emDocs

The EM Educator Series

The EM Educator Series: Ins & Outs of Hyperkalemia

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Case 1: A 69-year-old male is sent to the ED from the internal medicine clinic. He was being evaluated today for recurrent syncope. His potassium was 6.7 mEq/L.

Case 2: A 53-year-old female presents with repeated vomiting and weakness. She is currently being treated for a urinary tract infection with TMP/SMX and takes an ACE inhibitor for hypertension.

Case 3: A 84-year-old male presents with decreased urine output, weakness, and mental status changes. He has a history of heart failure and chronic kidney disease.

Questions for Learners:

1. What are the various etiologies of hyperkalemia? (Note: review the med list!)
2. What are the clinical presentations of hyperkalemia?
3. How sensitive are EKG changes? / Which are the most concerning?
4. What are essential components of ED management, and where do we go wrong?
5. Who needs emergent hemodialysis?

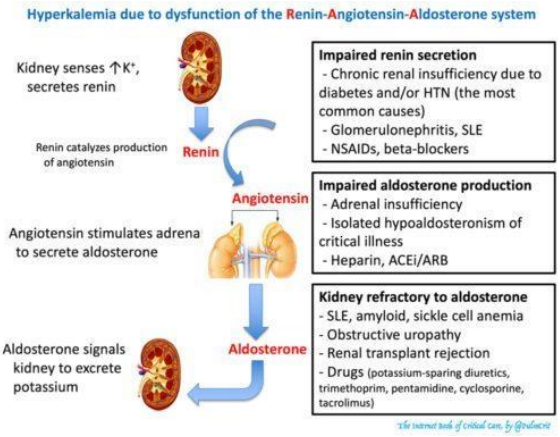
Suggested Resources:

- Articles
  - emDOCs Cases HyperK
  - IBCC HyperK
  - Emergency Medicine Cases
  - EMCrit Critical HyperK
  - First10EM HyperK
  - REBEL EM ECG Changes
  - ECG Pointers
  - emDocs Insulin Dosing
  - PulmCrit LR vs. NS in HyperK
  - PulmCrit Zirconium Cyclosilicate
- Journal Articles
  - JEM - HyperK Controversies
  - WJEM - Predicting outcomes with ECG
  - Emergency Medicine Practice - HyperK

Answers for Learners:

1. What are the various etiologies of hyperkalemia? (Note: review the med list!)

- Pseudohyperkalemia
  - Hemolysis
  - Severe leukocytosis/thrombocytosis
  - Delayed sample processing
- Iatrogenic
  - Potassium supplements
  - ACEi / ARB, aliskiren (renin-inhibitor)
  - NSAIDs
  - Beta-blockers (mostly nonselective agents, e.g. labetalol)
  - Potassium-sparing diuretics (amiloride, triamterene, spironolactone, eplerenone)
  - Antibiotics (trimethoprim, pentamidine, ketoconazole, IV penicillin[2])
  - Heparin
  - PRBC transfusion
  - Cyclosporine, tacrolimus
  - Digoxin toxicity, succinylcholine
- Diabetic ketoacidosis, hyperglycemic hyperosmolar non-ketotic syndrome (HHNS)
- Cellular lysis
  - Hemolysis, hematoma
  - Rhabdomyolysis
  - Tumor lysis syndrome
  - Tissue necrosis of other etiologies (e.g. trauma, infarction)
- Renal failure, primarily if there is:
  - Oliguria
  - GFR <15 ml/min
- Dysfunction of the renin-angiotensin-aldosterone system (Type IV renal tubular acidosis):



2. What are the clinical presentations of hyperkalemia?

Since potassium plays an important role in Na-K-ATPase physiology, hyperkalemia can result in several important effects, primarily cardiac and neuromuscular. However, clinical features are nonspecific and include general weakness, lethargy, or confusion. Deep tendon reflexes may be depressed or absent, though cranial nerves, diaphragm function, and sensation are typically normal. GI effects include nausea, vomiting, and diarrhea.

