Hyperkalemia

November 2, 2016 by Josh Farkas

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Diagnosis of hyperkalemia

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**clinical presentations**

- Left untreated, hyperkalemia may manifest in the following ways:
  - Neuromuscular weakness (uncommonly seen).
  - Bradycardia
  - Ventricular tachycardia/fibrillation, sudden cardiac death.
  - In practice, most patients are asymptomatic (even with severe hyperkalemia).
EKG diagnosis

- Hyperkalemia can cause a very wide range of EKG changes.
  - The textbook sequence of changes illustrated above often doesn’t occur. Instead, hyperkalemia can mimic a wide variety of pathologies (including STEMI and all varieties of bundle/conduction blocks).
  - Severe hyperkalemia (e.g. K>7 mM) can occur without obvious EKG changes.
- The following patterns are highly suggestive of hyperkalemia. In an unstable patient, it may be reasonable to give IV calcium based on patterns #2-4 below while awaiting a potassium level.
  1. **Peaked T-waves**
     - Narrow, pointy, prominent T-waves.
     - Often the most notable finding on the EKG (may be visible on bedside monitor as well).
  2. **Ventricular tachycardia mimic**
     - QRS wave widens and P-waves may disappear. If patient is tachycardic, this will look like ventricular tachycardia.
     - Useful clues: Compared to ventricular tachycardia, T-waves can be sharper than would be usual and heart rate is often slower than would be typical.
  3. **Sine-wave pattern**
     - Profound widening of QRS complex and peaked T-waves mimics a sine wave.
  4. ** Bradycardia**
     - Hyperkalemia can manifest with bradycardia (often in the context of other drugs that slow down the AV node).
     - There should always be a high suspicion for hyperkalemia in any bradycardic patient, especially if there are other EKG findings to suggest hyperkalemia.

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Sam Ghali, M.D.
@EM_RESUS

#ECG of an unconscious young man thought to be in "V-Tach"

His rate is ~110 bpm
When the rate is <120, think #Hyperkalemia
K+ was 9.5!

If you can recognize this without blood work one day you will save a life!#FOAMed
Lab diagnosis

- Hyperkalemia is variably defined as potassium >5.5 mM or >5.0 mM, depending on the source.
- **Pseudohyperkalemia** refers to artificially elevated potassium due to:
  - (a) Hemolysis
  - (b) Severe polycythemias causing potassium release during clotting (e.g., platelets > 1 million or WBC count > 50,000). This can be avoided by point-of-care testing or measuring labs in a heparinized tube.
  - (c) Prolonged tourniquet application.
- The first response to a lab report of hyperkalemia should be to look at the telemetry tracing and obtain an EKG.
  - If the telemetry/EKG shows features of hyperkalemia, this confirms the diagnosis.
  - If the lab reports severe hyperkalemia but the EKG is normal, repeat the lab.

**Causes of hyperkalemia**

**general concepts**

- Normally the kidney will prevent hyperkalemia by increasing urinary potassium excretion. Persistent hyperkalemia implies dysfunction in renal potassium excretion.
- Critically ill patients often develop hyperkalemia due to a combination of several factors (e.g. hypovolemia plus renal dysfunction plus ACE-inhibitor). Successful treatment may require addressing many of these problems simultaneously.

**differential diagnosis**

- Pseudohyperkalemia
  - Hemolysis
  - Severe leukocytosis/thrombocytosis
  - Delayed sample processing
- Iatrogenic
  - Potassium supplements

https://emcrit.org/ibcc/hyperkalemia/
- 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 penicillin²)
- Heparin
- PRBC transfusion
- Cyclosporine, tacrolimus
- Digoxin toxicity, succinylcholine
- Diabetic ketoacidosis, hyperglycemic hyperosmolar non-ketotic syndrome (HHNS)
- Cellular lysis
  - Hemolysis, hematomas
  - 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):

![Diagram of Hyperkalemia due to dysfunction of the Renin-Angiotensin-Aldosterone system]

**investigation**

- Review medication list and consider clinical context.
- Consider evaluation for cellular lysis (e.g. measure CK & LDH levels).
- Consider evaluation for adrenal insufficiency (e.g. random cortisol, ACTH stimulation test).³

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**Sam Ghali, M.D.**
@EM_RESUS

Replying to @EM_RESUS

These are the classic peaked T-waves of Hyperkalemia. Notice how they are tall and pointy. They become especially obvious if you can compare them to a baseline #ECG.

