IV D5W which can be titrated to the serum sodium over time [8]. A conservative strategy is to correct the free water deficit over 3 days while correcting for ongoing renal or non-renal free water losses. Serum sodium measurement in the ICU will be likely performed twice per day with the serum chemistry panel, and this monitoring schedule will suffice in most cases to allow for titration of IV or oral therapy.

Adverse Sequelae

Though more often seen in rapid correction of hyponatremia, it is reported that development of acute hypernatremia due to uncorrected severe water loss can rarely result in osmotic demyelination [9]. Too rapid correction of hypernatremia is unusual for reasons noted above [8]. Though demonstrated in children, the observation of cerebral edema with rapid correction of hypernatremia is not convincingly shown in adults [8]. Thus the of correcting serum sodium levels <12 mEq/L day is a reasonable and prudent therapeutic aim.

Evidence Contour

Diagnostics

Information that is useful for diagnostics include patient weight, serum Na, urine flow (volume over time), urine Osm, Plasma Osm, Urine Na, Urine K and Plasma Na. The following formulas are useful for determining the free water deficit and ongoing renal free water losses if present:

$$\text{Free water deficit} = \%\text{total body water} \times \text{kg weight} \times \left(\text{current Na} / \text{desired Na} - 1\right)$$

$$\text{Ongoing free water loss} = C_{H2O} = V - \left[\left(\text{Urine Osm} / \text{Plasma Osm}\right) * V\right]$$

or

$$C_{H2O} = V\left[1 - \left(\text{Urine Na} + \text{Urine K}\right) / \text{Plasma Na}\right]$$

V is urine flow (mL urine/24 h)

C_{H2O} will give the mL of free water that need to be replaced in 24 h to maintain the serum sodium at its current level.

Novel Treatments

There are no pharmacologic treatments for hypernatremia other than intravenous D5W or hypotonic fluids. Of note, 1 L of D5W will deliver 1 L of electrolyte free water while 1 L of half-normal saline will deliver only 0.5 L of electrolyte free water. Continuous Renal Replacement Therapy can be utilized as a strategy to slowly correct hypernatremia, especially in the context of Acute Kidney Injury or congestive heart failure [10, 11]. To blunt the fall in serum sodium, investigators report addition of 23% NaCl to the initial Continuous Renal Replacement Therapy solution [12]. A simpler strategy that the author has personal experience with is to titrate low dose 3%NaCl via a central line while Continuous Renal Replacement Therapy is running. To maintain therapeutic hypernatremia in patients with Acute Kidney Injury and Traumatic Brain Injury it is possible to utilize low dose peripheral hypertonic saline (3% NaCl) in combination with Continuous Renal Replacement Therapy [13].

Outcomes

Critically ill patients with hypernatremia are noted to have elevated mortality and longer lengths of stay in hospital when compared to those with normal serum sodium [1, 14]. Hospital or ICU-acquired hypernatremia in the critically ill has higher mortality compared to hospital admission hypernatremia [15, 16]. These mortality observations are similar to the U shaped outcome associations of most blood parameters measured in critical illness (K, Cl, Cr, BUN, etc.). Outcome in the ICU is certainly tied to severity of disease and chronic illnesses which is reflective in chemistry profiles. Causation of the hypernatremia-mortality association is limited by confounding. Indeed, hypernatremia is not always a sign of bad outcomes. For example, in patients with acute tubular necrosis hypernatremia is common [17] but is a sign of renal recovery during "post-ATN diuresis" due to electrolyte free water losses, and renal recovery is associated with better mortality in ICU patients with AKI [18].

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