Hyponatremia
Diagnosis and Treatment in the Acute Setting

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Patient Presentation:

MP is a 57yo male presenting to the ER with history of a syncopal episode and confusion. His friend had called EMS after finding the patient passed out on the bathroom floor of his home. Upon arrival to the ER, patient is awake and alert. The patient is oriented to person/time but not place. He complains of mild nausea and EMS notes that he vomited one time on the way to the hospital.

PMH:  PSH:  Medications
-HTN  -R inguinal hernia  -Metoprolol
-Anemia  
-GERD  

Family Hx:  Social Hx:
-Mother/Father – alcohol abuse  -Divorced x 5 years
-Father – heart disease  -Laid off from aircraft manufacturing job 1 month prior
  
Drugs: no illicit drug use

PHYSICAL EXAM:
GENERAL: Alert, Oriented x 2 He does not appear to be in acute distress.
HEENT: Normocephalic, atraumatic. Pupils equal, round, and reactive to light. Pupils are pupils are 3 mm. Mucous membranes are dry.
NECK: Supple without JVD, adenopathy, or thyromegaly.
LUNGS: Exam revealed diminished breath sounds bilaterally, but no wheezes or rales.
HEART: Regular rate and rhythm without murmur.
ABDOMEN: Soft and nondistended with positive bowel sounds. Mild epigastric tenderness to palpation. His liver is palpable and large.
NEUROLOGIC: No focal neurological deficit
EXTREMITIES: No cyanosis/edema
Hyponatremia

- Defined as serum sodium <135 mmol/L

- One of the most common abnormalities found in hospitalized patients

- Symptoms: nausea, vomiting, confusion, lethargy, disorientation

- Severe Hyponatremia <120 mmol/L – can have seizures, central herniation, coma, death.

- Symptoms caused by Hypo-Osmolar State
What tests will are necessary to determine etiology of the hyponatremia?

- Glucose
- Lipid Panel
- Serum Osmolality
- Urine Osmolality
What caused the hyponatremia, and how do I treat it?
Hyponatremia may be divided into three different categories based on serum osmolality:

- Hyper-Osmolar Hyponatremia
- Iso-Osmolar Hyponatremia
- Hypo-Osmolar Hyponatremia
Hyper-Osmolar Hyponatremia
- AKA Translocational or Hypertonic
- Glucose or Manitol added to blood
Glucose/Manitol Added
Intravascular Osmolality Increased to 320
→ Fluid Shift (0.5L) from Intracellular to Intravascular space to equilibrate Intravascular/Intracellular Osmolality

Intravascular space
-Osmolality:285
-Volume: 5L
Total Sodium: 700mEq
Na= 140mEq/L

Asymptomatic, as hypo-osmolality is not present

Glucose/Manitol Added
Intravascular Osmolality Increased to 320
↓
Fluid Shift (0.5L) from Intracellular to Intravascular space to equilibrate Intravascular/Intracellular Osmolality
↓
Intravascular space
-Osmolality:>290
-Volume: 5.5L
Total Sodium: 700mEq
Na= 126 mEq/L

Corrected Sodium :
Glucose/60 + Measured Serum Sodium
Iso-Osmolar Hyponatremia
- AKA Isotonic or Pseudo-Hyponatremia
- Elevated Triglycerides
- Triglycerides add intravascular VOLUME with minimal change in Osmolality because of very LOW osmolar weight.

- Most labs account for this when making Na calculation so generally a non-issue
Hypo-Osmolar Hyponatremia

-“True” Hyponatremia
CHECK URINE OSMOLALITY!!!

Increased Urine Osmolality (>100) – ADH Dependent
  - Hypovolemic
  - Euvolemic
  - Hypervolemic

Decreased Urine Osmolality (<100) – ADH Independent
  - Decreased Solute intake (Potomania/Malnutrition)
  - Increased H2O intake (psychogenic polydipsia)
High Urine Osmolality (>100)
- ADH is present, causing concentration of urine
- Further Diagnosis/Tx based on PHYSICAL EXAM
HYPOVOLEMIC
- **PE**: NO peripheral edema, lungs CTA, + orthostasis, tachycardic, dry mucus membranes, decreased skin turgor
- **Etiology**: Diuretics, Diarrhea, Vomiting, Decreased PO intake
  - ADH is increased secondary to hypotension
- **Treatment**: IV Fluids to eliminate orthostasis– Treat the cause!

