Hyperkalemia in the Emergency Department

Key Article

Rafique Z, Peacock F, Armstead T, et al. Hyperkalemia management in the emergency department: An expert panel consensus. Journal of the American College of Emergency Physicians Open. 2021;2(5).

Background

- Hyperkalemia (HK) is a potentially life-threatening disorder occurring in 1% to 10% of hospitalized and up to 2% to 3% of emergency department patients
- It is a quintessential ED diagnosis, that all of us face on a regular basis
- EKG changes can also be a signal of increased severity
- Management options have changed over the years, so the purpose of this review was to create a multidisciplinary review of the recognition and management of hyperkalemia for the ED physician.

Presentation and diagnosis - Peter

- Clinical Presentation & Diagnosis
    - In healthy individuals, K is absorbed in the GI tract and excreted by the kidneys
    - Patients with HK may be completely asymptomatic or may present with musculoskeletal disturbances (fatigue, weakness, even flaccid paralysis), cardiac (arrhythmias, cardiac arrest), or gastrointestinal (GI) disturbances (nausea, vomiting, and diarrhea).
    - A lot of times, patients present with very non-specific symptoms and it's not until your diagnostic testing comes back that you can identify the problem
    - Although there is no global definition of HK because of differences in laboratory assays, the European Resuscitation Council defines mild HK as 5.5–5.9 mEq/L, moderate as 6.0–6.5 mEq/L, and severe as > 6.5 mEq/L.
- EKG changes
    - HK decreases the transmembrane potassium gradient leading to increased potassium conductance and shortening the duration of the action potential
    - As we all know, this can result in ST depression, classic *peaked* T-waves, and QT shortening
    - As the extracellular potassium rises, PR and QRS intervals begin to lengthen and can eventually progress to the dreaded sinewave complex that precedes VF or asystole.
    - Other EKG abnormalities that can occur with hyperkalemia include: bradycardia, A-V blocks, or new bundle branch blocks.
    - **It is important to note**, that although some have proposed that ECG changes are sequential and predictable based on HK severity, this isn't necessarily true.

Treatment options

- Because of the changing treatment options for HK over the past decade, we'll start with discussing the treatment options then go through potential treatment algorithms as recommended by the article's authors last

- Ultimately, the conceptual goal for managing hyperkalemia is to first rapidly shift excess extracellular K to the intracellular space, then eliminate it later as this takes more time.

Medications to Redistribute K - Mike

- Insulin/Dextrose
    - Insulin lowers serum potassium by activating sodium-potassium ATPase (Na-K ATPase) and by moving sodium out of the cell in exchange for potassium into the cell.
    - **Effect/Timing**: Potassium can start to decrease within 15 minutes of administration and its effect may last several hours
    - **Pitfalls**: Hypoglycemia is common! Beware giving in patients with kidney disease or those who are already euglycemic. (consider halving dose of insulin and/or doubling D50 dose)
    - **Group discussion re**: insulin dose
- Beta 2 agonists
    - Work by shifting potassium into the cell by activation of the Na-K ATPase pump
    - **Effect/Timing:** Effect within 30 minutes, peak at 60 minutes, can decrease serum K by 1mEq/L and effect lasts for approximately 2 hours
- Sodium Bicarbonate
    - Sodium bicarbonate promotes uptake of potassium into skeletal muscle by increasing intracellular sodium via sodium-bicarbonate cotransport and sodium-hydrogen exchange and in turn increases Na-K ATPase activity
    - **Effect/Timing:** bicarbonate reduced serum potassium by 0.47 (±0.31) mEq/L at 30 minutes but was less effective than insulin or albuterol
    - **Pitfalls:** recent studies do not support its use in reducing potassium in the absence of metabolic acidosis
    - **Note:** Based on a recent Cochrane review, bicarbonate is not indicated in the acute treatment of HK anymore but can still be used in cases of acidosis

Medications to Eliminate K - Rob

- Loop diuretics
    - Loop diuretics (furosemide, bumetanide, and torsemide) increase potassium secretion into the distal tubule
    - Effect/Timing: Effect within hours
- Oral Binders
    - Probably the medication class with the most change due to increasing options
    - Historically, Sodium Polystyrene Sulfonate binders have been recommended for low acuity HK, but have significant, potentially lethal side effects in critically ill patients.
    - Newer binders appear to be safe, but research on safety and efficacy in critically ill patients is ongoing
    - Sodium Polystyrene Sulfonate (Kayexelate):
        - Binds potassium into the GI tract, often given orally at a dose of 15g up to 4 times/day
        - **Effect/timing:** Lowers potassium by 1.0 mEq/L in about 24 hours, or sometimes longer; Best used for outpatient hyperkalemia management
        - **Pitfalls:** in patients with slow GI motility, or poor gut perfusion, SPS binders have been associated with colonic ulcer, ischemia, and necrosis due to excessive crystal formation in critically ill patients.

    - Patiromer
        - Cation exchange resin that exchanges calcium for potassium, effective for patients on RAAS inhibitors (think ACE-I/ARB meds)
        - **Effect/Timing:** Well tolerated acutely and chronically, dosed at approx 8.4 grams/day, onset of action is about 2-7 hours after first dose.
    - Sodium Zirconium Cyclosilicate (brand name: Lokelma)
        - Selectively exchanges potassium for sodium in the intestine.
        - **Effect/timing:** Large study of > 1700 patients showed effectiveness. A 10mg dose reduced potassium by 0.11 mEq/L within 1hr and by almost 1mEq/L by 48 hours. Often given 3 times/day
- Hemodialysis or Continuous Renal Replacement Therapy
    - Remains the treatment of choice for K elimination in the critically ill patient.
    - **Effect/Timing:** K is removed over hours, but therapy is very effective
    - Requires temporary dialysis catheter placement, which can be a cause for delay if not able to be placed in a timely fashion.

Suggested Management Algorithm for HK - John

- **Step 1:** Probably obvious, but ask the simple question Is it real or spurious? If something doesn't fit, retest
- **Step 2:** Check your EKG: If there are EKG changes, there is a cardiovascular effect of the HK and the patient needs *calcium*. Give 1 gram of calcium gluconate through a PIV. If the patient is hemodynamically unstable, you can give 1g of calcium chloride, but be careful it doesn't extravasate as it can cause significant tissue necrosis.
- **Step 3:** Shift the K and make a plan for K elimination
    - K < 6: Consider insulin/dextrose, beta-agonists, +/- oral binders; Lasix vs. fluids based on the patient's volume status, cardiac function, and renal function
    - K of 6 – 6.5: In addition, *consider* urgent hemodialysis
    - K > 6.5: In addition to medical therapies above, consult for emergent hemodialysis
- **Step 4:** Reassess the patient's K every 2-4 hours as most temporizing treatments have their maximal effect within 2 hours but wear off around 4 hours. If the K on repeat is still > 6 mEq/L consider redosing treatments or discussing emergent HD with your local, friendly nephrologist.

Final Points

- Hyperkalemia remains a common diagnosis in critically ill patients presenting to the ED
- Insulin/dextrose remains one of the most effective treatments to immediately shift K to a safe, intracellular space – but also has common side effects *that can be amplified in patients with renal dysfunction.*
- Oral elimination agents historically have been avoided in critically ill patients, but newer agents may be safer and better tolerated.
- In patients with refractory hyperK or K > 6.5 consider early nephrology consultation for emergent HD