

# Hyperkalemia

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PGY -1

# Definition

- Hyperkalaemia is defined as a potassium level  $> 5.5$  mEq/L
- Moderate hyperkalaemia is a serum potassium  $> 6.0$  mEq/L
- Severe hyperkalaemia is a serum potassium  $> 7.0$  mEq/L

# Causes

- **Impaired renal excretion** ([acute renal failure](#), [chronic kidney disease](#), [diabetic nephropathy](#), [systemic lupus erythematosus](#))
- **Renal tubular acidosis type IV** (hypoaldosteronism or reduced release or action of aldosterone)<sup>(6)</sup>
  - normal anion gap hyperchloremic acidosis
  - occurs in setting of hypoaldosteronism or pseudohypoaldosteronism
  - Causes: transient early childhood hyperkalemia, mineralocorticoid def (Addison's disease) medications associated with RTA IV: **NSAIDs, ACE inhibitors**, K<sup>+</sup> sparing diuretics, heparin
- **K<sup>+</sup> redistribution or release into extracellular space**
  - hyperosmolar states e.g hyperglycemia
  - volume depletion (hypertonicity)
  - Acidosis
  - familial hyperkalemic periodic paralysis
  - Rhabdomyolysis, tumor lysis syndrome
  - lab error (for example, lysis during sample collection, red blood cell destruction)
- **Exogenous K<sup>+</sup>**
  - [potassium supplements](#), total parenteral nutrition – **Short guts!**
  - red blood cell transfusion
  - **Other meds:** Co-trimoxazole (Bactrim!), [penicillin G potassium](#), Tacrolimus/cyclosporine, azole antifungals, beta blockers

# H&P: History

- **CC: If present**, signs and symptoms may include muscular weakness, flaccid paralysis, cardiac arrhythmia, decreased bowel motility (ileus)
- **HPI** for a previously well child with acute hyperK<sup>+</sup> should focus on how the blood sample was obtained, K<sup>+</sup> intake or recent blood transfusion, risk factors for transcellular shift of K<sup>+</sup> (acidosis) or tissue death/necrosis, medication use
- Specific questions may include:
  - Urine output/color
  - Cola-colored urine (which may indicate acute glomerulonephritis)
  - Bloody stool (which may indicate [hemolytic-uremic syndrome](#) [HUS])
  - Drugs
  - Any history of trauma (crush injuries) or thermal injury (burns)
- **PMHx:**
  - renal impairment, hypoaldosteronism
  - diabetes mellitus ([type 1](#) or [type 2](#))
  - adrenal insufficiency
  - [systemic lupus erythematosus](#)
  - familial hyperkalemic periodic paralysis

# H&P: Physical

- often no clinical signs suggestive of hyperkalemia
- Cardiac: arrhythmia (ventricular ectopy, bradycardia, asystole)<sup>(</sup>
- Abdomen: decreased bowel sounds (ileus)
- Neuro: muscular weakness, flaccid paralysis

# Initial management

- Confirmation – recheck lab
- While waiting - EKG and cardiac monitor
- For **severe hyperkalemia** (K<sup>+</sup> level > 7.0 mEq/L) **or** lower K<sup>+</sup> level and ECG changes other than peaked T waves:
  - Stabilize myocardium : Slow infusion of calcium gluconate 10 % solution, dose of 0.5 mL/kg (maximum dose 20 mL [2 g]).
  - Therapy to shift extracellular potassium into cells including:
    - IV administration of regular insulin and glucose → regular insulin (dose of 0.1 units/ kg, maximum dose of 10 units) along with dextrose of 0.5 g/kg over 30 minutes.
    - And/or inhaled beta-adrenergic agonists, such as albuterol (salbutamol)
- Sodium bicarbonate - also causes transcellular K<sup>+</sup> movement, but its beneficial effect is uncertain. Not used as sole intervention.
- After initial management – search to correct causes

# Chronic/persistent or hyperkalemia (5.5 to 6.5 mEq/L)

- Diuretics – loop/thiazide
- Enteral cation exchange resins
  - sodium polystyrene sulfonate (Kayexalate) the most common given at 1 g/kg every four hours (maximum dose 30 g). It is dissolved in water and is given enterally or rectally

