Potassium disorder

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Potassium hemostasis

- A normal serum potassium range is typically between 3.5 and 5.0 mEq/L. Levels below this threshold can lead to a spectrum of physiological disruptions
- Total body potassium stores amount to approximately 50 mEq/kg (3500 mEq in a 70-kg person)
- Potassium is obtained through the diet. Common potassium-rich foods include meats, beans, tomatoes, potatoes, and fruits such as bananas. Gastrointestinal (GI) absorption is complete, resulting in daily excess intake of about 1 mEq/kg (60-100 mEq).

Potassium hemostasis

- Under normal conditions, approximately 90% of potassium excretion occurs in the urine, with less than 10% excreted through sweat or stool.
- The colon is the major site of gut regulation of potassium excretion. Therefore, potassium levels can remain relatively normal under stable conditions, even with advanced renal insufficiency. However, as renal function worsens, the kidneys may not be capable of handling an acute potassium load. An excess of only 100-200 mEq will increase the serum potassium concentration by about 1 mEq/L

The Role of Potassium

- Cardiac function : Potassium is vital for normal cardiac rhythm and contractility. Deviations can lead to arrhythmias and even cardiac arrest.
- Fluid balance : Plays a role in maintaining osmotic balance and fluid distribution between intracellular and extracellular compartments
- Neuromuscular activity : It regulates nerve impulse transmission and muscle contraction, including skeletal, smooth, and cardiac muscles.
- Metabolic process : Involved in carbohydrate and protein metabolism, including glycogen and protein synthesis.

Etiology of hypokalemia

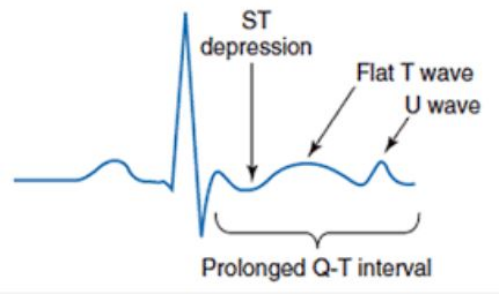
Reduced Intake	Increased Losses	Intracellular Shift
• Malnutrition • Anorexia nervosa • Prolonged IV fluid administration without potassium	• Gastrointestinal: Vomiting, diarrhea, laxative abuse • Renal: Diuretics (thiazide, loop), hyperaldosteronism, renal tubular acidosis • Sweating (excessive)	• Insulin administration (e.g., DKA treatment) • Beta-adrenergic agonists (e.g., albuterol) • Alkalosis

Causes and Risk Factors

- Renal potassium excretion is increased by the following:
- Aldosterone
- WNK1 and WNK4
- High sodium delivery to the distal tubule (eg, diuretics)
- High urine flow (eg, osmotic diuresis)
- High serum potassium level
- Delivery of negatively charged ions to the distal tubule (eg, bicarbonate)

Diagnosis

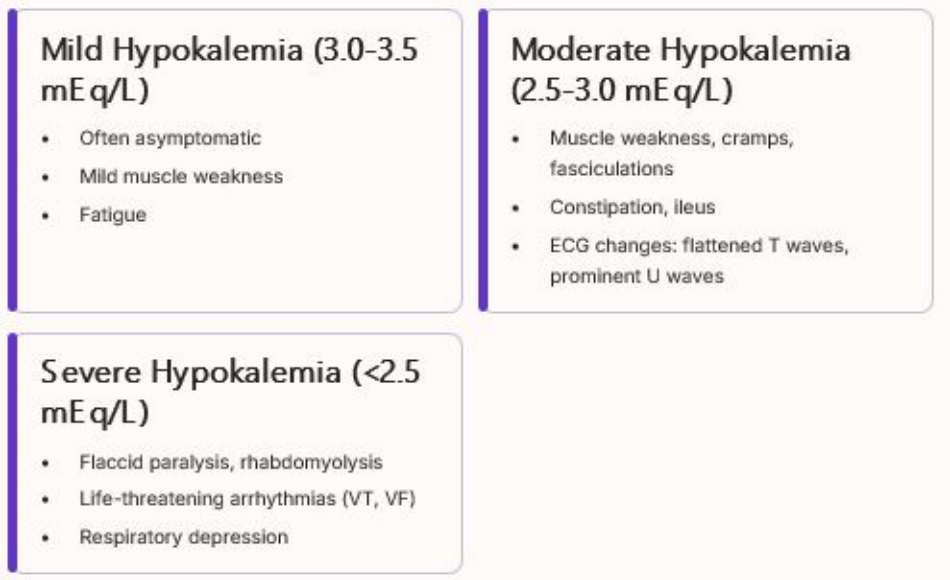
- **Electrocardiogram (ECG)**
- Characteristic ECG changes can provide immediate clues and guide urgent management in severe cases.
- Flattened T waves
- Prominent U waves
- ST segment depression
- Prolonged QT interval