Be able to recognize them and one day you will save a life! #FOAMed
Risk stratification

The first step of treatment requires determining whether hyperkalemia is life-threatening (severe). No evidence-based definition for "severe" hyperkalemia exists (various articles use a range of semi-arbitrary definitions). Ultimately clinical judgement is needed, with attention to the following factors:

- **Potassium level**: above 6.5-7 mM is more worrisome.
- **Chronicity**:
  - Chronic hyperkalemia is better tolerated (e.g. dialysis patients who frequently have hyperkalemia).
  - Acute hyperkalemia is more dangerous.
- **EKG changes**: bradycardia, QRS widening, or junctional rhythm are particularly worrisome.\(^4\)
- **Ongoing potassium release** (e.g. by tumor lysis syndrome or rhabdomyolysis) increases the likelihood of deterioration.

Treatment of moderate hyperkalemia

**step 1: treat any definable causes**

- Treat all identifiable causes of hyperkalemia.
Discontinue any nephrotoxins and establish a state of euvoeemia with adequate perfusion. Consider a renal diet with limited potassium intake.

**step 2: gentle kaliuresis (renal excretion of potassium)**

- Potassium excretion can generally be promoted using diuretic among patients able to produce urine (otherwise dialysis will be needed).
- Moderate hyperkalemia can generally be treated with a single diuretic (e.g. IV furosemide), followed by volume replacement with **Lactated Ringer’s** [link](https://emcrit.org/pulmcrit/myth-busting-lactated-riegers-is-safe-in-hyperkalemia-and-is-superior-to-ns/), to maintain a net even fluid balance.
- If this regimen fails, more aggressive kaliuresis may be utilized with additional medications discussed below.

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**Rx severe hyperkalemia: Temporizing measures**

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**IV calcium to stabilize the myocardium**

- Initial dose:
  - Peripheral access: 3 grams IV calcium gluconate over 10 minutes.
  - Central access: 1 gram IV calcium chloride over 10 minutes or slow IV push.
- Further doses of calcium may be indicated for persistent, dangerous arrhythmias (e.g. ongoing bradycardia with hypoperfusion).
  - Ideal dosing here is unknown. An expert guideline recommended re-dosing once or twice if needed, while admitting the lack of evidence.\(^5\)
  - In general hyperkalemia is more dangerous than hypercalcemia, so you're probably better off erring on the side of hypercalcemia. If you have a point-of-care electrolyte monitor available, check calcium levels and avoid pushing the ionized calcium >3 mM.
- Calcium only lasts 30-60 minutes, so it may need to be repeated.

**IV insulin to shift potassium into cells**

- Dose:
  - 10 units IV insulin (*must be given IV*).
  - 2 ampules of D50W (100 ml total), although this may be omitted if the glucose is already >250 mg/dL.\(^6\)
- Lasts for several hours, may need to be re-dosed.
- Follow glucose carefully (e.g. q1hr) to avoid hypoglycemia, especially in patients with renal dysfunction, in whom insulin may linger.
### beta-2 agonists

- **Albuterol**
  - Causes a small shift of potassium into cells.
  - Requires a lot of albuterol (10-20 mg, equal to about 4-8 nebulized treatments back-to-back). Logistically, the best way to achieve this dose is to provide albuterol as a continuous nebulized therapy.
- **IV epinephrine**
  - Should not be used solely for hyperkalemia. However, if the patient does require a vasopressor, then epinephrine may be a sensible choice.
  - Epinephrine is phenomenal for hyperkalemia-induced bradycardia, because it simultaneously treats both the hyperkalemia and the bradycardia.

### bicarbonate

**Hypertonic bicarbonate doesn’t work.**
- Ampules of hypertonic bicarbonate have been proven to be ineffective in RCTs.\(^7\)\(^8\)\(^9\)\(^10\)
- The hypertonic nature of the fluid pulls potassium out of the cells due to osmotic shifts (“solvent drag”).\(^11\) This counteracts the effect of increasing the pH, with an overall neutral effect on the potassium.