# EKG findings

- $K^+ > 5.5$  mEq/L is associated with **repolarization abnormalities**:
  - Peaked T waves (usually the earliest sign of hyperkalaemia)
- $K^+ > 6.5$  mEq/L is associated with **progressive paralysis of the atria**:
  - P wave widens and flattens , PR segment lengthens , P waves eventually disappear
- $K^+ > 7.0$  mEq/L is associated with **conduction abnormalities** and **bradycardia**:
  - Prolonged QRS interval with bizarre QRS morphology
  - High-grade AV block with slow junctional and ventricular escape rhythms
  - Any kind of conduction block (bundle branch blocks, fascicular blocks)
  - Sinus bradycardia or slow AF
- $K^+ > 9.0$  mEq/L causes **cardiac arrest** due to:
  - Asystole , Ventricular fibrillation, PEA with bizarre, wide complex rhythm



# Case #1

- A 22mo old boy with PMHx NEC, subsequent SBS and TPN dependence presents with acute onset vomiting that started this morning. Mom says he usually tolerates well his bolus feeds but has been vomiting with every feed. Denies fever, cough/congestion. However, she does report he has seemed a little weak.
- PE: Afebrile, HR is 160 o/w stable, he is a little tired appearing, not moving a lot, abd exam is remarkable for very weak bowel sounds. You decide to proceed with fluid resuscitation and screening labs.
- LABS come back with K<sup>+</sup> level 7.1, creatinine is at his baseline, normal anion gap, slightly elevated phos at 6.1, o/w unremarkable BMP.

What is your initial course of action?

- A. Recheck K+, order EKG, cardiac monitor
- B. Recheck K+, order EKG, give kayexalate rectally
- C. Order EKG, cardiac monitor, give IV calcium gluconate, IV insulin and glucose
- D. Order EKG, cardiac monitor, give kayexalate rectally

Repeat lab results confirm your initial result. Mom reports that she lost her job, and couldn't afford it to drive to get the labs checked last week. What is your next course of action?

- A) Continue TPN, IV calcium gluconate, IV insulin and glucose
- B) Continue TPN and give kayexalate
- C) Hold TPN, give IV calcium gluconate, IV insulin and glucose
- D) Hold TPN and give kayexalate

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# Case #2

3 yo male is brought to the ED by his mother when she noted bloody diarrhea earlier in the day. There is no fever, ill contacts, or recent exposures to children with diarrhea. He is noted to be pale. His family had attended a birthday party 7 days prior where the child had consumed hot dogs and hamburgers. He has maintained good urine output.

- PE: Afebrile, P 150, R 28, BP 100/45, O2 sat 100% in RA. His mucosa is moist and his tongue is pale. His neck is supple without adenopathy. His heart has a regular rhythm with tachycardia and a grade III/VI systolic ejection murmur. Lungs are CTAB. Abd is soft, NT with the liver edge palpable 2cm below the RCM. The spleen is non-palpable. His genitalia and anus are normal (no rectal prolapse). His pulses and perfusion are good. There are is no edema, rash, or petechiae.
- LABS: CBC: WBC 16,000 with 56% segs, 12% bands, 27% lymphs, 3% eos, 2% basos, hemoglobin 8 mg/dL, hematocrit 24.6, platelet count 75,000; peripheral smear shows schistocytes. BMP: Na 133, K 6.2, Cl 96, bicarbonate 16, BUN 45, creatinine 1.3, glucose 145 mg/dL, Ca 7.8, PO4 7.1, uric acid 7.3, and LDH 300. Coagulation studies are normal.

What is the most likely etiology of his hyperkalemia?

- A. Volume depletion
- B. Acute kidney injury
- C. Incidental medication ingestion
- D. Lab error

What EKG finding are you most likely going to find?

- A. Normal EKG
- B. Peaked T waves
- C. Prolonged QRS with bizarre QRS morphology
- D. Sinus bradycardia

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# References

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Up to date - Management of Hyperkalemia in Children

[https://sg.jhsmiami.org/contents/,DanaInfo=www.uptodate.com+management-of-hyperkalemia-in-children?source=search\\_result&search=hyperkalemia+children&selectedTitle=1%7E150](https://sg.jhsmiami.org/contents/,DanaInfo=www.uptodate.com+management-of-hyperkalemia-in-children?source=search_result&search=hyperkalemia+children&selectedTitle=1%7E150)

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