

Case Studies in Fluids and Electrolytes

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Question 1

- A 32 y.o. male with a history of schizophrenia and compulsive water drinking is admitted for vomiting, diarrhea and new onset seizure. His serum sodium is 115 mg/dL, three days ago it was 133 mg/dL. Your next step would be?
 - a. Sedation, intubation and mechanical ventilation
 - b. MRI of the brain
 - c. 80 mg of furosemide i.v.
 - d. Evaluate his volume status
 - e. Order a pro-BNP

In Any disorder of Sodium and/or Water, the Most critical factor is to determine the patient's volume status.

Res ipse loquitor

Question 2

- You are caring for a patient who has been on lithium for years for bipolar disorder and now complains of polydipsia and polyuria. He has a normal glucose level and you order an 8 hour water deprivation test followed by an i.v. dose of DDAVP. The following lab is then obtained: serum osmolality 271 mOsm/Kg, urine osmolality 514 mOsm/kg and after DDAVP 509 mOsm/kg. These results are consistent with which of the following?
 - a. Psychogenic polydipsia
 - b. Central diabetes insipidus
 - c. Nephrogenic diabetes insipidus
 - d. SIADH
 - e. I would like to buy a vowel

Interpretation of a Water Deprivation Test

	Primary Polydipsia Cortical Washout	Central Diabetes Insipidus	Nephrogenic Diabetes Insipidus
Serum Osmolality mOsm/kg	<300	>300	>300
Urine Osmolality mOsm/kg	>600 (>400-400 if chronic)	<200-300	<300
Post DDAVP dose Urine Osmolality mOsm/kg	No change	>400-600	No change

Mills F Biochemistry Department North Bristol NHS Protocol

Be **Familiar** with This Chart

Water Deprivation Test

- Goal to differentiate 3 causes of polyuria:
Psychogenic polydipsia, Central DI, or Nephrogenic DI (other etiologies are excluded)
- If first am void has a urine osmolarity >600 mOsm/kg DI is **EXCLUDED**

8 am have patient void and obtain: serum and urine osmolarity and weigh patient, make patient NPO

Every hour: weigh patient, record urine volume and urine osmolarity

Every 2 hours: serum osmolarity

Diabetes Insipidus

- Central-due to trauma, edema, ischemia or removal of the Pituitary, or congenital
- Nephrogenic-due to the kidneys loss of response to ADH, drugs, obstruction, ureteral reflux, salt wasting, chronic renal disease, congenital
- Large volumes of dilute urine (10-15 l/day)
- Polydipsia

Question 3

- You have a patient with a serum sodium of 122 mmol/L. The PMH is negative as is the exam, the patient is euvolemic. All of the following would be consistent with SIADH except?
 - a. Urine sodium of 15 mmol/L
 - b. Urine osmolality > 100 mOsm/kg and $>$ serum osmolality
 - c. Serum osmolality < 275 mOsm/kg
 - d. The serum sodium < 135 mmol/L
 - e. May I buy a consonant

Diagnosis of SIADH

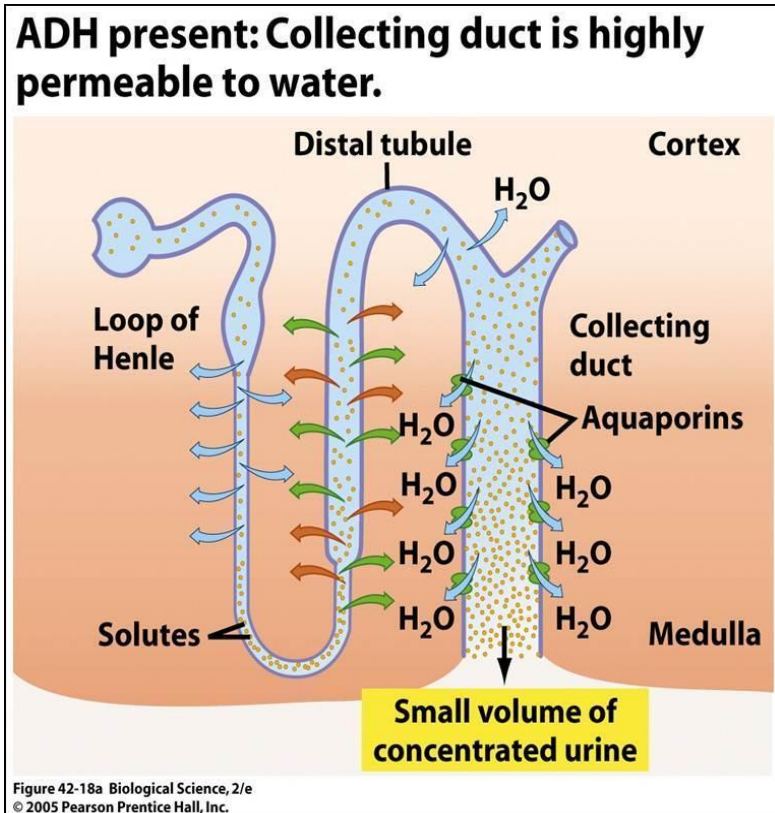
(Bartter and Schwartz 1967)

