Case: A 77yo woman is admitted to your service from a nursing home with altered mental status. Her admission Na is 159.
What additional information will you ask for?
How will you begin to work her up?
How will you treat her?

Hypernatremia

Jennifer Best, MD
Harborview Medical Center

How does hypernatremia develop?
Two ways: Urine is not adequately concentrated or hypotonic fluid is wasted without replacement

Who is at risk?
Elderly patients, patients on hypertonic infusions, tube feedings, osmotic diuretics, lactulose or mechanical ventilation, patients with altered mental status prohibiting access to water, uncontrolled diabetics with pronounced osmotic diuresis or those with other polyuric disorders.

What is the impact of hypernatremia?
Hypernatremia is less common than hyponatremia, but has a higher mortality (10-60% if chronic, 75% if Na >160 – difficult to sort out death from underlying cause from death related to high Na.)

How are the causes of hypernatremia classified?
*Water loss (*takes two forms):

Normal total body Na:

Causes: pronounced **pure** water loss. Extrarenal: fever, hyperventilation, mechanical ventilation. Renal: Diabetes insipidus - nephrogenic or central) or hypodipsia. **Clinically:** Seen in individuals without free access to (or capacity to recognize need for) water. Patients usually clinically euvolemic, but ECF contracted. **Uosm**: high if extrarenal, low if DI. **UNa:** variable

Low total body Na: Loss of hypotonic fluids (i.e. sodium and water lost together, but H20 losses >Na losses)

Causes: Renal losses (diuresis - osmotic via mannitol, glucose or urea OR loop diuretics) or extra-renal losses (diarrhea, sweating). **Clinically**: patients appear hypovolemic (e.g. orthostatic, tachycardic, dry mucous membranes). **UNa**: <10-20 if extra-renal, >20 if renal
OR

Sodium gain/ high total body Na: least common. Usually iatrogenic.

Causes: infusion of hypertonic solutions (3%NaCl, HCO3), salt tablets, dialysis complications, tube feedings, Na-containing antibiotics, hypertonic dialysis, glucocorticoid excess, hyperaldosteronism. **Clinically**: Hypervolemic. **UNa**: >20

How can I pinpoint the underlying cause?
History is most helpful. The urine osmolality is also useful. If >700-800, likely impaired water intake (impaired thirst, altered mental status, lack of H2O availability, illness). If <700-800, likely impaired water intake and impaired renal conservation (renal failure, DI, extrarenal losses); rule out an osmotic diuresis.

What are the signs and symptoms?
The development of symptoms generally requires a change resulting in a significant elevation (Na >158-160) over a short period of time. Symptoms are related to hyperosmolarity (hypernatremia is always a hyperosmolar state; Na is the primary contributor to serum osmolarity).

Neuro/Psych (most common): altered mental status, lethargy, irritability, restlessness, hyperreflexia, chorea, focal deficits, seizures (rare, except in cases of exogenous sodium loading or aggressive IVF), coma, bleeding secondary to alteration in brain volume (shrinkage occurs when water travels from brain cells into more hypertonic environment)
CV: symptoms of hypovolemia, including hypotension, tachycardia
MS: muscle twitching, spasticity
GI: nausea/vomiting
Pulm: dyspnea
Endo: increased thirst (may not persist)

Management

Two pronged approach: 1. Identify and treat the underlying cause.
2. Correct osmolar imbalance by replacing what was lost (water, hypotonic fluids +/- electrolytes) or ridding the body of excess sodium

Use the volume status to guide you:

Hypovolemic: low total body Na, orthostasis: restore hemodynamics with NS, then change to D5W or ½ NS

Hypervolemic: Excess total body Na. Give loop diuretics to increase Na excretion and then replace D5W to correct hypertonicity. Dialyze if kidneys are not working.

Euvolemic: normal total body Na. Give D5W.

How quickly can I decrease the serum Na?

If the Na has risen over a matter of <12 hours, it can be correctly quickly without consequence. If elevated for longer than 12 hours or if the onset is unclear, decrease Na by no more than 10 mmol/L/day or 0.5 mmol/L/hr. Goal is 145.

What is the primary consequence of inappropriate treatment of hypernatremia?

If the hypernatremia is prolonged, brain cells will have had enough time to re-equilibrate and regain lost mass by osmolar shifts. Aggressive treatment with hypotonic fluids can cause cerebral edema, as cells will swell under the effects of hypotonicity.

