ECHO Summary,13/SEP/2024
Session Title: Electrolyte Imbalances

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Definition

- Electrolytes are electrically charged atoms and ions when dissolved into a solution. They play a vital role in maintaining homeostasis in the body.
- They function in: osmosis (maintaining cell shape and size), enzyme function, acid-base status, and electrophysiology of excitable tissues (heart, muscle, nerves).
- Common examples include sodium (Na), potassium (K), calcium (Ca), phosphorus (P), and Magnesium (Mg).
- Pathophysiology
    - Electrolytes can be mostly intracellular or extracellular. Electrolyte imbalances, deficiency, or excess, can be due to several reasons:
        - Extracellular electrolytes e.g Na
            - Gain or loss from the body - intake, hormones, drugs, disease
            - Fluid status changes - intake, hormones, drugs, disease
        - Intracellular electrolytes e.g K
            - Cellular shifts - disease, drugs, hormones
            - Excretion - renal, gastrointestinal (GI), medications

Epidemiology

- Electrolyte imbalances are very common.
    - Hyperkalemia - up to 10% of patients admitted from the emergency department (ED)[1]

Risk Factors

Table 1. Risk factors by electrolyte derangement.*

Electrolyte	Risk Factors
Hypercalcemia[2]	● Hyperparathyroidism ● Malignancy
Hypocalcemia[3]	● Chronic kidney disease (which causes decreased vitamin D production) ● Hypoalbuminemia (due to malnourishment, alcoholism) ● Hyperphosphatemia (usually from chronic kidney disease) ● Hyperventilation (which causes respiratory alkalosis) ● Hypomagnesemia ● Hypoparathyroidism ● Massive blood transfusions (which cause iatrogenic hypocalcemia) ● Sepsis ● Severe pancreatitis (which causes calcium precipitation) ● Toxins (i.e. hydrofluoric acid) ● Tumor lysis syndrome ● Vitamin D deficiency (due to decreased dietary intake or low sunlight exposure)

Hyperkalemia[4]	● Acidosis ● Adrenal insufficiency ● Insulin deficiency ● Medications ○ Angiotensin-converting enzyme inhibitors ○ Aldosterone antagonists ○ Angiotensin II receptor blockers ○ Beta blockers ○ Blood products that have been stored ○ Digoxin ○ Direct renin inhibitors ○ Mannitol ○ Non-steroidal anti-inflammatory drugs ○ Penicillin G ○ Pentamidine ○ Potassium-sparing diuretics ○ Salt substitutes and salt alternatives ○ Succinylcholine ○ Trimethoprim ○ Verapamil ● Renal failure (most common cause) ● Renal tubular acidosis V ● Rhabdomyolysis
Hypokalemia[4]	● Alkalosis ● Burns ● Excess GI secretion ● Excessive sweating ● Excess renal secretion (most common cause in developed countries) ● Hyperaldosteronism ● Hypomagnesemia ● Poor intake ● Renal tubular acidosis I and II
Hypomagnesemia[5]	● Decreased intake ○ Alcohol abuse ○ Bariatric surgery ○ Inflammatory bowel disease ○ Malabsorption ○ Malnutrition ○ Total parenteral nutrition ● Increased losses ○ GI route: ■ Diarrhea ■ High output ostomy ■ Nasogastric tube output ■ Vomiting ○ Renal route:

	■ Acute tubular necrosis ■ Diuretics ■ End-stage renal disease ■ Intravenous fluids ■ Renal dysfunction

*Hypermagnesemia is not listed here because it is very uncommon and is usually iatrogenic in nature (i.e. magnesium sulfate administration for pre-eclampsia/eclampsia, or excessive patient ingestion of magnesium-containing antacids

Clinical features

- Features are nonsensitive and nonspecific.
- Wide range of presentations: from asymptomatic to extreme organ dysfunction, cardiac arrest and death.
- Often part of another disease.
- Signs suggestive of underlying cause: tumor, chemotherapy, hemolytic disease, rhabdomyolysis, crush injuries, burn, DM crisis, acidosis, ischemia, AKI/CKD, diet, GI bleed, transfusion.

Table 2. Unique clinical features by electrolyte.