- Euvolemia
- Normal cardiac, renal, hepatic and thyroid fxn, no edema
- Hyponatremia <135 mmol/L
- Hypoosmolar serum <275 mOsm/L
- Urine Na $>20-30$ mmol/L
- Urine osmolarity >100 mOsm/L and $>$ serum osmolarity
- Low serum uric acid

Question 4

- You are rounding on service and you have a patient who was admitted with SIADH and a serum sodium of 119 mmol/L. Your junior resident has already seen the patient and has given the 2 liters of normal saline. What will be the body's response to this treatment?
 - a. The serum sodium will increase with the added sodium
 - b. The kidney will urinate the excess water and no change in the sodium level
 - c. The serum sodium level will fall due to excess water reabsorption
 - d. The serum sodium will fall due to suppression of ADH from the fluid
 - e. The patient will develop acute kidney injury

Effects of Normal Saline in SIADH



- Water is maximally reabsorbed, while sodium is not, which further dilutes the serum sodium/osmolarity worsening the hyponatremia.

Therapy of SIADH

Proper Treatment

- Fluid restriction 800-1000 ml/day
- Stop offending drugs
- Correct underlying causes, if possible
- *Primum non nocere*
- 3% saline *if* indicated, *not* normal saline!!
- Vaptans (Tolvaptan, Conivaptan) V_2 and V_1 antagonists cautiously
- Urea

No Normal Saline, EVER in SIADH!



Question 5

- A 31 y.o. African-American female collapses at mile 20 of a marathon. Although the temperature is in the 60's she was advised by her coach to drink as much water as possible. She vomits a large volume of clear fluid and has a seizure. Her BP is 134/74. A serum sodium is found to be 122 mmol/L. Your next step would be?
 - a. 1 liter of normal saline
 - b. 1 liter of 3% saline
 - c. Amiloride
 - d. Furosemide
 - e. 100 ml of 3% saline every 10 minute up to 3 doses

Exercise Associated Hyponatremia and Exercise (EAH) Associated Hyponatremic Encephalopathy (EAH- E)

Definition: EAH is the occurrence of hyponatremia during or up to 24 hours after prolonged activity and define as a serum or plasma Sodium below the normal reference range of the lab performing the test (<135 mg/dl)

EAH Definition (2)

- EAH is a dilutional hyponatremia caused by an [RAPID] increase in total body water relative to the amount of total body exchangeable sodium. The primary etiological factor is the consumption of fluids (water and sports drinks) in excess to total body fluid losses: insensible (transcutaneous, respiratory and GI) and renal losses.

2nd Conference on Exercise Associated Hyponatremia 2007 (2nd Conference EAH)

Clin J Sport Med; 18(2): 111-21, 2008

EAH-Encephalopathy (EAH-E) Definition

Neurological changes associated with ingestion of large quantities of water and/ or hypotonic fluids. These changes are a direct result of cerebral edema. Manifestation may include: headache, dizziness, clouded sensorium, confusion, obtundation, coma, myoclonus, seizures and death.

2nd Conference EAH 2007

Indications for 3% Saline (513 mEq/L Na)

- Seizures
- Acute mental status change
- Severe headache
- Focal neurological findings
- Myoclonic jerks or new tremors
- Normal Saline 0.9%, 1l=154 mEq/l Na
3% Saline, 1l=513 mEq/l Na

Principles of Treatment 3% Saline

- A SMALL correction can lead to a good outcome. (2-3 mEq/l initially) Do *not* over correct or normalize.
- Do *not* correct unless evidence of hyponatremia is present or compelling clinical situation e.g. seizure, myoclonus, confusion, coma, etc.
- 100 ml of 3% Saline should provide some improvement, if none it may be repeated every 10 min. for a total of 3 doses or clinical improvement
- Fluid restriction
- Nephrology consult

2nd Conference Exercise Associated Hyponatremia *Clin J Sport Med*; 2008; 18:111-121.

