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Hyperkalemia

Author: Michael J Verive, MD, Medical Director, Pediatric Intensive Care, Department of Pediatrics, St Mary's Hospital for Women and Children
Contributor Information and Disclosures

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Introduction

Background

Hyperkalemia is defined as a serum potassium concentration greater than the upper limit of the normal range; the range in children and infants is age-dependent, whereas the range for adults is approximately 3.5-5.5 mEq/L. The upper limit may be considerably high in young or premature infants, as high as 6.5 mEq/L[1]. Because hyperkalemia can cause lethal cardiac arrhythmia, it is one of the most serious electrolyte disturbances.

Pathophysiology

Potassium is the primary intracellular cation; more than 95-98% of the total body potassium is found in the intracellular space, primarily in muscle. Normal homeostatic mechanisms serve to precisely maintain the serum potassium level within a narrow range. The primary mechanisms for maintaining this balance are the buffering of extracellular potassium against a large intracellular potassium pool (via the sodium-potassium pump) and urinary excretion of potassium.

Under normal, nonpathologic conditions, approximately 90% of potassium excretion occurs in the urine, with less than 10% of potassium excreted through sweat or stool. Within the kidneys, potassium excretion occurs mostly in the principal cells of the cortical collecting duct (CCD). Urinary potassium excretion depends on adequate luminal sodium delivery to the distal convoluted tubule (DCT) and CCD, as well as the effect of aldosterone and other adrenal corticosteroids with mineralocorticoid activity.

Laboratory hyperkalemia (fictitious or pseudohyperkalemia) can easily occur because of hemolysis, tissue lysis, and "milking" of extremities (which can introduce a significant amount of interstitial fluid into the blood sample) during phlebotomy, especially with heel-poke and finger-stick phlebotomy, which are commonly performed in infants and small children. Hemolysis can also be caused by fist clenching during phlebotomy or during prolonged tourniquet application, which can also lead to an acidotic sample with resultant hyperkalemia). Blood sampled "upstream" of an intravenous line with potassium-containing fluid (or from a multiple lumen central venous catheter where the sampling lumen is near the lumen containing potassium-rich infusate) can have falsely elevated levels of potassium that do not reflect circulating levels.

Thrombocytosis can also lead to false elevations of serum potassium levels. The normal serum potassium level is 0.4 mEq/L higher than the plasma level because of potassium release during clot formation. For every 100,000/mL elevation in the platelet count, the serum potassium increases by approximately 0.15 mEq/L. This can easily be corrected based on a measurement of whole blood potassium level. A similar effect on serum but not plasma potassium can also be seen with leukocytosis.

True hyperkalemia is caused by one of 3 basic mechanisms, although the root cause for any individual patient is often multifactorial.

- Increased K^+ intake: Increased K^+ intake is most commonly caused by intravenous or oral potassium supplementation. Packed RBCs (PRBCs) also carry potentially high concentrations of potassium that can lead to hyperkalemia during PRBC transfusion.[2]
- Transcellular K^+ shifts: In a transcellular potassium shift, a hydrogen ion enters a cell and leads to decreased K^+ uptake by the cell in order to maintain electrical neutrality. Acidosis is the most common cause of hyperkalemia due to transcellular potassium shift, but any process that leads to cellular injury or death (eg, tumor lysis syndrome, rhabdomyolysis, crush injury, massive hemolysis) can cause hyperkalemia, as intracellular potassium is released by disruption of the cell membrane. Other causes of hyperkalemia due to transcellular shift of potassium include propofol ("propofol infusion syndrome"),[3] toxins (digitalis intoxication or fluoride intoxication), succinylcholine, beta-adrenergic blockade, strenuous or prolonged exercise, insulin deficiency, malignant hyperthermia, and hyperkalemic periodic paralysis.
- Decreased K^+ excretion: The most common cause of decreased potassium excretion leading to hyperkalemia is oliguric renal failure. Other causes include primary adrenal disease (eg, Addison disease, salt-wasting forms of congenital adrenal hyperplasia), hyporeninemic hypoaldosteronism, renal tubular disease

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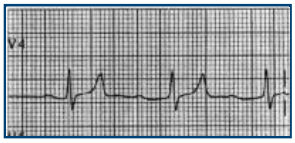
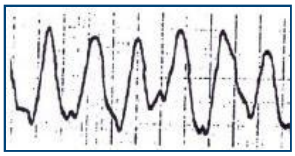
(pseudohypoaldosteronism I[4] or II), or medications (eg, ACE inhibitors, angiotensin II blockers, spironolactone or other potassium-sparing diuretics).

Plasma potassium levels are generally maintained at 3.5-5 mEq/L in adults. Levels greater than 7 mEq/L can lead to significant hemodynamic and neurologic consequences. Levels exceeding 8.5 mEq/L can cause respiratory paralysis or cardiac arrest and can quickly be fatal. High levels of potassium cause abnormal heart and skeletal muscle function by lowering cell-resting action potential and preventing repolarization, leading to muscle paralysis. Classic ECG findings begin with tenting of the T wave (as is shown in the image below), followed by lengthening and eventual disappearance of the P wave and widening of the QRS complex.[5]



Peaked T waves.

Just before the heart stops, the QRS and T wave merge to form a sinusoidal wave (as is shown in the image below).


Sinusoidal wave.