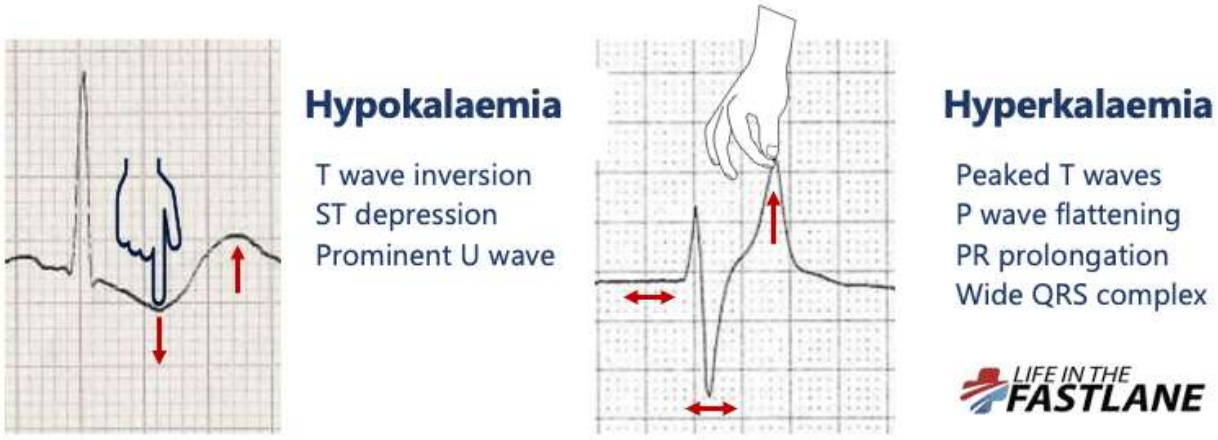
Electrolyte	Clinical Features
Hyperkalemia[4]	● Cardiac dysrhythmias ● Flaccid paralysis
Hypokalemia[4]	● General: fatigue, myalgias ● Neurological: ascending paralysis → respiratory failure, numbness, paresthesias, tetany ● GI: constipation (due to ileus), nausea, vomiting ● GU: nephrogenic diabetes insipidus, polyuria → hypovolemia
Hypercalcemia[2]	● Bones, stones, groans, and neuropsychiatric overtones ○ “Bones” (skeletal): Bone pain, arthralgia, osteoporosis ○ “Stones” (renal): Nephrolithiasis, renal failure ○ “Groans” (abdominal): Nausea/vomiting, anorexia, abdominal pain, pancreatitis ○ Psychiatric “overtones”: Lethargy, altered mental status, coma, hallucinations
Hypocalcemia[3]	● “CATs go Numb”: ○ Convulsions ○ Arrhythmias ○ Tetany (starting in hands) ○ Numbness/paresthesia (hands, feet, perioral)
Hypomagnesemia[5]	● Cardiovascular: dysrhythmias

	● GI: anorexia, fatigue, nausea, vomiting ● Neurological: neuromuscular excitability

