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A Physiologic-Based Approach to the Treatment of a Patient With Hypokalemia

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Abstract

Hypokalemia is common, and can be associated with serious adverse consequences including paralysis, ileus, cardiac arrhythmias, and death. As a result, the body maintains serum potassium concentration within very narrow limits via tightly regulated feedback and feed-forward systems. Whereas the consequences of symptomatic hypokalemia and severe potassium depletion are well appreciated, chronic mild hypokalemia can accelerate the progression of chronic kidney disease, exacerbate systemic hypertension, and increase mortality. Persistent hypokalemia may reflect total body potassium depletion or increased renal potassium clearance. In a patient with simple potassium depletion, potassium replacement therapy should correct serum potassium concentration, but may have little effect when renal potassium clearance is abnormally increased from potassium wasting. In such cases addition of potassium sparing diuretics might be helpful. Serum potassium concentration is an inaccurate marker of total body potassium deficit. Mild hypokalemia may be associated with significant total body potassium deficits and conversely, total body potassium stores can be normal in hypokalemia due to redistribution. The speed and extent of potassium replacement should be dictated by the clinical picture and guided by frequent reassessment of serum potassium concentration. The goals of therapy should be to correct any potassium deficit if present without provoking hyperkalemia. Oral replacement is preferred except when there is no functioning bowel or in the setting of EKG changes, neurological symptoms, cardiac ischemia, or digitalis therapy.

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Note from Feature Editor Jeffrey A. Kraut, MD: This article is part of a series of invited case discussions highlighting either the diagnosis or treatment of acid-base and electrolyte disorders. Advisory Board member John Harrington, MD, served as the Consulting Editor for this case. The present case discussion is the second of 2 articles discussing hypokalemia. In this article, Drs Asmar, Mohandas, Wingo present their approach to the treatment of hypokalemia; in the first teaching case, Dr Palmer describes a physiologic-based approach to its diagnosis and evaluation.¹

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INDEX WORDS

hypokalemia; treatment; replacement; potassium; Liddle syndrome

Introduction

Hypokalemia reflects either total body potassium depletion or redistribution from extracellular fluid to intracellular fluid without potassium depletion. Discerning the underlying physiologic mechanisms of hypokalemia is important to establish a diagnosis as well as to make appropriate therapeutic decisions. The goals of hypokalemia management are to prevent the development of life threatening consequences, to identify the definitive cause of hypokalemia, and to correct any potassium deficit while avoiding hyperkalemia. To illustrate these principles, we discuss our approach to a patient with chronic hypokalemia and hypertension.

Case Report

Clinical History and Initial Laboratory Data

A 41-year-old woman presented with acute onset of severe headaches and accelerated hypertension. She was admitted to an intermediate care unit and her blood pressure was decreased with intravenous labetalol. Her course was complicated by persistent hypokalemia despite potassium chloride supplementation in excess of 160 mEq/d (160 mmol/d) and poorly controlled hypertension. Physical examination was significant for a BP of 180/110 mm Hg, a prominent S4, and grade II hypertensive retinopathy on fundoscopic examination. Laboratory data included sodium 138 mEq/L (138 mmol/L); potassium 2.6 mEq/L (2.6 mmol/L); chloride 100 mEq/L (100 mmol/L); bicarbonate 30 mEq/L (30 mmol/L); serum urea nitrogen 18 mg/dL (6.4 mmol/L); creatinine 0.8 mg/dL (70.7 μ mol/L; corresponding to an estimated GFR of 79 ml/min/1.73m²[1.3 mL/s/1.73 m²] calculated using the *** equation); glucose 135.0 mg/dL (7.5 mmol/L); calcium 8.5 mg/dL (2.1 mmol/L); magnesium 2.0 mg/dL; and phosphate 2.5 mg/dL. CXR-showed borderline cardiomegaly, EKG was consistent with left ventricular hypertrophy, and urinalysis showed a specific gravity of 1.014, proteinuria (1+), and rare RBCs without casts.