3. How sensitive are EKG changes? / Which are the most concerning?

Classic teaching of the chronological ECG changes of hyperkalemia include:

1. Peaked T waves
2. Prolongation of PR interval
3. Widening QRS Complex
4. Loss of P wave
5. "Sine Wave"
6. Asystole

How good are ECGs at predicting hyperkalemia? [3]

- 87 patients admitted from the ED with hyperkalemia
- ECG read by 2 EM physicians blinded to potassium level
- Sensitivity: 34 – 43%
- Specificity: 85 – 86%
- When only patients with potassium > 6.5 mmol/L were analyzed: Sensitivity 55 – 62%
- Conclusion: Based on this study the ECG has a poor correlation with detection of hyperkalemia

Neither the ECG nor the plasma potassium alone are an adequate index of the severity of hyperkalemia, and providers should have a low threshold to initiate cardiac membrane stabilization in the setting of hyperkalemia and no ECG changes.

4. What are essential components of ED management, and where do we go wrong?

Effect	Agent	Dose	Onset	Duration
Membrane Stabilization	Calcium Gluconate (10%)	10mL IV over 10 min	Immediate	30 – 60 minutes
	Hypertonic (3%) Normal Saline	50mL IV push	Immediate	Unknown
Shifters	Insulin (Short Acting)	10 units IV push with 25 – 40 g dextrose (50% solution)	20 minute	4 – 6 hours
	Albuterol	10 – 20 mg in 4 mL of Normal Saline nebulized over 10 minutes	30 minute	2 hours
Excreters	Furosemide	40 – 80 mg IV x1	15 minute	2 -3 hours
	Sodium Bicarbonate	150mmol/L IV at variable rate	Hours	Duration of Infusion
	Sodium Polystyrene Sulfonate	15 – 30 g in 15 – 30 mL (70% sorbitol orally)	> 2 hours	4 – 6 hours
Definitive	Hemodialysis	-----	Immediate	3 hours

- **1 g Calcium Chloride (CaCl) = 3 g Calcium Gluconate:** Bryan Hayes (@PharmERToxGuy) had a great post on Academic Life in EM on myths associated with different calcium regimens.

- **Hypertonic (3%) Saline:** Should be restricted to patients with hyponatremia and concurrent hyperkalemia. Its effects have not been established in eunatremic patients.
- **Insulin:** 10 Units of regular insulin + dextrose (25 g as 50% solution) lowers plasma potassium by about 0.6 mmol/L. Be careful as a single bolus of 25 g of dextrose has been shown to be inadequate ion prevention of hypoglycemia at 60 minutes.
- **Beta Agonists:** 10 mg and 20 mg of inhaled albuterol decreased serum potassium by 0.6 mmol/L and 1 mmol/L, respectively. Mild tachycardia is most common side effect.
- **Bicarbonate:** Bolus injection of sodium bicarbonate has not been studied. The dogma of bolus bicarbonate was derived from studies using a prolonged (4 – 6 hr) infusion of bicarbonate which does not reduce plasma potassium in patients with dialysis-dependent renal failure. Infusion sodium bicarbonate over 4 – 6 hours may have some benefit in excretion of serum potassium in the setting of metabolic acidosis.
- **Exchange Resin:** The onset of action is very long (2 hours to onset, and 6 ours to maximum effect) and there have been case reports of patients developing colonic necrosis. Also there have been studies that show no statistical benefit at 4, 8, and 12 hours [6].
- **Hemodialysis:** Definitive treatment of hyperkalemia. Watch out for rebound hyperkalemia which can occur in 30% of patients an hour after dialysis.

5. Who needs emergent hemodialysis?

- This is the most efficacious way to remove potassium, with potassium decreasing by 1 mmol/L at 1 hour and 2 mmol/L by 2-3 hours. Lower potassium dialysate and increased blood flow can result in faster serum potassium decreases.
- Increases in machine blood pump speed and plasma-to-dialysate concentration can decrease potassium within minutes; thus the importance of dialysis (especially in cardiac arrest).
- Rebound hyperkalemia may be seen after dialysis, likely related to predialysis potassium levels.
- Hemodialysis should be considered early in patient with diagnosed renal failure, inability to produce urine, hyperkalemia resistant to other treatment, cardiac arrest, and marked tissue destruction.