Diagnostics

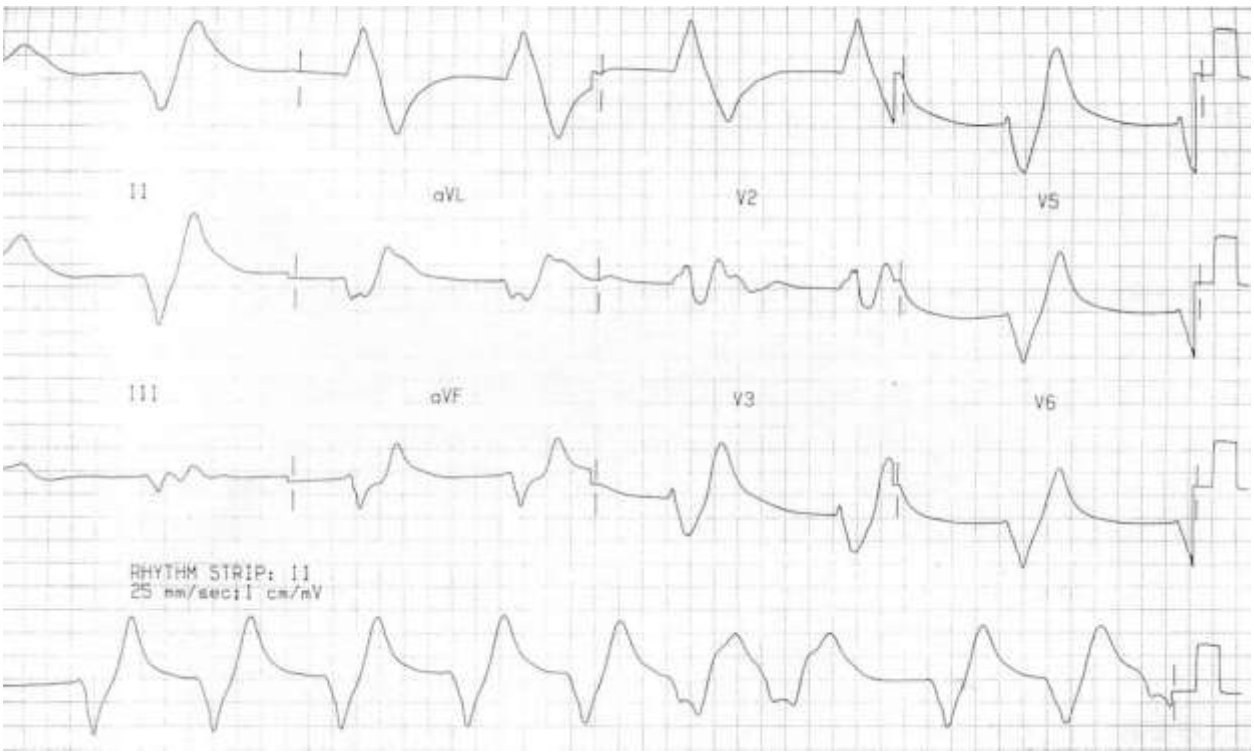
- ECG
  - Don't rely on ECG changes to make the right diagnosis, especially in chronic cases
  - Hypercalcemia - short QT, ventricular dysrhythmias, heart blocks[2]
  - Hypocalcemia - long PR, long QT[3]
  - Hyperkalemia - bradycardia, tented T waves, absent P wave, widened QRS, ST-T wave changes, sine wave, ventricular tachycardia (VT), ventricular fibrillation (VF), asystole[6]
  - Hypokalemia - long QT, U waves, ST depression, tiny T waves[4]
  - Hypomagnesemia - long QT[5]
- Labs: K, Na, Mg, Ca, complete blood count (CBC), arterial blood gas (ABG), glucose, urine electrolytes and osmolarity.
  - Consider creatine kinase (CK) in patients with suspected rhabdomyolysis.
- Targeted workup for underlying diagnosis.

Figure 1. Hypokalemia vs. hyperkalenmia ECG.



https://litfl.com/wp-content/uploads/2021/04/PushPull-K-ECG.png

Figure 2. Hyperkalemia sine wave on ECG.



https://litfl.com/hyperkalaemia-ecg-library/

Figure 3. Hypercalcemia vs. hypocalcemia ECG.

https://litfl.com/wp-content/uploads/2018/08/ECG-QT-changes-Hypocalcaemia-Hypercalcaemia-2.png

Treatment

- In the critically ill, always start with ABCDE.
    - **AIRWAY/BREATHING**: may be compromised in patients with hypokalemia/hypomagnesemia/hypocalcemia in particular. Provide supplemental oxygen/respiratory support as needed.
    - **CIRCULATION**: IV fluids, vasopressors. If in cardiac arrest, perform CPR.
    - **DISABILITY:** closely monitor the Glasgow Coma Scale (GCS), glucose.
    - **EXPOSURE**: rule out signs of concomitant trauma or burns, which can precipitate potassium derangements.
- Hyperkalemia[6]

