

TREATMENT OF HYPERKALEMIA

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Objectives

- ⦿ Explain hyperkalemia and the signs and symptoms
- ⦿ Identify the causes of hyperkalemia
- ⦿ Review the treatment of hyperkalemia
- ⦿ Discuss a patient who presented to the hospital with hyperkalemia

Background

- ⊙ Normal plasma potassium concentration: 3.5-5 mEq/L
- ⊙ Primary intracellular ion
- ⊙ Potassium homeostasis modulated by:
 - Short-term (internal balance): insulin, catecholamines, and acid-base balance
 - Long-term (external balance): kidneys and intestines




Causes

- ⊙ Increased intake
- ⊙ Shift of potassium from intracellular to extracellular:
 - Acidosis
 - Insulin deficiency (i.e. DKA)
 - Beta adrenergic blockade
 - Digoxin overdose
 - Rewarming after hypothermia
 - Succinylcholine
- ⊙ Reduced urinary excretion:
 - Kidney dysfunction
 - Intravascular volume depletion
 - Hypoaldosteronism
 - Medications

Signs and Symptoms

- ◎ Cardiac effects:
 - EKG changes:
 - Peaked T wave
 - Widening of the QRS complex
 - Loss of the P wave
 - “Sine wave” configuration
 - Ventricular fibrillation
 - Asystole
 - Enhanced by a variety of factors
 - EKG changes may be absent and should not delay treatment of severe hyperkalemia

Signs and Symptoms

| Serum potassium | Typical ECG appearance | Possible ECG abnormalities |
|--------------------------|---|--|
| Mild (5.5-6.5 mEq/L) |  | Peaked T waves Prolonged PR segment |
| Moderate (6.5-8.0 mEq/L) |  | Loss of P wave Prolonged QRS complex ST-segment elevation Ectopic beats and escape rhythms |
| Severe (>8.0 mEq/L) |  | Progressive widening of QRS complex Sine wave Ventricular fibrillation Asystole Axis deviations Bundle branch blocks Fascicular blocks |

Signs and Symptoms

- ⊙ Neuromuscular effects:
 - Paraesthesias
 - Weakness progressing to flaccid paralysis
 - Deep tendon reflexes are depressed or absent

- ⊙ Metabolic effects:
 - Mild hyperchloremic metabolic acidosis

Treatment

- ⊙ Given emergently in the following situations:
 - Plasma potassium > 6.5 mEq/L

 - EKG manifestations of hyperkalemia regardless of potassium value

Treatment

- ◎ Goals of therapy (in order of importance):
 1. Antagonize the effect of potassium on excitable cell membranes
 - Calcium
 2. Redistribute extracellular potassium into cells
 - Insulin
 - Beta agonists
 - Bicarbonate
 3. Enhance elimination of potassium from the body
 - Bicarbonate
 - Diuretics
 - Exchange resin (Kayexalate®/sodium polystyrene sulfonate)
 - Dialysis

Treatment

- ◎ **B**-beta agonist and bicarbonate
- ◎ **I**- insulin (regular)
- ◎ **G**- gluconate (calcium)
- ◎ **K**- Kayexalate[®]
- ◎ Then there is also dialysis and diuretics

Treatment: Calcium

- ⊙ Given for symptomatic hyperkalemia to stabilize the myocardium preventing abnormal arrhythmias
- ⊙ Calcium gluconate preferred over chloride
 - Chloride salt is 3x more concentrated
 - Chloride salt can cause tissue necrosis
- ⊙ Dose: calcium gluconate 1 g IV over 2-10 minutes; repeat in 5 minutes if no improvement in EKG
- ⊙ Onset: immediate
- ⊙ Duration: 30-60 minutes

Treatment: Insulin

- ⊙ Given to redistribute potassium intracellularly
- ⊙ Dose: 10 units IV of regular insulin in combination with 25 g of dextrose 50% in normoglycemic patients
- ⊙ Onset: < 15 minutes
- ⊙ Duration: 4 to 6 hours
- ⊙ Typically lowers potassium by 0.5-1.5 mEq/L

Treatment: Beta Agonists

- ⊙ Given to redistribute potassium intracellularly
- ⊙ Dose: Albuterol 10-20 mg nebulized over 10 minutes
- ⊙ Onset: 30-90 minutes
- ⊙ Duration: 2 hours
- ⊙ Typically lowers potassium by 0.5-1.5 mEq/L
- ⊙ Resistance:
 - Patients taking non-selective beta blockers
 - As many as 40% of patients not taking beta blockers are non-responders

