

QUICK PIMPS: Treatment of Hyperkalemia

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DidacticsOnline

Understanding Causes

- Hyperkalemia is most often due to decreased urinary potassium excretion secondary to acute or chronic kidney disease and/or disorders or drugs that inhibit the RAAS.
- Less commonly, total body potassium can remain the same or even decrease and potassium can move out of the cells secondary to various processes (such as hyperglycemia). This results in redistributive hyperkalemia.

Indications for treatment

- Hyperkalemia per lab results with EKG changes present
- Serum potassium greater than 6.5 to 7 meq/L
- A serum potassium that is rapidly increasing
 - Note here that in patients with substantial tissue breakdown (eg, rhabdomyolysis, crush injury, tumor lysis syndrome) large amounts of potassium is released from the cells, which can lead to rapid and substantial elevations in serum potassium. So aggressive treatment is indicated even with only slight increase in potassium per serum studies.

- Essentially the treatment comes down to acutely managing hyperkalemia and its adverse results and then ultimately removing excess potassium if the situation calls for it.
 - This can be broken down in to three approaches to the treatment
 - 1) antagonizing the membrane effects of potassium
 - Using calcium
 - 2) driving extracellular potassium into the cells
 - Insulin with glucose, beta-2-adrenergic agonists and sodium bicarbonate
 - 3) Removing excess potassium from the body
 - Diuretics, Resins or Dialysis

ANTAGONIZING MEMBRANE EFFECTS OF POTASSIUM

Calcium

- **Mechanism:** directly antagonizes the membrane actions of hyperkalemia.
 - High levels of potassium induce depolarization of the resting membrane potential which leads to inactivation of sodium channels and decreased membrane excitability. This leads to the cardiotoxicity of hyperkalemia. This is a potentially fatal complication of hyperkalemia so calcium tx is very important early in severe cases.
- **Onset:** Effect of IV calcium is seen within minutes and lasts for 30-60 minutes.
- **Indications:** Severe manifestations with wide QRS complexes or loss of P waves. Essentially in patients where it is too risky to wait the 30 minutes to an hour it takes of insulin to provide benefits.
- **Effect:** protects heart but DOES NOT change serum potassium levels.
- **Dose:** Calcium gluconate 1000 mg over 2-3 minutes. Calcium chloride 500 to 1000 mg over 2-3 minutes. Central access preferred with calcium chloride due to possible peripheral vein irritation and extravasation leading to tissue necrosis. Repeat either after 5 minutes if EKG changes persist.
- **Special note:** in patients being treated with digitalis calcium should be administered more slowly and with a more dilute solution. This is to avoid cardiotoxic effects of Calcium that patients on digitalis are susceptible to.

DRIVING EXTRACELLULAR POTASSIUM INTO CELLS

Insulin with Glucose

- **Mechanism:** insulin enhances the activity of the Na-K-ATPase pump in skeletal muscle thus driving potassium in to the cells and reducing serum concentrations.
 - Glucose is given to prevent hypoglycemia but is not needed if blood glucose is above 250g/dl
- **Onset:** effect of insulin begins in 10 to 20 minutes but does not peak until 30 to 60 minutes. The effects last for 4-6 hours.
- **Effect:** up to 0.85 meq/L decrease in potassium in one hour.
- **Dose:** either drip or bolus.
 - Drip is 10 units of regular insulin in 500 ml of 10% dextrose over 60 minutes.
 - Bolus is 10 units of regular insulin followed by 50ml of 50% dextrose.
 - Bolus regimen provides a better reduction in serum potassium due to higher insulin levels achieved by bolus but there is a higher incidence of hypoglycemia in patients receiving bolus therapy.
- **Special note:** giving glucose alone theoretically raises endogenous insulin levels providing a therapeutic effect but endogenous release is highly variable. If endogenous insulin release is impaired, hyperglycemia can result which will actually raise plasma osmolality promoting water and potassium movement out of cells and worsen hyperkalemia.

Beta-2-adrenergic Agonists

- Essentially this is either Albuterol (or Terbutaline) or epinephrine. Albuterol is used rarely and epinephrine should really not be used at all. Epinephrine also has alpha-adrenergic activity which can actually cause potassium movement out of cells, not into them.
 - **Mechanism:** like insulin albuterol increases the activity of the Na-K-ATPase pump in skeletal muscle. In addition it also activates the Na-K-2Cl cotransporter. Both act to drive potassium into cells.
 - **Onset:** peak effect is 90 minutes with nebulization and 30 minutes when given IV.
 - **Effect:** Has been shown to lower potassium concentrations by 0.5 to 1.5 meq/L.
 - **Indications:** Albuterol and insulin with glucose have an additive effect. Albuterol should not be used as a monotherapy with patients in end-stage renal disease with hyperkalemia but can be added to insulin with glucose to maximize effect. Due to potential tachycardia and possible angina albuterol should be avoided in patients with coronary disease.
 - **Dose:** nebulized or IV. Nebulized form is 10-20 mg in 4ml of saline by nebulization over 10 minutes (interestingly this is 4-8 times the dose used traditionally for respiratory therapy). IV form is 0.5 mg by intravenous infusion.
 - **Special note:** subcutaneous terbutaline can also be used if patient cannot tolerate albuterol nebulizer and IV albuterol is not available.

Sodium Bicarbonate

- **Mechanism:** raises the serum pH resulting in hydrogen ion release from cells to buffer. This H release is accompanied by potassium movement into cells to maintain electroneutrality
- **Onset:** within 15 minutes via IV with duration of action of 1-2 hours.
- **Indications:** limited evidence supporting the acute management of hyperkalemia with sodium bicarbonate. It should not be used alone in acute therapy but can be beneficial in chronic kidney disease patients with metabolic acidosis regardless of potassium.
- **Dose:** in acute setting (again not as monotherapy) should be given as 150 meq in 1L of D5W. If given in normal saline you are giving increased sodium so plasma osmolality will increase and slow the correction of hyperkalemia

REMOVING EXCESS POTASSIUM

Diuretics

- Increase potassium loss in patients with normal or only mildly to moderately impaired renal function. In patients with chronic hyperkalemia their kidney function may be severely impaired and diuretics have not been proven to be effective in this patient population.

Cation Exchange Resins

- Main resin used is sodium polystyrene sulfonate. Brand names Kayexalate and SPS among others.
- In the GI tract sodium polystyrene sulfonate takes up cations (potassium as well as calcium and magnesium) and releases sodium.
- Sodium polystyrene sulfonate actually causes concretions which can lead to severe constipation. For this reason it is often mixed in a suspension containing sorbitol which leads to the copious diarrhea associated with the treatment of hyperkalemia in many cases. The sorbitol suspension can also lead to intestinal necrosis so its use is controversial.
- Even though sodium polystyrene sulfonate in sorbitol is the most widely used treatment for hyperkalemia there should still be a discussion on its usefulness.
- I urge you to listen to EMCrit episode 32 entitled “Is Kayexalate useless?” ... listen, read on the subject, and be able to discuss controversy with attendings.

Dialysis

- Dialysis is the ultimate tx if all other modalities have failed or the hyperkalemia is extremely severe or increasing rapidly.
- Hemodialysis is preferred over peritoneal dialysis.
- Hemodialysis can remove 25 to 50 meq of potassium/ hour



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