Additional Investigations

A thorough history revealed the patient had been diagnosed with hypertension in her early 20s. She had been advised to take potassium supplements and briefly treated with spironolactone before it was stopped for a lack of efficacy. The patient was not taking diuretics and there was no history of licorice, exogenous glucocorticoid, or mineralocorticoid use. The patient had poor contact with other family members but did know some relatives with early onset of severe hypertension.

Urine electrolytes revealed a sodium 46 mEq/L (46 mmol/L), potassium 72 mEq/L (72 mmol/L), chloride 41 mEq/L (41 mmol/L), and creatinine 170 mg/dL (15,028 μ mol/L). Morning cortisol was 19 μ g/dl (524.2 nmol/L; reference 7.0–22 μ g/dl [193.1–607 nmol/L]), aldosterone <1.6 ng/dL (<0.04 nmol/L; reference 4–31 ng/dL [0.11–0.86 nmol/L]), and

plasma renin activity 0.10 ng/mL/hr (0.03 ng/L/s; reference 0.5–4 ng/mL/h [0.14–1.11 ng/L/s]).

Diagnosis

In this patient with chronic hypokalemia, hypertension, and suppressed plasma renin activity and serum aldosterone, a diagnosis of Liddle syndrome was considered likely.

Clinical Follow-up

The patient was started on amiloride 5 mg daily initially which was increased to 10 mg daily. On follow up, the blood pressure improved to 135/85 mmHg, serum potassium concentration was 4.0 mEq/L (4.0 mmol/L), and serum bicarbonate was 25 mEq/L (25 mmol/L). She was weaned off of all other antihypertensive agents.

Discussion

Because the approach to diagnosis of hypokalemia has been discussed in a previous teaching case,¹ our discussion will be limited to treatment. Our patient had hypokalemia that was chronic in nature with no associated symptoms or signs. She had been treated with potassium supplements without success. With addition of the potassium sparing diuretic amiloride, hypokalemia resolved. Symptoms and signs of hypokalemia (Box 1) can be subtle and easily overlooked without a carefully directed history and physical examination, and careful interpretation of the electrocardiogram.

The management of hypokalemia should take into consideration the following: 1) the amount of potassium necessary 2) the potassium preparation and route of administration, and finally, 3) the rate of potassium repletion.

A critical issue in replacing potassium losses is estimating the amount of potassium supplements required. Serum potassium concentration is maintained within a narrow range (3.5–5.0 mEq/L) by feedback and feedforward systems². A feedback mechanism responds to changes in potassium concentration, and is mediated in part by aldosterone. Recent evidence suggests that factors other than aldosterone such as progesterone³ and tissue kallikrein⁴ may also play a role in renal potassium homeostasis. The body also possesses acute adaptive mechanisms as evidenced by the brisker kaliuresis that occurs with either gastrointestinal or portal intravenous potassium administration than with the same amount of potassium administered intravenously⁵. This rapidly adaptive system responds to an anticipated increase in serum potassium concentration and involves a kaliuretic reflex initiated by receptors in the gut, portal vein or liver, independent of aldosterone⁶. These dual mechanisms normally correct serum potassium concentration to acute and chronic challenges to body potassium balance^{2,7}.

The total body potassium for a normal 70 kg adult is ~3500 mEq, 98% of which is intracellular. Serum potassium concentration represents only a small fraction of the total body potassium (~2%) and is an imprecise estimate of total body potassium stores or potassium deficit. With simple potassium depletion, it has been conservatively estimated every 0.3 mEq/L decrease in serum potassium concentration corresponds to > 100 mEq

deficit in total body potassium. Serum potassium concentrations of <3 mEq/L and <2 mEq/L may reflect, respectively, total body deficits of >200 mEq and of >800 – 1000 mEq⁸. In such cases of simple potassium depletion, serum potassium concentration will increase to normal when body stores are replete. The estimated potassium deficit will vary with body weight and does not account for ongoing potassium losses or nitrogen balance. Moreover, as potassium depletion develops, certain tissues (particularly skeletal muscle) show signs of rapid depletion of intracellular potassium that serves to stabilize serum potassium concentration. Thus, with early potassium depletion even slight lowering of serum potassium concentration can be associated with substantial total body potassium depletion.