Hypokalemia Overview

- More than 98% of total body K is intracellular
- Hypokalemia is one of the most common electrolyte disturbances seen in clinical practice. The condition is more prevalent than hyperkalemia
- Hypokalemia is classified according to severity. A serum potassium level of 3 to 3.4 mmol/L is classified as mild.
- A serum potassium level of 2.5 to 3 mmol/L is classified as moderate.
- Serum potassium levels less than 2.5 mmol/L are classified as severe.

Clinical Manifestations: Recognizing the Signs and Symptoms



Signs and Symptoms

- Effects of Prolonged hypokalemia on kidney structure and function:
- Impaired concentrating ability (NDI)
- Increased ammonia production
- Altered sodium reabsorption
- Increased bicarbonate absorption
- Hypokalemia may also result in **glucose intolerance** by reducing insulin secretion.

Signs and Symptoms

- **Potassium role in regulating cardiac electrical activity**:
- cardiac arrhythmias such as atrial fibrillation, and potentially life-threatening ventricular tachycardia or fibrillation.
- In severe hypokalemia cases, profound cardiac electrical activity disturbances can result in cardiac arrest and sudden death if left untreated.

Signs and Symptoms

- **Severe hypokalemia**
- paralysis, particularly in extreme cases where potassium levels are critically low.
- Respiratory failure
- Muscle cramps, rhabdomyolysis, and myoglobinuria. Rhabdomyolysis results from muscle fibers breaking down and releasing their contents into the bloodstream, leading to kidney damage and potentially life-threatening complications.
- Potassium deficiency can affect smooth muscle function in the gastrointestinal tract, leading to impaired intestinal motility and constipation.

Signs and Symptoms

- Potassium deficiency may increase Digitalis to toxic levels in the bloodstream.
- Hypokalemia can also reduce insulin receptor sensitivity, leading to insulin resistance and potentially contributing to the development of type 2 diabetes

Differential Diagnoses: Other Conditions to Consider

Hypomagnesemia Often coexists with hypokalemia and can hinder potassium repletion. Magnesium is essential for potassium transport

Hypocalcemia Can present with muscle cramps and weakness, sometimes mimicking hypokalemia.

Thyrotoxic Periodic Paralysis An acute, reversible cause of severe muscle weakness due to intracellular potassium shift, often in Asian males with hyperthyroidism.

Renal Tubular Acidosis Can lead to chronic potassium wasting, presenting with hypokalemia and metabolic acidosis.

Management Strategies

Oral Potassium Supplementation Preferred for mild to moderate hypokalemia (K > 2.5 mEq/L) without severe symptoms or ECG changes. Potassium chloride is the most common form.

Intravenous Potassium Reserved for severe hypokalemia (K < 2.5 mEq/L), significant symptoms, or inability to tolerate oral intake. Administer slowly to avoid hyperkalemia.

Address Underlying Cause

Crucial for long-term management. This might involve discontinuing diuretics, treating diarrhea, or managing endocrine disorders

Special Considerations: High-Risk Patients and Complications

- **Cardiac patients** Patients with pre-existing heart disease, especially those on digoxin, are at increased risk of arrhythmias with hypokalemia.
- **DKA** Insulin treatment can cause rapid potassium shifts into cells, exacerbating hypokalemia. Close monitoring is essential.
- **Renal impairment** Patients with kidney disease require careful potassium management due to altered excretion capacity
- **Complications** Untreated hypokalemia can lead to life-threatening cardiac arrhythmias, respiratory failure, and paralytic ileus

Treatment / Management

- The therapeutic goals for hypokalemia are to prevent or treat life-threatening complications, correct the potassium deficit, and address the underlying cause.
- Therapeutic urgency depends on the severity of hypokalemia, the existence of comorbid conditions, and the rate of decline of serum potassium levels
- Concomitant **hypomagnesemia** should also be corrected if present

Treatment / Management

- Mild-to-moderate hypokalemia may be asymptomatic. Thus, repletion is often not urgent in such cases. Mild-to-moderate hypokalemia is typically treated with oral potassium supplements.

