Fluid and Electrolyte Imbalance

Presenter: Dr. Milta Little
Disclosure Statement: I have nothing to disclose.

Objectives: By the end of the session, participants will be able to…

- Describe how the body regulates water and sodium balance
- Describe the cellular mechanism for maintaining potassium balance
- List the most common causes of prerenal azotemia, dysnatremia, and potassium disorders
- List and describe the treatment strategies for fluid, sodium, and potassium imbalances

Expected Outcomes (Desired change in practice):

- Distinguish in-facility versus hospital management for fluid and electrolyte disorders
- Effectively communicate with the care team regarding fluid and electrolyte imbalances

Article for Review:

- Martinez LC, Khan SF, Bowman BT. Approach to Electrolyte Abnormalities, Prerenal Azotemia, and Fluid Balance. Prim Care Clin Office Pract 2020; 47:555-569.

Outline for Rapid Fire session

1. Case presentation: Fluid and Electrolyte Imbalance

   Mr. I. M. Balance is a 92 y/o male with advanced Alzheimer's Dementia, HTN, diastolic heart failure, glaucoma, and BPH is being seen for a follow-up visit. Two weeks ago, he was treated for aspiration pneumonia and hypoxia. He responded well to antibiotics and oxygen therapy. Labs at the time of his illness were normal, except for a white blood cell count of 14,000. Follow-up labs were obtained today and his sodium is now 127. He is alert and mentation is at baseline. Exam is significant for bibasilar crackles and trace LE pitting edema. His mucus membranes are dry. The aides report that his appetite has been down from his usual. What are your next steps in work-up and management?

2. Volume regulation
    a. Renin-Angiotensin-Aldosterone System (RAAS)
        i. Sodium reabsorption = **water follows sodium**
        ii. Antidiuretic hormone (ADH) drives water reabsorption through aquaporins in collecting ducts: tightly regulates serum osmolality to <u>280-290 mOsm/L</u>
        iii. Osmoreceptors in hypothalamus; baroreceptors in vasculature

b. Identifying prerenal azotemia
    i. History and physical – beware of the age-related changes that mimic dehydration. Check for arm pit sweat and orthostatic hypotension
    ii. Elevated BUN:creatinine ratio, typically >20:1
    iii. FE_{UN} <35% (preferred if on diuretics)
    iv. FE_{NA} <1

3. Dynatremias – **Disorders of Water Metabolism**

Normal plasma sodium 135-145, regulated by the kidneys through variable water excretion

a. Hyponatremia = <135
    i. Prevalence and complications
        1. Mild cases (125-135) is most common electrolyte abnormality in ambulatory settings
        2. Prevalence in NH residents up to 30%
        3. Cognitive impairment, gait disturbances/falls, bone fractures, mortality
    ii. Pathogenesis – distinguishing the causes
        1. First step is to consider tonicity (serum osmolality) – usually not necessary
            a. Hypertonic hyponatremia (sOsm >190) – rule out hyperglycemia. Other causes (IV mannitol, IVIG, surgical irrigants) less common
            b. Isotonic hyponatremia, "pseudohyponatremia" (sOsm 180-190) – hyperlipidemia, obstructive jaundice, plasma cell dyscrasias
            c. Hypotonic hyponatremia (sOsm <180) – everything else
        2. Baroreceptor activation in vasculature in response to decreased circulating volume stimulates ADH – sodium reabsorption – excess water reabsorption

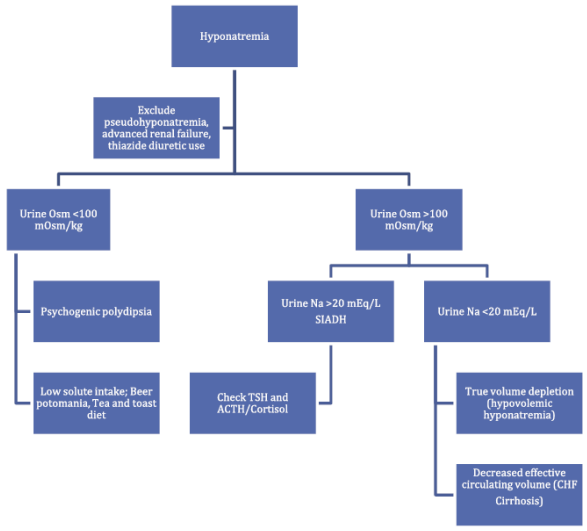

Fig. 2. Diagnostic approach to hyponatremia.

