HYPERKALEMIA

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IMMEDIATE CONSIDERATIONS

FINDINGS

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

• Diagnostic Findings
  o 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
  o Reduced urinary excretion of potassium
    ▪ Can be:
      • Secondary acute or chronic renal failure
      • Reduced aldosterone secretion
      • Aldosterone resistance
• Reduced distal sodium and water delivery

  o 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
  o 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
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

- 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
- 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 exchange resins have been implicated in intestinal necrosis
REFERENCES & ACKNOWLEDGEMENT