    - Stabilize the heart: Give calcium chloride (1 g IV) or calcium gluconate (1-3 g IV) over 15 - 20 mins
        - Need central venous access to give calcium chloride - do NOT administer this through a peripheral IV! It is caustic to the veins
    - Shift potassium intracellularly:
        - Insulin 5-10 units) IV and IV dextrose (25-50 g) (to avoid hypoglycemia)
        - Salbutamol 12.5 - 25 mg (5 x standard nebulizer dose)
        - Sodium bicarbonate (if acidemic) 50 mEq (1 ampule) IV
    - Excretion:
        - Diuretics, such as furosemide in large doses (up to 240mg) to enhance excretion
            - May need to replace fluids as they diuresis
        - Dialysis
        - Resins (not helpful acutely)
            - Sodium polystyrene sulfonate is no longer recommended due to the risk of GI necrosis
            - If you have patiromer or sodium zirconium cyclosilicate, use those instead
- Hypokalemia[4]
    - Severe: Rapid IV KCl until severe symptoms resolve
        - KCl 20 mEq may be administered centrally over as little as 20 minutes
    - Mild-moderate: Give oral potassium or slow infusion
    - For the IV route, a central line is preferred.
    - Patients receiving IV KCl need cardiac monitoring
    - Give magnesium sulfate 2 g IV for any associated hypomagnesemia
    - Since potassium is mostly intracellular, there is usually a huge deficit intracellularly. We normalize the blood levels but continue oral potassium to correct the total body levels.
- Hypercalcemia[2]
    - Grading
        - Mild: 10.5-12 mg/dL (2.6-3 mmol/L)
        - Moderate: 12-14 mg/dL (3-3.5 mmol/L)
        - Severe (hypercalcemic crisis): 14-16 mg/dL (3.5-4 mmol/L)
    - Administer 0.9% normal saline IV bolus until the patient is not in shock (these patients are often volume-depleted), then move to 200-300 mL/hour to maintain a urinary output of 2 L/day
        - Will need foley catheter or condom catheter (for men) to monitor urine output closely
    - Calcitonin 4 IU/kg intramuscular (IM) or subcutaneous (SQ) twice daily (if available)
    - Bisphosphonates AFTER IV fluid repletion in patients whose hypercalcemia is thought to be due to malignancy
        - Pamidronate 60-90 mg intravenous over 2-24 hours, OR
        - Zoledronic acid 4 mg intravenous over 15 minutes

    - Speak to oncology or endocrinology as to whether corticosteroids are indicated
- Hypocalcemia[3]
    - Grading
        - Mild to moderate: corrected serum calcium level 7.6-10.4 mg/dl [2-2.1 mmol/L] or serum ionized calcium level 3.6-5 mg/dl [0.9-1.2 mmol/L]
        - Severe: corrected serum calcium level ≤7.5 mg/dl [≤1.9 mmol/L] or serum ionized calcium level ≤3.5 mg/dl [≤0.8 mmol/L]

Table 3. Calcium dosing for hypocalcemia.[3]

Grade	Calcium gluconate	Calcium chloride*
Mild	1-2 g over 2 hours	0.67 g over 2 hours
Moderate	4 g over 4 hours	1.3 g over 4 hours
Severe	1-2 g over 10 minutes; repeat every hour until symptoms resolve/calcium levels normalize	1 g over 10 minutes; repeat every hour until symptoms resolve/calcium levels normalize

*Must be given through central line.

- Hypomagnesemia[5]
    - 4-8 g over 24 hours
        - Use smaller doses/more caution in patients with renal dysfunction
        - Oral repletion is preferred over IV if possible
    - 1-4 g over 5-30 minutes for critically ill patients
        - Monitor for respiratory depression when infusing rapidly!
- Treat underlying disease.
- Continue organ support as needed.

Complications
- Cardiac or respiratory arrest
- Organ failure
- Death

Disposition
- Hyperkalemic and hypercalcemic patients should be admitted.
- Intensive care unit for patients who are critically ill.
- May discharge home patients who have mild hypokalemia, hypomagnesemia, or hypocalcemia and whose symptoms resolve with electrolyte repletion.

Special Notes
- Have a high index of suspicion for electrolyte imbalances.

- The management approach can be summarized by 5S's - Spot (Recognize), Save (Resuscitate), Sustain (Organ support), Stabilize (the underlying mechanism), Salve/palliate (underlying disease).

Collaborating Partners

1. Ministry of Health of the Republic of Uganda
2. Seed Global Health
3. Techies Without Borders

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