HYPERKALEMIA

DEFINITION:

- Serum potassium level greater than 5.0 mEq/L.
- Pseudohyperkalemia: Occurs with marked leukocytosis or thrombocytosis and from hemolysis of a blood specimen; indicated by a serum potassium concentration (clotted) 0.2-0.3 mEq/L greater than a plasma potassium concentration (unclotted).

INCIDENCE IN CRITICAL ILLNESS: Less common than hypokalemia.

ETIOLOGY:

- Excessive potassium administration.
- Impaired potassium excretion: Renal failure; mineralocorticoid deficiency (Addison's disease, type 4 renal tubular acidosis, heparin-induced inhibition of aldosterone synthesis, hereditary enzyme deficiencies); pseudohypoaldosteronism (chronic kidney failure in diabetes mellitus and tubulointerstitial disease); medications (potassium-sparing diuretics, ACE inhibitors, ARBs, NSAIDs, trimethaphan, cyclosporine, tacrolimus, pentamidine, trimethoprim, azole antifungals, fluoride, herbal supplements, penicillin G potassium); ureterojejunostomy (increased jejunal resorption of urinary potassium).
- Shift of potassium out of cells: Hypertonicity; tissue destruction (rhabdomyolysis, burns, trauma); cellular destruction (tumor lysis, acute intravascular hemolysis); medications (beta-adrenergic blockers, digoxin, succinylcholine, lysine, arginine); familial hyperkalemic periodic paralysis; insulin deficiency or resistance.
- Traditionally, it was thought that an inverse relationship between serum pH and serum potassium level existed. However, this has been disproven; the relationship is complex and incompletely understood.

CLINICAL MANIFESTATIONS:

- Mild hyperkalemia is usually asymptomatic.
- Cardiovascular: Characteristic ECG changes (peaked T waves, wide QRS complex, AV conduction blocks, sine waves, ventricular fibrillation, asystole).
- **Neuromuscular:** Paresthesias; weakness of the extremities, symmetrical flaccid paralysis, ascending to the trunk and respiratory muscles (cranial nerve sparing).

TREATMENT:

- Emergent treatment: Indicated for a serum potassium level > 6.5 mEq/L or ECG changes consistent with hyperkalemia.
- Renal excretion of potassium: Plasma volume expansion + furosemide.
- Direct antagonism of the hyperkalemic effect on cell membrane polarization (cardiac cell membrane stabilization): Calcium chloride.
- Movement of extracellular potassium into the intracellular compartment: Regular insulin + glucose; albuterol; sodium bicarbonate (controversial); aminophylline.
- Gastrointestinal excretion of potassium: Sodium polystyrene sulfonate.
- Extracorporeal removal of potassium: Intermittent hemodialysis or continuous renal replacement therapy.

KEY REFERENCES:

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- Adrogue HJ, Madias NE. Changes in plasma potassium concentration during acute acid-base disturbances. Am J Med 1981;71:456-467.

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