



Guidelines

For The Management Of

Hypokalemia

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**F.I.C.M.S
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& Guidelines**

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Potassium is one of the body's major ions. Nearly 98% of the body's potassium is intracellular. The kidney determines potassium homeostasis, and excess potassium is excreted in the urine. The reference range for serum potassium level is 3.5-5 mEq/L, with total body potassium stores of approximately 50 mEq/kg (i.e., approximately 3500 mEq in a 70-kg person).

Definition

Hypokalemia is defined as a potassium level less than 3.5mEq/L.

Moderate hypokalemia is a serum level of 2.5-3 mEq/L.

Severe hypokalemia is defined as a level less than 2.5 mEq/L.

Symptoms depend on degree and duration of hypokalemia.

Patients are often asymptomatic, particularly with mild hypokalemia; symptoms are often due to the underlying cause of the hypokalemia rather than the hypokalemia itself.

Symptoms generally do not become manifest until the serum potassium is below 3.0mEq/L, unless the serum potassium falls rapidly or the patient has a potentiating factor such as a predisposition to arrhythmia due to the use of digitalis

Hypokalemia should be suggested by a constellation of symptoms that involve the GI, renal, musculoskeletal, cardiac, and nervous systems.

Common symptoms include the following:

- Palpitations
- Skeletal muscle weakness or cramping
- Paralysis, paresthesia
- Constipation
- Nausea or vomiting
- Abdominal cramping
- Polyuria, nocturia, or polydipsia
- Psychosis, delirium, or hallucinations
- Depression

Physical finding that are consistent with severe hypokalemia include the following:

- Signs of ileus
- Hypotension
- Ventricular arrhythmias
- Bradycardia or tachycardia
- Premature atrial or ventricular beats
- Hypoventilation, respiratory distress Respiratory failure
- Lethargy or other mental status changes
- Decreased muscle strength, fasciculations, or tetany
- Decreased tendon reflexes

Muscle weakness usually does not occur at potassium concentrations above 2.5mEq/L if the hypokalemia develops slowly, the weakness begins with the lower extremities, progresses to the trunk and upper extremities, and can worsen to the point of paralysis

Causes

1-GI losses: vomiting or nasogastric suctioning, diarrhea, enemas or laxative use

2-Medication: diuretics (most common cause). diuretics, beta-adrenergic agonist steroids, theophylline, aminoglycosides.

3-Transcellular shift: insulin, alkalosis, hypokalemic periodic paralysis

4-Renal losses: renal tubular acidosis, hyperaldosteronism, Cushing syndrome, magnesium depletion, renal artery stenosis.

5-Malnutrition or decreased dietary intake, parenteral nutrition

6- Spurious hypokalemia may occur where unseparated blood has been stored at high, ambient temperatures i.e. during hot weather

Laboratory Studies

- Serum potassium level < 3.5 mEq/L (3.5 mEq/L)
- BUN & creatinine level
- Glucose, calcium, phosphorus, chloride & magnesium
- Thyroid screening studies (if hypokalemia periodic paralysis is suspected).
- Serum cortisol and aldosterone
- Electrocardiography
 - T-wave flattening or inverted T waves
 - Prominent U wave that appears as QT prolongation
 - ST-segment depression
 - Atrial & ventricular arrhythmias
- Consider arterial blood gas (ABG): Alkalosis can cause potassium to shift from extracellular to intracellular.
- Urine tests
 - Urinary potassium*: where low suggests poor intake, shift into the intracellular space or GI loss, high level suggests renal loss.
 - Urinary chloride*: high level indicates renal loss, low level indicates extra-renal loss
 - Urinary sodium*: low urinary sodium combined with high urinary potassium suggests secondary hyperaldosteronism.

Imaging Studies

CT scan of the adrenal & pituitary glands is indicated if hyperaldosteronism or Cushing's syndrome is suspected respectively.

Renal angiogram to exclude renal artery stenosis

Management

At a pH of 7.40, serum potassium of 3.0mEq/L approximates a 10%total body deficit & at level of 2.5mEq/L approximates a 20% total body deficit. This deficit should be replaced over 1 to 3days.

Rule of thumb in patients with normal renal function: every 10 mEq KCL given will raise the serum potassium level by 0.1mEq/dL.

The serum potassium concentration can transiently rise acutely after an oral dose by as much as 1- 1.5 mEq/L after of 40 to 60 mEq, and by 2.5 to 3.5 mEq/L after 135 to 160mEq; the serum potassium concentration will then fall back towards baseline, as most of the exogenous potassium will then be taken up by the cells so serial monitoring is indicated.

The requirement of potassium given IV &/or orally depending on patient's condition & according to IV/oral guidelines supplementation, when potassium level falls below 3.5 mEq/L, as follows:

3.4-3.5 give 60 mEq of KCL

3.2-3.3 give 80 mEq of KCL

3.0-3.1 give 100 mEq of KCL

The maximum oral single dose is **20 mEq** (average 2 of 600 mg KCl tablet).

Adult doses from 40-100 mEq/day may be required for potassium repletion given in 2 - 4 divided doses per day

Oral potassium chloride can be given in crystalline form (salt substitutes), as a liquid, or in a slow-release tablet or capsule. Salt substitutes contain 50-65 mEq per level

teaspoon; they are safe, well tolerated and much cheaper than the other preparations, and thus may be an option if cost is a concern .