Question 6

- Another water deprivation test is performed and DDAVP given at the completion of the test with the following results: Serum osmolality 320 mOsm/Kg, Urine osmolality 272 mOsm/Kg and after DDAVP 270 mOsm/kg. These results are consistent with which diagnosis?
 - a. Psychogenic polydipsia
 - b. Central diabetes insipidus
 - c. Nephrogenic diabetes insipidus
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Mills F Biochemistry Department North Bristol NHS Protocol

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Treatment of Nephrogenic DI

- If due to Lithium either change medication, if not feasible add Amiloride (blocks the ENaC Channel and decreases the uptake of Li^{++})
- Chronic Lithium therapy can lead to irreversible CKD/ESRD
- If due to other medication, stop if feasible
- Adequate water intake, electrolyte monitoring
- Salt and Protein restriction to decrease urine output
- NSADS: blocking certain Prostaglandins (PGE_2 interaction on AQ-2?)
- Thiazide diuretics: induce a mild volume depletion to enhance water reabsorption (non ADH dependent)

Question 7

- You are caring for a patient with bipolar disorder which is refractory to every medication except lithium. Because you remember something from your endocrine lectures, what would you consider in the patient to diminish their risk of nephrogenic diabetes insipidus?
 - a. Stop the lithium and see what happens
 - b. Furosemide
 - c. Spironolactone
 - d. Tolvaptan
 - e. Amiloride

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Question 8

- A 32 y.o. white male is referred to you by a local urologist for recurrent calcium oxalate stone formation. Which medication is an appropriate treatment for this patient?
 - a. Hydrochlorothiazide
 - b. Furosemide
 - c. Calcitonin
 - d. Allopurinol
 - e. Vitamin D₃

Question 9

- You mother has decided to get on a health improvement program and is taking some of Dr. Mercola's wonderful Vitamin D and Calcium supplements along with her daily HCTZ for HTN. She is admitted for confusion and lethargy, her serum Ca level is 13.1 mg/dL (n 8.5-10.5 mg/dL) and her bicarbonate is 32 mg/dL (normal 23-26 mg/dL). Her most likely diagnosis is?
 - a. Hypercalcemia of malignancy
 - b. Sarcoidosis causing the hypercalcemia
 - c. Acute kidney injury
 - d. Milk alkali syndrome
 - e. Hypoparathyroidism

Milk-Alkali Syndrome

- Old treatment for peptic ulcers/GERD: milk/cream and CaCO_3 (Tums)
- Elevated calcium and metabolic alkalosis
- Seen today in older patients (F>>M) taking excess Calcium supplements, Vitamin D and exacerbated by Thiazides and dehydration

Question 10

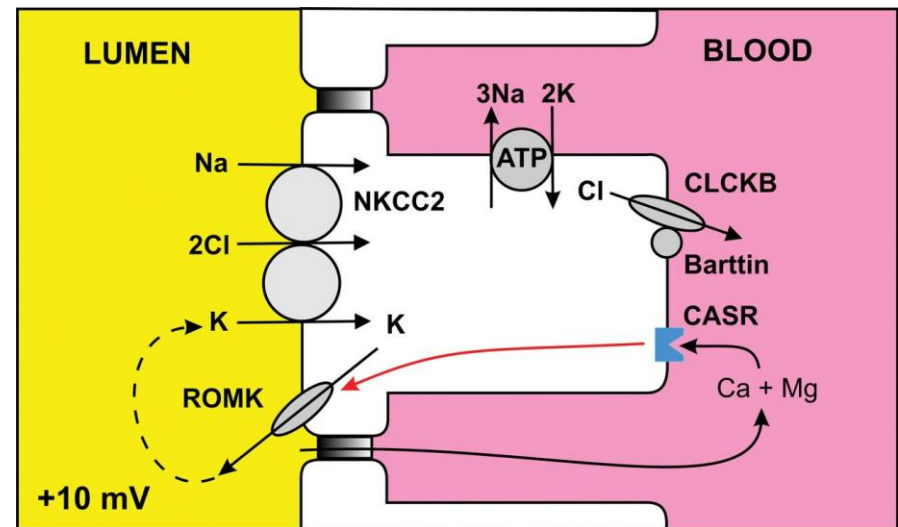
- You have a patient with severe CHF requiring high dose combination diuretics for fluid management. Despite aggressive K replacement this patient's K remains low. What is your next step in the management of this patient?
 - a. Stop all diuretics and change to a potassium sparing diuretic
 - b. Start a calcium supplement
 - c. Check the renal function
 - d. Renal ultrasound
 - e. Check a magnesium level