EUVOLEMIC
- **PE**: NORMAL!!! – No orthostasis, no edema, lungs CTA, normal vitals
  - ADH is INAPPROPRIATELY increased!
- **Etiology**: SIADH, Addisons, Hypothyroidism
- **Treatment**: Fluid restriction, Treat the cause!

HYPERVOLEMIC
- **PE**: Peripheral edema, pulmonary edema, ascites, pleural effusion
  - ADH is increased secondary to hypotension
- **Etiology**: CHF, Decreased oncotic pressure secondary to decreased albumin level - (Cirrhosis, Nephrotic syndrome, Malnutrition)
- **Treatment**: Fluid AND Salt restriction, Diuretics
Low Urine Osmolality (< 100)
- Beer Potomania
- Psychogenic polydipsia
- Malnutrition

These conditions all involve a decreased solute load

Treatment is to INCREASE solute load and/or DECREASE fluid intake.
- Normal patients can **dilute** their urine to a **minimal** osmolality of 60 mmol/L

- Additionally, most patients can **concentrate** their urine to a **maximal** osmolality of 1200 mmol/L

- A normal Patient will eat, on average, 10mmol/kg/day of solute
  – The body will excrete what they take in to preserve steady state

  **Example Patient:** 60kg
  - Consumes 600 mmol of solute daily
What range of fluid intake can this patient have per day to maintain steady state?

**Maximal Concentration:**
600 mmol/1200mmol/L = 0.5 L

**Maximal Dilution:**
600mmol/60mmol/L = 10 L
Examples:

- 57 yo depressed male:
  - drinks 12 pack daily (4.25L)
  - significantly decreased appetite
    (3mmol/kg/day of solute)
  - Maximal H2O output (dilute urine)
    180mmol intake/60mmol/L dilution = 3L

- Net + 1.25 L daily!!!
-36 yo schizophrenic female:
  - Smokes 2.5 packs per day, and drinks 13 “Big Gulp” cups of sweet tea per day. (30 oz each = 390 oz or 11.5L)
  - Normal Appetite / Intake
  - Maximal H2O output (dilute urine)
    600 mmol intake/60mmol/L dilution = 10L

-Net + 1.5 L daily!!!

Treatment for BOTH:
Decrease Fluid intake And/Or increase solute load!
Treatment Goals for Hyponatremia

1) Correct symptoms of hyponatremia
   Usually occurs with increase of sodium by 4-6mEq
   Hypertonic saline ONLY if severe sx (seizure, coma, etc)

2) Correct hyponatremia
   Goal: increase by 4-6mEq in 24 hours
   No more than 9mEq in 24 hours and 18mEq in 48 hours

3) Avoid rapid correction because of Central Pontine Myelinolysis (CPM) risk
   -Greatest risk is those with chronic hyponatremia (>2 days)
Osmotic Demyelination syndrome/ Central Pontine Myelinolysis

Most cases occur in patients with:
- severe hyponatremia (<120 meq/L) for >2-3 days
- sodium corrected >10-12meq/24 hr or >18 in 48 hours.

Other risk factors
- Alcoholism
- malnutrition
Intravascular

300

250

270

Brain

300

270

H₂O

Na, K, Organic salts

H₂O

270

230

Credit: Dr. J. Moussa, MD
Symptoms present 2-6 days after rapid correction
- Often irreversible or only partially reversible
- Dysarthria, dysphagia,
- Paraparesis/quadraparesis
- Behavioral disturbances, lethargy, confusion, disorientation, obtundation, coma, ‘locked-in’

Poor Prognosis
- Prevention is key
- Supportive treatment for 6-8 weeks to determine whether irreversible, as some patients do recover

- Lesion may not appear on MRI until 4 weeks after disease onset
The ER gave my patient the ‘standard’ 2L normal saline bolus, and my patient’s serum sodium is rising too fast!!! What do I do???

Decrease Serum Sodium to obtain 24 and 48 hour goals as able:

-D5W - 6mL/kg over 2 hours
  -Lowers serum sodium about 2meq/L
  -Repeat until reach goal

-Desmopressin - 2mcg IV or SC q 6 hours
  -Continue even if reached goal to prevent overly rapid correction

Rate of relowering
  -1meq/hr
SUMMARY:

-One of the most common abnormalities found in hospitalized patients
-Lab tests needed for workup: Glucose, Lipid Panel, Serum Osmolality, Urine Osmolality
-”True” Hyponatremia differentiated by urine osmolality
-Find/Treat the cause!
-Correct Slowly!
Sources:

-Woxland, H (2013) *Salt: the good, the bad, the ugly* [PowerPoint slides], Courtesy of the author.