Table 2
Causes of syndrome of inappropriate antidiuretic hormone secretion

Central nervous system disorders	Infarction, hemorrhage, vasculitis Brain surgery, trauma Neoplasm Infections Guillian-Barre syndrome
Pulmonary disorders	Pneumonia • Viral • Bacterial • Tuberculous Asthma
Malignancies	Small cell carcinoma of the lung Head and neck squamous cell tumors
Endocrine disorders	Glucocorticoid deficiency Severe hypothyroidism
Drugs (common causes)	Anticonvulsants • Carbamazepine • Sodium valproate • Lamotrigine Antipsychotics and antidepressants • Haloperidol • MAOIs • SSRIs • Tricyclic antidepressants • Venlafaxine Anticancer drugs • Vinca alkaloids • Platinum compounds • Cyclophosphamide • Methotrexate Nonsteroidal antiinflammatory drugs "Ecstasy"/MDMA
Others	Nausea Pain AIDS Alcohol withdrawal

Abbreviations: MAOIs, monoamine oxidase inhibitors; MDMA, methylenedioxymethamphetamine; SSRIs, selective serotonin uptake inhibitors.

iii. Management
1. Send to hospital if acute, symptomatic, and/or severe (<120 mEq/L)
2. If total body water overload (CHF, Cirrhosis) = diurese
3. If total body water depleted (hypovolemic) = fluids
4. If euvolemic, consider SIADH and need for free water restriction.
    a. SALT TABLETS ARE NOT FIRST LINE and usually should be accompanied by a loop diuretic
    b. I recommend consulting with a nephrologist prior to using ADH inhibiting medications
5. Slow correction to avoid osmotic demyelination syndrome - the goal of initial therapy is to raise the serum sodium by 4 to 6 mEq/L in 24 hours

b. Hypernatremia
i. Pathogenesis – impaired thirst, lack of access to water, deficient ADH secretion, renal failure (lack of response to ADH)
ii. Management – slow repletion of water to goal reduction less than 12 mEq/L of Na in 24 hours (to prevent cerebral edema)

4. Potassium disorders
a. Regulation of potassium concentration
i. Predominately an intracellular cation
ii. 1-2% located in ECF, maintained in 3.5-5.1 mEq/L range
iii. Achieved by rapid intracellular shifts (extracellular acidification) and long-acting regulatory systems in kidneys and GI
b. Hyperkalemia – K > 5.0 – Arrhythmia alert!
i. Causes

Box 1
Causes of hyperkalemia

- Artifactual findings such as pseudohyperkalemia from phlebotomy or tourniquet use
- Transcellular shifts due to hyperosmolar states of cell lysis (rhabdomyolysis or tumor lysis syndrome)
- Impaired distal nephron sodium delivery as may occur in decreased effective circulating volume
- Inadequate GFR/reduced renal function to excrete a dietary potassium load
- Hypoaldosteronism and low aldosterone-like states
- Pharmacologic effects may occur from blockade of the renin-angiotensin-aldosterone system, NSAIDs, calcineurin inhibitor use, or potassium-sparing diuretics

Abbreviation: NSAIDs, nonsteroidal antiinflammatory drugs.

ii. Management
    1. Consider hospital transfer for level >6.0 mEq/L
    2. EKG necessary for levels $\geq$ 5.5 mEq/L (classic sign = peaked T waves)
    3. Rapid intracellular shift with IV insulin and inhaled beta-agonists
    4. Definitive treatment
        a. Renal excretion with loop diuretics
        b. GI sequestration with oral binding resins – patiromer and zirconium cyclosilicate are approved for chronic hyperkalemia only
    5. Hold potentially offending medications and restrict dietary potassium until corrected

c. Hypokalemia – K < 3.5 mEq/L – weakness, muscle cramps, arrhythmias, rhabdomyolysis
    i. Causes and diagnostic approach

Table 3
Causes and clinical conditions associated with hypokalemia

Causes of Hypokalemia	Clinical Conditions
Transcellular shifts; secondary to increased activity of the Na-K-ATPase pump	• Increased insulin availability (ie, refeeding syndrome) • Increased β-adrenergic activity • Thyrotoxicosis • Alkalosis • Hypokalemic periodic paralysis • Theophylline use
Gastrointestinal losses (extrarenal losses)	• Vomiting • Diarrhea
Renal losses	• Diuretic use (carbonic anhydrase inhibitors, thiazide, and loop diuretics) • Bartter/Gitelman syndromes • Primary aldosteronism • Liddle syndrome (apparent mineralocorticoid excess)

    1. 24-hour urine potassium – feasible?
    2. Urine potassium:creatinine ratio <13 mEq/g indicated GI loss, transcellular shift, or poor intake

ii. Management
    1. Always check and replete magnesium
    2. Initiate oral potassium chloride (faster than IV)
    3. In renal potassium wasting, potassium-sparing diuretic