What is the best way to administer fluids?

PO or PFT replacement in the form of free H20 bolus is preferred when possible. IV is second line. Only hypotonic fluids should be used (D5W, ¼ NS, ½ NS). Use NS only when there is hemodynamic instability, and only until this is corrected, at which point another fluid should be chosen. LR is another alternative for establishing hemodynamic stability; it has less sodium than NS, but the same rule applies.

How do I decide how much fluid is necessary?

One approach:

1. *Calculate the total body water* (TBW): ***0.6 x (wt in kg)***
2. Select your fluid and identify the amount of Na in mmol/L

D5W	0
¼ NS	34
½ NS	77
LR	130
NS	154

3. *Calculate the effect of 1 L of your selected fluid on serum Na* according to this formula:

Change in serum Na for 1L of fluid of choice =[IVF Na - serum Na] divided by [TBW + 1]

If you are also giving K in your IVF, modify the formula as follows:

Change in serum Na for 1L of fluid of choice =[(IVF Na + IVF K) - serum Na] divided by [TBW + 1]

4. *Decide how quickly you want to correct*. In cases of prolonged hypernatremia, divide 10 (the desired drop) by the number obtained above to calculate the amount of IVF required over the next 24 hours to decrease the serum Na appropriately. When hypernatremia has been shorter-lived, divide the number necessary to reach 145 by the number of hours over which you want to correct.
5. *Account for average obligatory 24 hour water losses* (1.5L or so)
6. *Convert to mL and divide by 24 to obtain mL/hour*

NOTE: It is felt by some authors that the classic formula: ***water deficit = TBW x (1-[140/serum Na])*** is only useful in cases of pure water loss, and is not accurate if hypotonic fluid is lost or if potassium infusion is necessary.

Another proposed formula:
Fluid requirement = [desired Δserum Na x TBW] / [{serum Na - (IV Na + IV K)]

How do I monitor my progress?

Obtain electrolytes every 6-8 hours. Be vigilant for hyperglycemia if D5W is used.

Case follow up. A call to the nursing home revealed that the patient had recently stopped eating and drinking, and had also been vomiting for the past few days. On physical exam, the patient was tachycardic and mildly hypotensive, but not unstable. WBC was 14. K was 3.0. Urine Na was <20. Given her vomiting and hypokalemia, the decision was made to use ½ NS with 20mEQ of KCl. As her weight was 50kg, her TBW was calculated to be 30kg. As her hypernatremia was of uncertain duration, it was planned to correct no faster than 10 over the first day.

Change in serum Na = (IVF Na + IVF K) - serum Na/TBW + 1
Change in serum Na = (77 + 20) - 159/30 + 1
*1*L of 1/2NS with 20KCl would correct by 2 pts
5L would be necessary to correct 10 pts
+ 1L was added for ongoing losses = 6L = 250cc/h

Urine culture was positive for Klebsiella. The patient was treated for pyelonephritis with antibiotics and antiemetics. Her sodium normalized over the ensuing 3 days and her mental status improved, as did her PO intake.

Clinical Pearls

1. A urine osmolarity of <700-800 implies that the kidney is not concentrating adequately and a search for the cause should result.
2. Assume that hypernatremia is chronic and treat it accordingly, unless you have definitive evidence to the contrary.
3. Normal saline has no role in the treatment of hypernatremia, with the exception of short term use to reestablish hemodynamic stability.
4. Be sure to account for ongoing obligate free water losses in repleting fluids.
5. Control hyperglycemia with insulin if it develops while using D5W, as this can contribute to further free H2O losses.
6. Use the GI tract whenever possible to replace free water in hypernatremia.

References

1. Kumar A, Berl T. Sodium. Lancet 1998;352:220-228
2. Adrogue HJ, Madias NE. Hypernatremia. N Engl J Med 2000;342(20):1493-1499.
3. Kang S-K, Kim W, Oh MS. Pathogenesis and treatment of hypernatremia. Nephron 2002;92(suppl 1):14-17.
4. Verbalis JG. Disorders of body water homeostasis. Best Pract Res Clin Endocrinol Metab. 2003 Dec;17(4):471-503.
5. Weiss-Guillet E-M, Takala J, Jakob SM. Diagnosis and management of electrolyte emergencies. Best Pract Res Clin Endocrinol Metab. 2003 Dec;17(4):623-651.

Last update: January 13, 2005/JAB