In comparison, potassium chloride solutions are often unpalatable, and the slow-release preparations can in rare cases cause ulcerative or stenotic lesions in the gastrointestinal tract due to the local accumulation of high concentrations of potassium

A saline rather than a dextrose solution is recommended for initial therapy, since the administration of dextrose can lead to a transient 0.2 to 1.4 mEq/L reduction in the serum potassium particularly if only 20 mEq/L of potassium chloride is provided in the solution.

An intravenous or oral potassium chloride preparation is generally preferred over potassium citrate or potassium bicarbonate, in particular among patients with metabolic alkalosis due to diuretic therapy, vomiting, and hyperaldosteronism.

Potassium citrate or potassium bicarbonate is often preferred in patients with hypokalemia and metabolic acidosis as occurs in renal tubular acidosis and chronic diarrheal states.

Potassium phosphate can be used for patients with combined potassium and phosphate depletion (e.g. in liver cirrhosis or diabetic ketoacidosis)

Mild to moderate hypokalemia: serum potassium concentration between 3.0 - 3.5mEq/L; usually produces no symptoms, except for patients with heart disease particularly if they are taking digitalis or undergoing cardiac surgery or in patients with advanced cirrhosis. Treatment in this setting is directed toward replacing the lost potassium and toward treating the underlying disorder.

Start with 10-20 mEq of potassium chloride given two to four times per day (20-80mEq/day) depending on the severity of hypokalemia and on whether hypokalemia developed acutely or is chronic.

Sequential monitoring of the serum potassium is essential to determine continued requirements with frequency of monitoring dependent on the severity of hypokalemia.

Severe hypokalemia: Potassium must be given more rapidly to patients with more severe hypokalemia (serum potassium 2.5-3.0mEq/L) or symptomatic hypokalemia (arrhythmias, marked muscle weakness).

Intravenous therapy:

Never give bolus KCl as it can cause fatal arrhythmias.

Peripheral IV solutions should not contain more than **40 - 60 mEq of KCl/L**.

The maximum infusion rate via central line is **20mEq/hr (0.25 mEq/kg/hr)**

However, as much as **0.5 mEq/kg/hour (40 mEq/hr)** can be given to selected patients with paralysis or life-threatening arrhythmias with careful monitoring i.e. **in the intensive care unit & under senior consultation:** such concentrations are often painful, and should be infused into a large central vein, the rate of potassium repletion should be slowed to 10-20 mEq/hr once there is improvement. Careful monitoring is required both of clinical condition and blood level 1-3 hourly.

Monitoring

If using immediate release preparations (KCl powder, solution or IV dose), potassium level should be checked no sooner than 60 minutes, if a sustained release product used, potassium level should be checked no sooner than 3 hours. Monitoring continued until the plasma serum potassium concentration remains above 3.0 to 3.5mEq/L, and symptoms resolve.

The rapidity and method of potassium repletion is based upon the:

1-Severity of hypokalemia

2-The presence or absence of signs and symptoms (cardiac conduction abnormalities, muscle weakness).

3-The presence of associated conditions & whether continued losses are expected

Ongoing losses: for chronic replacement oral KCl preparations, salt substitutes sprinkled on food (containing 50-65 mEq KCl per level teaspoon) or the addition of a potassium sparing diuretic.

If a potassium-sparing diuretic is used in combination with potassium supplements, it must be used with extreme caution in patients with decreased kidney function, patients on an ACE inhibitor &/or angiotensin receptor blocker.

Monitor the serum potassium concentration approximately every three to four months in all patients receiving chronic potassium supplementation, or if clinically indicated.

Potassium-rich foods are encouraged; include dates, fruits, potatoes, bananas spinach, broccoli tomatoes, and young coconuts

Hypomagnesemia —Hypokalemia is a common event in hypomagnesemic patients, occurring in approximately one-half of cases. The hypokalemia in this setting is relatively refractory to potassium supplementation and requires correction of the magnesium deficit.

MgSO₄: 2.0 gm (25-30mg/kg) IV diluted in 50-100 ml saline or glucose given over 5-15minutes then start infusion of same dose over the next 4-8 hours at a rate of 20mg/min till serum magnesium is maintained.

Complications

1- Respiratory distress syndrome, respiratory failure serious cardiac arrhythmias

2- Contributes to the development of hepatic encephalopathy in cirrhosis

3-Hypokalemia can potentiate digitalis toxicity in patients taking digoxin

4- Rhabdomyolysis and myoglobinuria

5- Replacing potassium too quickly can cause a rapid rise in the blood potassium level, leading to relative hyperkalemia with subsequent cardiac complications

Further reading

Renal Potassium Loss

Hypertensive
High Renin

Malignant hypertension
renal artery stenosis
renin-producing tumors

Hypertensive
Low Renin, High Aldosterone

Primary hyperaldosteronism

Hypertensive
Low Renin, Low Aldosterone

Cushing's syndrome
Congenital adrenal hyperplasia
exogenous mineralcorticoids (Florinef
Licorice, chewing tobacco)

Normotensive
Metabolic acidosis

Renal tubular acidosis
(Type I or II)

Normotensive
Metabolic alkalosis

(urine chloride <10 mEq/day)
Vomiting

Normotensive
Metabolic alkalosis

(urine chloride >10 mEq/day)
Barter's syndrome, Diuretics
Hypomagnesemia

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