Chapter 36

POTASSIUM DISORDERS

The kidneys are the principal regulator of potassium balance, adjusting excretion based on intake. Factors affecting renal potassium excretion include aldosterone, acid-base status, serum potassium concentration, and renal function. The intracellular potassium concentration is approximately 30 times the extracellular potassium concentration. A variety of conditions alter the distribution of potassium between the intracellular and extracellular compartments, potentially causing either hypokalemia or hyperkalemia. The plasma concentration does not always reflect the total body potassium content.

HYPOKALEMIA

Decision-Making Algorithm
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Hypokalemia

Etiology

Hypokalemia is common in children, with most cases related to gastroenteritis. Spurious hypokalemia occurs in patients with leukemia and elevated white blood cell counts if plasma for analysis is left at room temperature, permitting the white blood cells to take up potassium from the plasma. There are four basic mechanisms of hypokalemia (Table 36-1). Low intake, nonrenal losses, and renal losses all are associated with total body potassium depletion. With a **transcellular shift**, total body potassium is normal unless there is concomitant potassium depletion secondary to other factors.

The transcellular shift of potassium after initiation of insulin therapy in children with diabetic ketoacidosis (see Chapter 171) can be dramatic. These patients have reduced total body potassium because of urinary losses, but they often have a normal serum potassium level before insulin therapy from a transcellular shift into the extracellular space secondary to insulin deficiency and metabolic acidosis. Children receiving aggressive doses of β-adrenergic agonists (albuterol) for asthma can have hypokalemia resulting from the intracellular movement of potassium. Poor intake is an unusual cause of hypokalemia, unless also associated with significant weight loss (anorexia nervosa).

Diarrhea has a high concentration of potassium, and the resulting hypokalemia usually is associated with a metabolic acidosis secondary to stool losses of bicarbonate. With emesis or nasogastric suction, there is gastric loss of potassium, but this is fairly minimal given the low potassium content of gastric fluid (approximately 10 mEq/L). More important is the gastric loss of hydrochloride, leading to a metabolic alkalosis and a state of volume depletion. Metabolic alkalosis and volume depletion increase urinary losses of potassium.

Urinary potassium wasting may be accompanied by a metabolic acidosis (proximal or distal renal tubular acidosis) (see Chapter 37). Loop and thiazide diuretics lead to hypokalemia and a metabolic alkalosis. **Bartter syndrome** and **Gitelman syndrome** are autosomal recessive disorders resulting from defects in tubular transporters. Both disorders are associated with hypokalemia and a metabolic alkalosis. Bartter syndrome is usually associated with hypercalciuria, often with nephrocalcinosis; children with Gitelman syndrome have low urinary calcium losses, but hypomagnesemia secondary to urinary losses.

In the presence of a high aldosterone level, there is urinary loss of potassium, hypokalemia, and a metabolic alkalosis. There also is renal retention of sodium, leading to hypertension. A variety of genetic and acquired disorders can cause high aldosterone levels. **Liddle syndrome**, an autosomal dominant disorder caused by constitutively active sodium channels, has the same clinical features as hyperaldosteronism, but the serum aldosterone level is low.

Clinical Manifestations

The heart and skeletal muscle are especially vulnerable to hypokalemia. **Electrocardiographic (ECG) changes** include a flattened T wave, a depressed ST segment, and the appearance of a U wave, which is located between the T wave (if still visible) and P wave. Ventricular fibrillation and torsades de pointes may occur, although usually only in the context of underlying heart disease. Hypokalemia makes the heart especially susceptible to digitalis-induced arrhythmias.

The clinical consequences in skeletal muscle include muscle weakness and cramps. **Paralysis** is a possible complication (generally only at potassium levels <2.5 mEq/L). Paralysis usually starts with the legs, followed by the arms. Respiratory paralysis may require mechanical ventilation.

Some hypokalemic patients develop **rhabdomyolysis**, especially following exercise. Hypokalemia slows gastrointestinal motility; potassium levels less than 2.5 mEq/L may cause an ileus. Hypokalemia impairs bladder function, potentially leading to urinary retention. Hypokalemia causes polyuria by producing secondary nephrogenic diabetes insipidus. Chronic hypokalemia may cause kidney damage, including interstitial nephritis and renal cysts. In children, chronic hypokalemia, as in Bartter syndrome, leads to poor growth.

Diagnosis

It is important to review the child's diet, history of gastrointestinal losses, and medications. Emesis and diuretic use can be surreptitious. The presence of hypertension suggests excess mineralocorticoids. Concomitant electrolyte abnormalities are useful clues. The combination of hypokalemia and metabolic acidosis is characteristic of diarrhea, distal renal tubular acidosis, and proximal renal tubular acidosis. A concurrent metabolic alkalosis is characteristic of gastric losses, aldosterone excess, diuretics, Bartter syndrome, or Gitelman syndrome.

Treatment

Factors that influence the therapy of hypokalemia include the potassium level, clinical symptoms, renal function, presence of transcellular shifts of potassium, ongoing losses, and the patient's ability to tolerate oral potassium. Severe, symptomatic hypokalemia requires aggressive treatment. Supplementation