HYPERKALEMIA

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IMMEDIATE CONSIDERATIONS

FINDINGS

- Signs & Symptoms
 - ECG changes
 - o Arrhythmias
 - Severe muscle weakness
 - Often asymptomatic

• Diagnostic Findings

- ECG changes
 - Early changes include:
 - Tall peaked T waves in multiple leads
 - Shortened QT interval
 - More severe changes include:
 - Progressive lengthening of PR interval and QRS duration
 - Disappearance of P wave
 - QRS widening to sine wave pattern

• Predisposing Conditions

- Reduced urinary excretion of potassium
 - Can be:
 - Secondary acute or chronic renal failure
 - Reduced aldosterone secretion
 - Aldosterone resistance

- Reduced distal sodium and water delivery
- Increased production of potassium:
 - Tissue catabolism
 - Necrosis
 - Medications
 - Beta-blockers
 - ACE inhibitors
 - Digoxin
 - Potassium-sparing diuretics
 - Metabolic acidosis
 - Exercise
 - Insulin deficiency
 - Hyperkalemic periodic paralysis
 - Massive red blood cell transfusion with hemolysis

• Differential Diagnoses

- Pseudohyperkalemia
 - Commonly due to hemolysis of red blood cells during or after specimen collection
 - Repeat the test if hyperkalemia does not fit the clinical picture
 - Elevation in measured serum potassium is due to potassium movement out of the cells during or after the specimen is drawn (hemolysis)

DIAGNOSTIC INTERVENTIONS

- First priorities
 - o Immediate ECG to assess for dangerous manifestations of hyperkalemia
 - Any ECG changes should prompt immediate empiric treatment

- o Repeat electrolyte panel to confirm hyperkalemia if it does not fit the overall clinical picture
 - May be pseudohyperkalemia

• Second priorities

- Perform additional testing to assess for cause of renal failure, including:
 - Blood urea nitrogen
 - Creatinine
 - Serum electrolytes
 - Sodium
 - Bicarbonate
 - Chloride
 - Calcium
 - pH
 - Glucose levels
 - Creatinine kinase
- \circ Also consider adding urine microscopy and urine electrolytes to assist in the differential

diagnosis of renal failure

• Consider renal ultrasound with doppler to evaluation for renal perfusion abnormalities

• Ongoing priorities

- Continuous cardiac monitoring
- o Serial serum potassium measurements until the level normalizes
- Monitor urine output

THERAPEUTIC INTERVENTIONS

- Medications
 - In presence of ECG abnormalities and/or cardiac arrhythmias:

- Intravenous calcium
 - Antagonizes membrane action of hyperkalemia to prevent lethal cardiac arrhythmias
 - Calcium gluconate or calcium chloride can be used, but gluconate is safer
 - The temporizing effect of calcium therapy is very short-lasting and **does not correct the problem**
- Intravenous insulin and dextrose, inhaled beta-2 adrenergic agonists, and intravenous sodium bicarbonate will temporarily shift potassium into the intracellular space
 - These therapies also do not correct the problem but temporize until potassium removal
- Diuretic therapy and hemodialysis
 - In the case of severe oligoanuric renal failure
 - Will actually remove potassium from the body
- Cation exchange resins may aid in removing potassium from the body via the gastrointestinal tract, but this approach is unpredictable
 - Ex. sodium polystyrene sulfonate
- Identify and treat reversible underlying causes for hyperkalemia
- Stop any potassium supplementation or associated medications

MANAGEMENT AFTER STABILIZATION

- Follow-Up
 - Follow potassium levels every 4-6 hours until stable and/or underlying cause is reversed
- Persistent hyperkalemia despite diuresis may suggest tissue necrosis

CAUTIONS

- Lethal cardiac arrhythmia can result from delayed empiric intervention
 - These arrhythmias can occur unpredictably with relatively mild hyperkalemia
- Cation exhange resins have been implicated in intestinal necrosis

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