

# Hyponatremia

## Definition

- Mild: Serum Na 130-134mmol/L
- Moderate: Serum Na 120-129mmol/L
- Severe: Serum Na <120mmol/L
- Acute: <48hrs since development of hyponatremia
- Chronic: >48hrs since development of hyponatremia. (hyponatremia should be considered chronic whenever the duration is unknown).

**Evaluation:** Order serum osmolality, urine sodium, urine osmolality, TSH, Lipid panel.

Tips: Free water balance (Urine osmolality) is regulated by ADH. Sodium excretion (urine sodium) is regulated by aldosterone. If a patient has ESRD, the cause of hyponatremia is excess free water intake in the setting of impaired Kidney water excretion, and is not mediated by ADH.

**Step 1: Differentiate from true hyponatremia from pseudohyponatremia.**

- A normal or elevated effective serum osmolality (280mOsm/kg or greater) suggests pseudohyponatremia.
- Hypertonic states, like hyperglycemia or mannitol use, can cause hyponatremia by drawing water extracellularly and lowering serum sodium concentration. This is a true hyponatremia. Serum osmolality will be high, sodium should normalize with correction of hypertonic state.
- Correction formula for hyperglycemia:  $\text{corrected Na} = \text{measured Na} + [(\text{serum glucose} - 100)/100] \times 1.6$ . You can also use MD Calc
- Hyperlipidemia or increased protein can also cause a lab error that results in a falsely low sodium result.

**Step 2: is ADH high or low? Compare serum osmolality to urine osmolality.**

- If serum osm > urine osm, then ADH is low (kidneys are appropriately responding by maximizing water excretion). Causes are excess water intake or inadequate solute intake. A urine osm <100 also suggests normal free water excretion.
- **If serum osm < urine osm, ADH is high. Proceed to step 3**
- Check for iatrogenesis: are there high rates of hypotonic solution infusing?

- Look at what medications are mixed in (ex: IV abx in 500ml D5W 4x/day = 2L of free water).
- Check volume of free water flushes in patients getting tube feeds or other hypotonic oral intake.
- Consider primary polydipsia.
- Causes of inadequate solute intake: tea and toast diet (carbs and fats metabolize to water and CO<sub>2</sub>, so do not count as solutes), beer potomania (high beer intake relative to solute intake).

### Step 3: If ADH is high, what is the volume status?

If ADH is activated, urine osmolality is usually  $>100$ . Urine Na can help to determine RAAS activation, which can narrow the differential in cases where volume status is not clear. A low urine Na ( $<20$ ) suggests RAAS activation, as seen in hyper or hypovolemia; a high urine sodium may suggest SIADH.

#### Hypervolemic: DDx: **CHF, nephrotic syndrome, liver failure.**

- ADH is released in response to low effective arterial blood volume due to third spacing or poor perfusion.
- Urine Na will be low ( $<20$ ) since the RAAS is activated in response to low arterial blood flow.

#### Hypovolemic:

- ADH is being released in response to low effective arterial blood flow due to fluid loss
- **Extrarenal losses**, like GI loss: urine Na will be low ( $<20$ ) since aldosterone will also be activated
- **Renal salt wasting**: urine Na will be high ( $>20$ ). Causes include salt-wasting nephropathy, adrenal insufficiency, cisplatin, thiazide diuretic use.

#### Euvolemic:

- **Hypothyroidism**: check TSH (The main mechanism for the development of hyponatremia in patients with chronic hypothyroidism is the decreased capacity of free water excretion due to elevated antidiuretic hormone levels)
- **Glucocorticoid deficiency**: check AM cortisol
- **SIADH**: Inappropriate release of ADH independent of effective arterial blood flow. Urine Na will be high, since aldosterone is low.
- **SIADH is a diagnosis of exclusion with following features:**
  - Clinical euvolemia
  - Normal thyroid and adrenal function, no recent diuretic use
  - Lab findings: Urine osmolality  $>150$  mOsm, serum osm  $<275$  mOsm, Urine Na  $>20$  mmol/L with normal dietary salt intake.

**Management:** Repeat BMP q4hr. Goal to increase Na no more than 4 to 6 mEq/L in 24hrs to prevent osmotic demyelination syndrome. Start treatment based on volume status

Evaluate and treat severe symptoms emergently in all patients

- The presence of these signs suggests cerebral edema and required rapid treatments: visual changes, neurologic deficits, encephalopathy, coma, respiratory arrest, and seizures.
- Start on hypertonic 3% saline immediately at rate 15 to 30mL/hr and consult nephrology. Monitor serum sodium hourly while on hypertonic saline. If sodium is correcting too fast, stop hypertonic saline and start on D5W infusion.

Select treatment approach based on volume status, severity, and etiology.

### **Hypovolemia:**

- Start on IV fluids Normal saline @100mL/hr. Caution: once volume status is corrected, a brisk aquaresis may ensue and cause overcorrection, thus, strict urine output and frequent sodium monitoring is critical. The earliest and most concerning indication of overcorrection is brisk urine output and/or a decrease in urine osmolality.

### **SIADH:**

- Treatment of underlying cause/withdrawal of causative agents as possible.
- Start with fluid restriction 1-1.5L/day but restriction alone is often inadequate. Do not restrict beyond 1L, as that is unlikely to add additional benefit and can cause significant discomfort.
- Use urine electrolytes (Urine sodium + Urine potassium) to guide therapy:
  - $UNa + UK < \text{serum Na}$ : Positive free water clearance. Patient is still urinating out free water but not enough to improve hyponatremia. This suggests fluid restriction will be an effective treatment.
  - $UNa + UK > \text{serum Na}$ : Negative free water clearance. All free water is reabsorbed and any urination will continue to lower serum Na. This suggests osmole supplementation (hypertonic saline or salt tabs) is needed to treat hyponatremia.
  - Any IV fluids with Na content less than  $UNa + UK$  will result in worsening hyponatremia because the kidney is able to excrete the solute but reabsorb the free water from the infused solution. Therefore,  $UNa + UK > 154$  is typically an indication that hypertonic saline is needed to correct hyponatremia.
- Additional therapies for SIADH:
  - Hypertonic saline: Effective (3% = 513mEq/L), necessary when  $UNa+UK$  is very high. Requires good venous access. Not a long term therapy.

- NaCl tabs: 1g NaCl tab = 17mEq Na and 17 mEq Cl. Not a very high osm load per tab (typically need upwards of 2g TID). Difficulty pill burden, can stimulate thirst counteracting fluid restriction and cause GI upset.

### **Hypervolemia:**

- Free water restriction (1-1.5L/day)
- Loop diuretics to optimize volume status
- Hypertonic saline and salt tabs generally NOT recommended as they will worsen volume overload.

### **If the serum sodium has been overcorrected:**

- IV D5W
- Can give 3mL/kg/hr D5W to lower [Na] by approximately 1mEq/L/hr if overcorrected
- Pay attention to ongoing losses: if urine output is brisk (>150mL/hr) and dilute, the patient is losing free water rapidly and it is raising serum Na rapidly. Suggest nephro consultation to assist with safe correction strategies (eg: titrating D5W drip to a % of urine output or using DDAVP to inhibit the aquaresis)

### **Diagnosis and Management of disorders of body tonicity-Hyponatremia and Hypernatremia**

#### **Clinical practice guideline on diagnosis and treatment of hyponatremia**

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