True potassium depletion can be caused by renal or extrarenal losses. Urinary potassium excretion rate is infrequently measured in clinical practice, but is the most practical method to determine body potassium balance and should be obtained when the cause for hypokalemia is not readily apparent. Our patient's urinary potassium excretion rate was high in the setting of hypokalemia, which indicates kaliuresis or an impaired sensing of the patient's potassium status which increases renal potassium clearance. Failure to correct serum potassium concentration despite very large doses of potassium (>200 mEq/day) without extra-renal sources of potassium loss, as was the case in our patient, suggests either an underlying "potassium-wasting nephropathy" or an abnormal "set point" for serum potassium concentration regulation. Measurement of total dietary potassium intake may be necessary to distinguish these two conditions. In the former the patient exhibits negative potassium balance unless potassium intake is abnormally large, whereas in the latter the patient is able to remain in potassium balance and conserve potassium, despite hypokalemia, on a low potassium diet. In the original report of Gitelman syndrome, the authors compared potassium intake and potassium excretion, and, surprisingly, this patient was capable of potassium conservation albeit at a markedly reduced serum potassium concentration⁹. An altered potassium set point can be inferred if potassium excretion decreases with a stepwise decrease in dietary potassium intake. Catecholamines, aldosterone, insulin, and certain drugs stimulate potassium redistribution into the intracellular compartment and can provoke frank hypokalemia in the absence of total body depletion¹⁰. In patients where these hormonal mechanisms are believed to be transient, overzealous potassium replacement might cause rebound hyperkalemia. The combination of low plasma renin activity and aldosterone levels along with hypertension and hypokalemia narrows the differential diagnosis to Liddle syndrome, Cushing syndrome, and syndrome of apparent mineralocorticoid excess. Our patient's earlier unsuccessful trial of spironolactone and normal serum cortisol argue against the latter two diagnoses.

Repletion of body potassium stores can be done with potassium chloride, potassium phosphate, or potassium bicarbonate¹⁰. Potassium chloride is generally preferred, especially if there is accompanying chloride responsive metabolic alkalosis. Potassium chloride administration results in the distal nephron chloride reabsorption in exchange for bicarbonate secretion, which corrects the metabolic alkalosis. Since metabolic alkalosis enhances renal potassium clearance, correction of the metabolic alkalosis with potassium chloride further serves to restore serum potassium concentration to normal. Thus, the correction of serum potassium concentration is generally faster with potassium chloride than with other salts. Potassium chloride is available as a liquid, a slow-release tablet, or capsule.

Slow-release preparations are generally better tolerated than the liquid form, but can still be associated with gastrointestinal ulceration¹¹ and should be taken with food and ample fluid. In patients with coexisting metabolic acidosis, potassium bicarbonate or potassium citrate is preferred. Potassium phosphate therapy is recommended in patients with concomitant phosphorous deficiency, which occurs with diabetic ketoacidosis or Fanconi syndrome. Hypokalemia can promote metabolic alkalosis by stimulating ammonia genesis¹² and renal adenosine triphosphatase hydrogen/potassium pump activity¹³. On the other hand, hypokalemia inhibits aldosterone production¹⁴ which reduces renal bicarbonate absorption and favors metabolic acidosis. These complex effects probably explain why potassium depletion in humans produces little change in net acid-base balance unless aldosterone levels are primarily or secondarily elevated.

Signs and symptoms should be used in conjunction with serum potassium concentration level to guide the rate of potassium repletion. Although the dangers of severe hypokalemia are well recognized, mild hypokalemia often goes untreated and can also have serious consequences. Chronic hypokalemia results in hypokalemic nephropathy, a tubulointerstitial disease that is characterized by nephrogenic diabetes insipidus, alkalosis, and progressive GFR loss^{15,16}. Hypokalemia has also been associated with worsening hypertension¹⁶ and increased mortality in patients with heart disease, chronic kidney disease (CKD)¹⁷, and cerebrovascular disease¹⁸. This point is particularly relevant in CKD where physicians restrict dietary potassium due to concerns about the risks of hyperkalemia. Such indiscriminate restriction of potassium in earlier stages of CKD can have deleterious effects on blood pressure and progression of CKD.