Providing 60 to 80 mmol/day over days to weeks is usually sufficient. Oral supplementation can irritate the gastrointestinal mucosa and cause bleeding or ulceration. However, oral potassium replacement is associated with a lower risk of rebound hyperkalemia.

Increasing **dietary potassium** is usually inadequate to treat hypokalemia because most of the potassium in foods is coupled with phosphate

Most cases of hypokalemia involve chloride depletion and respond best to potassium chloride replacement. Intravenous (IV) repletion is administered if oral therapy is not tolerated.

Treatment / Management

- Replacement therapy must be provided rapidly when severe hypokalemia or clinical symptoms are present. Potassium chloride of 40 mmol given every 3 to 4 hours for 3 doses is preferred. Rapid correction is via oral intake, IV administration, or both. IV administration is preferred in the presence of cardiac dysrhythmias, digitalis toxicity, and recent or ongoing cardiac ischemia
- Generally, 20 mmol/h of potassium chloride will increase serum potassium levels by an average of 0.25 mmol/h.
- Potassium should not be repleted using dextrose-containing solutions because dextrose stimulates insulin secretion, exacerbating hypokalemia
- IV replacement should be done cautiously, as rapid potassium infusion can cause cardiac arrest

Treatment / Management

- Serum potassium levels should be checked every 2 to 4 hours. Potassium replacement can occur more slowly once the serum potassium level is persistently above 3 mmol/L or clinical symptoms have resolved. Regardless of severity, careful serum potassium level monitoring is required, as hyperkalemia commonly develops in hospitalized patients
- The potassium deficit varies directly with the severity of hypokalemia. Every 0.3 mmol/L incremental decrease accounts for a reduction of approximately 100 mmol in total body potassium stores.

Treatment / Management

- For example, a patient with a serum potassium concentration of 2.0 mEq/L may have a 400 to 800 mEq potassium deficit. Accurate quantification is difficult, especially when transcellular shifts cause hypokalemia. Therefore, careful monitoring is required to prevent hyperkalemia from excessive supplementation.
- A potassium-sparing diuretic should also be considered when hypokalemia involves iatrogenic renal potassium wasting, as potassium replacement therapy alone may not suffice.
- Rebound hyperkalemia is a potential complication of potassium therapy when cellular redistribution is the cause of hypokalemia.

Hyperkalaemia

- Hyperkalaemia is a potentially life-threatening electrolyte disturbance characterised by elevated serum potassium levels, typically above 5.5 mmol/L.
- Potassium is crucial for normal cellular function, particularly in nerve and muscle cells, including cardiac myocytes. Even slight deviations from the normal range (3.5-5.0 mmol/L) can have profound physiological consequences.
- Understanding its definition is the first step in effective diagnosis and management.

- 5.5-6.0 mEq/L – Mild
- 6.1-7.0 mEq/L – Moderate
- ≥7.0 mEq/L – Severe

Causes and Risk Factors

- Impaired Renal Excretion:
- The kidneys are the primary regulators of potassium excretion. Conditions like acute kidney injury, chronic kidney disease, and certain medications (e.g., potassium-sparing diuretics, ACE inhibitors, ARBs) can lead to reduced potassium clearance.
- Transcellular Shifts: Movement of potassium from the intracellular to extracellular fluid can elevate serum levels. This occurs in acidosis, insulin deficiency (e.g., diabetic ketoacidosis), rhabdomyolysis, and tumour lysis syndrome. Beta-blockers can also cause such shifts.

Causes and Risk Factors

Increased Potassium Intake : While less common in individuals with normal renal function, excessive dietary intake of potassium, oral potassium supplements, or intravenous potassium administration can contribute, especially in predisposed patients.

Pseudohyperkalaemia

This is an artefact where potassium is released from cells during or after blood sampling, leading to a falsely elevated result. It can occur with prolonged tourniquet application, vigorous fist clenching, or conditions like severe thrombocytosis or leukocytosis.