Select Factors Affecting Plasma Potassium

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Factor	Effect on Plasma K^+	Mechanism
Aldosterone	Decrease	Increases sodium resorption, and increases K^+ excretion
Insulin	Decrease	Stimulates K^+ entry into cells by increasing sodium efflux (energy-dependent process)
Beta-adrenergic agents	Decrease	Increases skeletal muscle uptake of K^+
Alpha-adrenergic agents	Increase	Impairs cellular K^+ uptake
Acidosis (decreased pH)	Increase	Impairs cellular K^+ uptake
Alkalosis (increased pH)	Decrease	Enhances cellular K^+ uptake
Cell damage	Increase	Intracellular K^+ release
Succinylcholine	Increase	Cell membrane depolarization

Frequency

United States

Hyperkalemia is a manifestation of a disease and is not a disease by itself. The incidence of hyperkalemia in the pediatric population is unknown, although the prevalence of hyperkalemia in extremely low birth weight premature infants can exceed 50%.[6] Hyperkalemia in pediatric patients is most commonly associated with renal insufficiency, acidosis, and with diseases that involve defects in mineralocorticoid, aldosterone, and insulin function.[7]

Mortality / Morbidity

Sudden and rapid onset of hyperkalemia can be fatal. With slow or chronic increase in potassium levels, adaptation occurs via renal excretion, with fractional potassium excretion increasing by as much as 5-10 times the reference range.

Race

No racial predilection is observed.

Sex

No sex-related predilection is observed. However, neuromuscular disorders including myotonic and muscular dystrophies and related disorders that can predispose patients to hyperkalemia with succinylcholine administration are more prevalent in males.[8]

Age

Extremely low birth weight premature infants are particularly prone to hyperkalemia primarily due to immature renal function. Even otherwise full-term infants may have transient hyperkalemia and hyponatremia due to decreased

responsiveness to aldosterone (pseudohypoaldosteronism I).[7]

Clinical

History

History for a previously well child with acute hyperkalemia should focus on how the blood sample was obtained, potassium intake or recent blood product transfusion, risk factors for transcellular shift of potassium (acidosis) or tissue death/necrosis, medication use (by the child, other family members, pets, etc) associated with hyperkalemia, and presence or signs of renal insufficiency.

Specific questions may be focused on the following:

- Urine output (last void or number of wet diapers) and fluid intake
- Cola-colored urine (which may indicate acute glomerulonephritis)
- Bloody stool (which may indicate hemolytic-uremic syndrome [HUS])
- Presence of drugs in the household (or used by recent visitors), such as potassium preparations, digoxin, and diuretics
- Any history of trauma (crush injuries) or thermal injury (burns)

Medical history, family history, and review of systems should be explored for any of the following:

- Acute or chronic renal failure
- Hypertension
- Diabetes
- Adrenogenital syndromes
- Malignancy (tumor lysis syndrome)

Family history (hyperkalemic periodic paralysis, miscarriages, deaths of very young siblings)

- Neuromuscular disorders
- Malignant hyperthermia

Physical

High potassium levels interfere with repolarization of the cellular membrane following completion of the action potential. Findings depend on the degree of hyperkalemia and primarily relate to the deleterious effects of elevated plasma potassium levels on cardiac conduction. Children with hyperkalemia can present with cardiac arrest due to wide-complex tachycardia or ventricular fibrillation.

Symptoms short of circulatory collapse/cardiac arrest include respiratory failure and weakness that progresses to paralysis. Patients may report nausea, vomiting, and paresthesias (eg, tingling). Most often, patients with hyperkalemia are asymptomatic, with the first clinical manifestation of the condition either ECG changes (peaked T waves) or sudden cardiac arrest.

Nonspecific findings can include muscle weakness (skeletal, respiratory), fatigue, ileus with hypoactive or absent bowel sounds, and depression.

Causes

Although the etiology of hyperkalemia can be multifactorial, differential diagnoses include fictitious hyperkalemia and hyperkalemia due to increased potassium intake, transcellular potassium shift, or decreased potassium excretion.

- Fictitious hyperkalemia
  - Hemolysis, tissue lysis, or tissue ischemia during phlebotomy
  - Contamination of blood sample with potassium-containing fluids
  - Thrombocytosis or leukocytosis (affects serum K^+ but not plasma K^+)
- Hyperkalemia due to increased K^+ intake
  - Blood transfusion (increasing risk with increased duration of cell storage)
  - Intravenous (IV) or oral potassium
  - Maintenance K+ in IV or oral solutions combined with decreased renal function
- Hyperkalemia due to transcellular K^+ shift
  - Metabolic acidosis
  - Acute tubular necrosis
  - Electrical burns
  - Thermal burns
  - Cell depolarization
  - Head trauma
  - Rhabdomyolysis

- Digitalis toxicity
- Fluoride toxicity[9]
- Propofol infusion syndrome
- Tumor lysis syndrome
- Succinylcholine use in a child with neuromuscular disease, prolonged bed rest (including patients in ICUs), or more than 24 hours after crush or burn injury[10]

- Hyperkalemia due to decreased K^+ excretion
  - Acute renal failure
  - Primary adrenal disease (Addison disease, salt-wasting congenital adrenal hyperplasia)
  - Hyporeninemic hypoaldosteronism
  - Renal tubular disease

- Medications (eg, potassium sparing diuretics, ACE inhibitors, angiotensin II blockers, trimethoprim, nonsteroidal anti-inflammatory agents [NSAIDs])

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