Certain instances warrant more urgent correction of serum potassium concentration, including cardiac arrhythmias, muscle weakness, or ECG changes of hypokalemia. Hypokalemia predisposes to cardiac arrhythmias by several mechanisms including increased cardiac automaticity, slowed conduction, and delayed ventricular repolarization¹⁹, predominantly in patients with ischemic heart disease or on digitalis. Following acute myocardial infarction, risk of arrhythmias is increased with serum potassium concentration less than 3.9 mEq/L²⁰. Severe hypokalemia can also cause skeletal muscle weakness including complete paralysis. Diaphragmatic muscle paralysis, though rare, can lead to respiratory arrest. In these cases, potassium chloride 5 to 10 mEq over the span of 20–30 minutes can be administered to increase serum potassium concentration level >3.0 mEq/L¹⁶, resulting in clinical improvement. Serum potassium concentration should be checked frequently after repletion of 40–60 mEq potassium chloride.

Prophylactic measures should be entertained for patients on diuretics who are at risk of hypokalemia, given that almost half develop serum potassium concentration less than 3.5 mEq/L²¹. Strategies to prevent hypokalemia include adhering to a low salt diet and concomitant use of beta blockers, ACE inhibitors, or potassium sparing diuretics reduces the risk of diuretic-induced hypokalemia. Dietary counseling may help to prevent potassium deficiency and address protein and caloric needs.

Dietary intake may not be sufficient for acutely replacing potassium losses associated with chloride depletion secondary to diuretic treatment, vomiting or nasogastric suction. If

potassium supplementation is needed, 20 mEq/d of potassium chloride orally is a reasonable starting dose. Alternatively a potassium-sparing diuretic could be used. Magnesium deficiency can exacerbate potassium wasting making it refractory to correction with potassium replacement alone²². Serum magnesium should be checked and appropriately replaced in patients with unexplained hypokalemia, refractory hypokalemia, and diuretic-induced hypokalemia.

Potassium may be replaced through oral or intravenous route. The risk of rebound hyperkalemia is lowest with the oral route, possibly due to the kaliuretic reflexes arising from putative potassium receptors in the gut. The oral route is preferred unless the patient is unable to take oral medicine or medical urgency necessitates IV potassium delivery. Generally, 20 mEq/h of potassium chloride will increase serum potassium concentration by an average of 0.25 mEq/h, but this rate can be associated with ~2% incidence of mild hyperkalemia²³. Thus, these approximations are not a substitute for frequent monitoring of serum potassium concentration. In non-emergent situations requiring intravenous potassium replacement, 20–40 mEq of potassium can be added to each liter of glucose-free solution. Glucose may predispose to arrhythmias or neuromuscular paralysis by stimulating insulin release and potassium shift into cells²⁴. Infusions of potassium concentration greater than 40 mEq/L require central venous access, and are irritating, painful, and can cause venous sclerosis. In general, potassium chloride replacement rates should not exceed 20mEq/hour and cardiac monitoring is recommended at such high rates. For the patient with a GFR of < 30 mL/min/1.73 m² (<0.5 mL/s/1.73 m²), these rates should be reduced by 80–50% with frequent (2–4h) re-assessment of serum potassium concentration. A suggested management outline is summarized in Box 2. Close monitoring of the serum potassium concentration and EKG are crucial to reduce the risk of inadvertent hyperkalemia during replacement therapy. This risk is more pronounced in patients with decreased kidney function and patients with reduced adrenal and pancreatic beta cell function.