Treatment: Bicarbonate

- ⊙ Given to redistribute potassium intracellularly vs enhanced renal elimination
- ⊙ Dose: Sodium bicarbonate 50 mEq over 5 minutes or as a continuous infusion
- ⊙ No effect on decreasing potassium should be anticipated
- ⊙ Efficacy is controversial

Treatment: Kayexalate®

- ⦿ Cation-exchange resin that exchanges sodium for potassium, resulting in intestinal excretion of potassium
- ⦿ Dose: 15-30 g PO/PR Q6H prn
- ⦿ Onset: > 2 hours
- ⦿ Duration: 4-6 hours
- ⦿ Efficacy: highly variable and unpredictable
- ⦿ Not for emergent treatment of hyperkalemia

Treatment: Kayexalate®

- ⦿ Constipation is a side effect of the resin complex
- ⦿ Sorbitol component is used as a cathartic
- ⦿ 70% sorbitol mixture no longer recommend due to intestinal necrosis
- ⦿ Caution use in those with kidney or heart failure due to the sodium component of the medication

Treatment: Dialysis

- ◎ Hemodialysis (HD):
 - Method of choice for removal of potassium from the body
 - Expected decrease of potassium:
 - ~1 mEq/L in the first hour
 - ~2 mEq/L after 3 hours
 - Rebound of potassium:
 - 35% of reduction abolished after 1 hour
 - 70% of reduction abolished after 6 hours
 - Magnitude of post-HD rebound is proportional to pre-HD potassium value
- ◎ Peritoneal dialysis (PD):
 - Rate of removal is much slower when compared to HD
 - Decrease of potassium is due mainly to the glucose load of the PD solution pushing the potassium back intracellularly

Treatment: Diuretics

- ◎ Enhanced renal elimination of potassium
- ◎ Ineffective in those with advanced kidney disease
- ◎ Loop diuretics:
 - Furosemide or bumetanide:
 - Onset: 15 minutes
 - Duration: 2-3 hours

Patient Case

- ⊙ 95 YO F admitted with AKI, hyperkalemia, and volume depletion
- ⊙ PMH: recurrent UTIs, h/o of DVT, HTN, HLD, hypothyroidism, GERD, bradycardia s/p pacemaker, dementia, h/o PNA
- ⊙ SH: negative for EtOH and tobacco and lives at Alexian Villiage
- ⊙ Home meds: Norvasc, Cipro (completed), Celexa, Clonidine, Depakote, Aricept, Synthroid, Ativan, Cozaar, Risperdal

Patient Case

- ⊙ Pertinent labs:

| | SCr | BUN | K ⁺ | pH | pCO ₂ | HCO ₃ |
|----------|-------|-----|----------------|------|------------------|------------------|
| Baseline | 1-1.2 | -- | -- | -- | -- | -- |
| 6/25 | 16.8 | 163 | 7.6 | 7.26 | 32 | 13.7 |
| 6/26 | 15.11 | 156 | 7.1 | -- | -- | -- |

Patient Case

⊙ Hospital treatment course:

- 6/25 ($K^+ = 7.6$):
 - NaHCO_3 100 mEq @ 1632
 - Regular insulin 10 units IV @ 1729
 - Dextrose 50% 1 syringe @ 1729
 - Calcium gluconate 1 amp @ 1729
- 6/26 ($K^+ = 7.1$):
 - D5W with NaHCO_3 150 mEq @ 75 mL/hr then changed to 100 mEq @ 120 mL/hr (x1 liter)
 - Regular insulin 10 units IV @ 1032
 - Dextrose 50% 1 syringe @ 1032
 - Calcium gluconate 1 amp @ 1441
- 6/27: Patient transferred to hospice care
- 6/28: Discharged to hospice

Patient Case

⊙ What are some potential causes of patient's hyperkalemia?

- Decreased PO intake
- Impaired kidney excretion
- Medication-Cozaar
- Acidosis

⊙ Was the patient treated appropriately?

- Beta agonists?
- Calcium gluconate given 4 plus hours after insulin/dextrose on 4/26. This should have been given first.

Conclusion

- ⦿ Hyperkalemia is one of the few potentially lethal electrolyte disorders and is considered a medical emergency
- ⦿ Treatment of hyperkalemia should not be withheld despite no EKG changes
- ⦿ Treatment should be given for a plasma potassium of > 6.5 mEq/L and/or EKG changes

References

- ⦿ Kraft MD, Btaiche IF, Sacks GSS, et al. "Treatment of electrolyte disorders in adult patients in the intensive care unit." *Am J Health Syst Pharm* 2005;62:1663-82.
- ⦿ Weisburg, LS. "Management of severe hyperkalemia." *Crit Care Med* 2008;36:12.