Causes and Risk Factors

- Renal potassium excretion is **decreased** by the following:
- Absence, or very low levels, of aldosterone
- WNK1 and WNK4 mutations
- Low sodium delivery to the distal tubule
- Low urine flow
- Low serum potassium level
- Kidney injury

Clinical Presentation

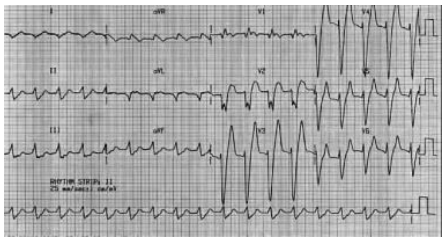
- The symptoms of hyperkalaemia can be non-specific and vary depending on the severity and rapidity of onset. Early signs are often subtle, but progression can lead to severe cardiac complications.
- **Neuromuscular:** Muscle weakness, fatigue, ascending paralysis, paraesthesias.
- **Cardiac:** Palpitations, chest pain (due to arrhythmias), bradycardia
- **Gastrointestinal:** Nausea, vomiting, abdominal cramps, diarrhea
- Always consider hyperkalaemia in patients with unexplained muscle weakness or cardiac rhythm disturbances.

Electrocardiogram (ECG) Changes

- ECG changes are crucial indicators of hyperkalaemia's severity and potential for cardiac arrest. They typically progress in a predictable sequence.
- Peaked T-waves
- Early and most common sign. Tall, tented, and narrow T-waves, especially in precordial leads
- PR prolongation
- Increased duration of the PR interval, indicating delayed atrioventricular conduction
- Loss of P-waves
- As potassium levels rise, atrial depolarisation is suppressed, leading to absent P-waves.

Electrocardiogram (ECG) Changes

- Widened QRS complex
- Progressive widening of the QRS complex, often merging with the T-wave to form a sine wave pattern
- Ventricular fibrillation/Asystole
- Terminal events leading to cardiac arrest
- Immediate intervention is required when significant ECG changes are observed



Diagnostic Work-up

- Serum Potassium Measurement
- The definitive diagnostic test. Repeat measurement to rule out pseudohyperkalaemia
- Electrocardiogram (ECG) Assess for cardiac manifestations and severity, guiding immediate management.
- Renal function Tests
- Urea, creatinine, and estimated GFR to assess kidney function and identify renal causes

Diagnostic Work-up

- Blood Gas Analysis
- To evaluate acid-base status (e.g., acidosis can cause potassium shifts)
- Other Electrolytes
- Check sodium, calcium, magnesium, and phosphate to identify co-existing abnormalities
- Medication Review
- Thorough review of current medications for potential culprits (e.g., ACE inhibitors, ARBs, spironolactone).

Acute Management

- Immediate action is crucial in severe hyperkalaemia, especially with ECG changes. The goals are to stabilise the myocardium, shift potassium intracellularly, and remove potassium from the body.
- Cardiac Stabilisation
- **Calcium Gluconate (IV):** Does not lower potassium but protects the heart from its effects. Administer immediately for ECG changes.

Acute Management

- Shift Potassium Intracellularly
- **Insulin/Dextrose (IV):** Insulin drives potassium into cells; dextrose prevents hypoglycaemia.
- **Salbutamol (Nebulised):** Beta-2 agonists also promote intracellular potassium shift.
- **Sodium Bicarbonate (IV):** For metabolic acidosis, it can shift potassium into cells

Acute Management

- Remove Potassium
- **Diuretics (e.g., Furosemide):** Increase renal potassium excretion, effective if kidneys function.
- **Cation-exchange Resins (e.g., Patiromer, Sodium Zirconium Cyclosilicate):** Oral agents that bind potassium in the gut.
- **Haemodialysis:** Most effective for rapid and definitive potassium removal, especially in renal failure.

Chronic Management and Prevention

- Addressing the Root Cause
- Long-term management focuses on identifying and treating the underlying cause of hyperkalaemia.
- **Optimise Medications:** Adjust doses or switch potassium-raising drugs if possible (e.g., ACE inhibitors, ARBs). Consider non-potassium-sparing diuretics.
- **Dietary Modification:** Educate patients on low-potassium diets. Reduce intake of high-potassium foods (bananas, oranges, potatoes, tomatoes, dark leafy greens, nuts).

Chronic Management and Prevention

- **Renal Disease Management:** Aggressive management of chronic kidney disease to preserve renal function
- **Potassium Binders:** For chronic hyperkalaemia, oral potassium binders can be used to prevent recurrence.
- **Regular Monitoring:** Frequent monitoring of serum potassium, renal function, and medication effects.