In summary, most causes of hypokalemia will be evident from a careful history, physical examination, and initial laboratory data. For most cases, the treatment consists of administration of potassium supplements, but in the rare patient with intact renal potassium conservation but an altered "set point", potassium sparing diuretics are preferred. It is not known whether such individuals have the same risk from "hypokalemia" that is present in the general population. Key teaching points are listed in Box 3, and general principles of management in Figure 1.

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Box 1. Clinical manifestations of hypokalemia

- **Cardiovascular System**

ECG changes: prominent U wave, flattened or inverted T waves, ST segment depression, T and U wave fusion giving appearance of QT interval prolongation with severe hypokalemia

Arrhythmias: atrial tachycardia with or without block, premature ventricular contraction, ventricular tachycardia and/or fibrillation, torsades de pointes

Worsening hypertension

Sudden death

- **Kidney**

Polyuria due to decreased concentrating ability

Hypokalemic nephropathy

Chloride-depletion metabolic alkalosis

Increased risk of nephrolithiasis

- **Neuromuscular**

cramp, myalgia, rhabdomyolysis, weakness, paralysis, paresthesia

- **Gastrointestinal tract**

Altered gastrointestinal motility (nausea, vomiting, constipation, paralytic ileus)

Worsening of hepatic encephalopathy

- **Genitourinary tract**

Hypotonic bladder

- **Respiratory System**

Respiratory acidosis secondary to respiratory muscle weakness

- **Endocrine System**

insulin resistance and impairment in insulin release

Box 2. Suggested Management for Hypokalemia

- Mild to moderate hypokalemia (serum potassium concentration 3.0–3.5 mEq/L)
 - If possible, treat the underlying disorder with 60–80 mEq/d of potassium chloride orally in divided doses
 - Recheck serum potassium concentration after replacement therapy
- Severe hypokalemia (serum potassium concentration < 3.0 mEq/L)
 - Preferred: potassium chloride 40 mEq orally every 3–4 hours × 3
 - If necessary: IV potassium chloride (10–20 mEq/hr) in the setting of cardiac arrhythmias, digitalis toxicity, and recent or ongoing cardiac ischemia. This should be done with continuous cardiac monitoring.
 - Recheck serum potassium concentration every 2–4 hr to ensure that serum potassium concentration is > 3.5 mEq/L

Box 3. Key Teaching Points

- 1-** Serum potassium concentration does not accurately reflect total body potassium deficits. Even mild hypokalemia may be associated with significant deficits and require correction over several days, especially in the setting of ongoing losses.
- 2-** Chronic hypokalemia, even mild, can be associated with progressive kidney disease and increased mortality, and should be evaluated and treated.
- 3-** The oral route is the preferred mode of potassium replacement therapy in hypokalemia. Intravenous therapy should be reserved for those with malfunctioning gut, neurological symptoms, cardiac arrhythmias, digitalis toxicity, and recent or ongoing cardiac ischemia.
- 4-** The preferred salt for replacement therapy of hypokalemia is potassium chloride as most causes of hypokalemia are accompanied by metabolic alkalosis and require chloride repletion.
- 5-** Though serum potassium concentration and symptoms might be dramatic in hypokalemia from cellular shifts, total body stores are normal and careful monitoring during potassium replacement is necessary to prevent hyperkalemia.
- 6-** Many patients with potassium depletion, especially those on loop diuretics, may also have magnesium deficiency, which will lead to refractory hypokalemia unless identified and treated early.
- 7-** A careful history, physical, and systematic approach can identify the etiology of most hypokalemia, including obscure ones.

Figure 1.

General Principles of Hypokalemia Management. These steps should be helpful in most cases of hypokalemia; however, clinical judgment should be exercised when applying it to individual patients. Serum potassium levels must be checked no sooner than one hour after an IV dose is given (2 hours after an oral dose). Parenteral potassium should be avoided except in urgent conditions listed and transitioned over to oral preparations as soon as possible. Serum potassium levels should be carefully monitored especially in patients with kidney or cardiac disease.

Abbreviations: IV, intravenous; KCl, potassium chloride. Hypokalemia