Role of Magnesium in Potassium balance

- In patients with refractory hypokalemia, hypomagnesaemia may be the cause.
- Causes: diuretics (thiazide and loops), diarrhea, chronic ETOH and withdrawal, malnutrition, PPI, refeeding, hungry bone, pancreatitis, diuretic phase of AKI, treatment of DKA

Role of Magnesium in Hypokalemia (TAL)

- In refractory hypokalemia hypomagnesemia is the cause
- Mg via the CaSR regulates the Renal Outer Medullary K Channel (ROMK) which secretes K into the lumen
- In Hypokalemia, Mg acts on the ROMK to decrease K secretion
- If Mg is low=K is lost despite replacement



<http://www.clinsci.org/content/112/4/203.figures-only>

Question 11

You are seeing a 56 y.o. Type 2 Diabetic, his lab is :
Glucose 260, Na 138, K 5.7, CO₂ 17, BUN 29,
Creatinine is 1.7 he is chronically non compliant with
all of his meds and diet. What is the explanation of
his lab?

- a. Occult alcohol abuse
- b. Dehydration
- c. Addison's Disease
- d. Type IV RTA
- c. Uncontrolled Diabetes

Type IV RTA

- A non anion gap metabolic acidosis
 - Hyperkalemia
 - Urine pH < 5.5
 - Frequently associated with low GFR/CKD
- + Urinary Anion Gap (UAG) thus less NH_4^+ is being produced due to a *defect* in H^+ secretion and urinary acidification

Causes of Distal Type IV RTA

- Diabetes mellitus (most common cause)
- CKD other causes
- Adrenal insufficiency/Addison's
- ACE-I/ARBs/DRIs
- Reduced distal tubule Na delivery
- Long term Heparin use (inhibits aldo synthesis)
- K sparing diuretics, spironilactone, triamptere, eplerenone, trimethoprim (Bactrim), pentamidine
- Calcineurin inhibitors, NSAIDs
- Congenital hypoaldosteronism, Type 1 and 2
Pseudohypoaldosteronism

Treatment of Type IV RTA

- Replace mineralocorticoids (may or may not help and may be contraindicated in CHF/HTN)
- Added Na to the diet
- Low K diet
- Loop diuretics

Question 12

You have a patient who is on torsemide, chlorthalidone, and potassium bicarbonate/citrate. Her potassium usually runs a bit low, 3.4 mg/dl and her magnesium is normal. She had been vomiting, but is still able to take her medications. On exam her BP is 90/60 dry mucous membranes and poor skin turgor.

Today her lab reveals: Na 136, K 2.9, Cl 96, CO₂ 33. Mg is low at 1.0.

Question 12 cont.

Optimal fluid replacement would include:

- a. Saline, potassium bicarbonate and magnesium
- b. Potassium chloride and magnesium
- c. Potassium bicarbonate, and magnesium
- d. Potassium chloride, saline and magnesium
- e. Saline and magnesium

Hypokalemia/Alkalosis Treatment

- Potassium Chloride (KCl) is the preferred replacement
- KCl will raise the K level faster than other forms
- Metabolic alkalosis is frequently seen with hypokalemia, unless Chloride is also replaced bicarbonate will continue to be produced
- Potassium bicarbonate/citrate (K-lyte) will further aggravate the alkalosis and hypokalemia

Am J Physiol 1975;229:161

Am J Med 1965;38:172

Question 13

You are seeing a patient with a history of lung cancer with metastasis, his serum Na is 109. He is only on extended release morphine and has been eating, however, but feels weak; he appears cachectic with a BP of 130/68 and no evidence of volume depletion. Your next step would be?

- a. Renal ultrasound
- b. Urine Sodium
- c. Urine Osmolarity
- d. Serum Osmolarity
- e. b, c, and d.

Question 14

The most likely etiology of the patient's hyponatremia is?

- a. Dehydration
- b. Sepsis
- c. Surreptitious use of diuretics
- d. Syndrome of Inappropriate secretion of ADH
- e. Diabetes Insipidus

Question 15

You are consulted by the Trauma Service; a 22 y.o. male was admitted for evacuation of a subdural hematoma following an accident. Despite aggressive post-op fluids, the patient had had significant polyuria, his Na is 154 with an Osmolarity of 320. The cause of the patient's Sodium abnormality is?

- a. Over secretion of ADH
- b. Over responsiveness of ADH receptors on the kidney
- c. Dehydration
- d. Under secretion of ADH
- e. Under responsiveness of ADH receptors in the kidney

Question 16

In the previous patient, the best treatment would be?

- a. Fluid restriction
- b. Demeclocycline
- c. Thiazide
- d. Amiloride
- e. Desmopressin (DDAVP)