**Isotonic bicarbonate does work in metabolic acidosis.**
- Isotonic bicarbonate is generally obtained by adding three amps of bicarbonate to a liter of D5W (this creates a 150 mM solution of bicarbonate).
- Isotonic bicarbonate decreases the potassium in three ways: (1) dilution, (2) shifting of potassium into muscle cells, (3) renal potassium excretion is promoted by alkalosis.\(^12\)
- This has been demonstrated to work, but only for patients with metabolic acidosis.\(^8\)\(^13\)\(^14\) Unfortunately, this requires giving 1-2 liters of fluid, a volume which many patients will be unable to tolerate.
- Dosing is discussed in the section below on [volume resuscitation](#fluid).

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**Rx severe hyperkalemia: Potassium elimination**

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https://emcrit.org/ibcc/hyperkalemia/
dialysis vs. kaliuresis

- Ultimately, most patients will require elimination of excess potassium from the body. This may be achieved either via the kidneys (kaliuresis) or via dialysis.
  - Neither kayexalate nor patiromer has been proven to lower potassium acutely. Neither one currently has a role for emergent therapy of hyperkalemia.
  - Patients with end-stage renal disease on chronic dialysis will require emergent dialysis (there is no point in attempting kaliuresis). For most other patients, kaliuresis should be attempted prior to emergent dialysis.

kaliuresis step #1: volume resuscitation if hypovolemic

- Many patients present with renal failure and hyperkalemia due to volume depletion. The first step in managing these patients is volume repletion.
- **Isotonic bicarbonate is the preferred resuscitative fluid in metabolic acidosis** (excluding lactic acidosis or ketoacidosis).
  - The isotonic bicarbonate should be dosed with the goal of bringing the patient's serum bicarbonate level back to a high-normal level (e.g. bicarbonate 24-28 mM). The dose can be estimated by calculating the patient's bicarbonate deficit (MDCalc [https://www.mdcalc.com/bicarbonate-deficit]). Divide the bicarbonate deficit by 150 to estimate the number of liters of isotonic bicarbonate needed. The dose is usually 1-2 liters.
  - Bicarbonate should be infused rapidly for patients with hypovolemia and severe hyperkalemia (e.g. 500-1,000 ml/hour).
  - If the patient remains hypovolemic after receiving enough sodium bicarbonate to normalize the serum bicarbonate level, then residual hypovolemia can be treated with lactated ringers.
  - In the absence of metabolic acidosis, lactated ringers is preferred as the resuscitative fluid (whereas normal saline is contraindicated).
    - The traditional dogma that lactated ringers is contraindicated in hyperkalemia is wrong. In fact, normal saline tends to cause an acidosis which exacerbes hyperkalemia ([https://emcrit.org/pulmcrit/myth-busting-lactated-ringers-is-safe-in-hyperkalemia-and-is-superior-to-ns/]).
    - Plasmalyte or normosol are also fine choices here.

kaliuresis step #2: consider fludrocortisone

https://emcrit.org/ibcc/hyperkalemia/
Oral fludrocortisone (0.2 mg daily) may help stimulate the kidneys to secrete potassium. This is primarily useful in patients with mineralocorticoid insufficiency (green boxes above, for example patients on ACEi/ARB or NSAIDs).

**Kaliuresis Step #3: Diuretic Cocktail**

- The backbone of kaliuresis is a combination of potassium-wasting diuretics, which synergize to cause potassium excretion in the urine.
  - Diuretic dose should be adjusted based on the severity of the hyperkalemia and the degree of the renal dysfunction (renal dysfunction generally causes diuretic resistance).
  - In emergent hyperkalemia, it's better to err on the side of giving excessive diuretic. If the patient experiences a large-volume diuresis, this can be easily corrected by giving back IV fluid. Alternatively, if in inadequate diuretic dose is given, this may cause the patient to be dialyzed unnecessarily.
  - For maximum efficacy a combination of three diuretics may be used (when given at maximal doses this is termed the nephron bomb).
    - 1) Loop diuretic: furosemide 80-160 mg IV or bumetanide 2-4 mg IV
    - 2) Thiazide diuretic: chlorothiazide 500-1000 mg IV
    - 3) Acetazolamide 500-1000 mg IV

**Kaliuresis Step #4: Determine Response to Diuretic**

- If the patient doesn't produce urine in response to diuretic, dialysis will generally be required.
- If the patient does produce urine:
  - Urine volume should generally be replaced with Lactated Ringers to prevent volume depletion.
  - Electrolytes (including magnesium) should be checked frequently and repleted as needed.

**Algorithm**

*Printable Version* [here](https://emcrit.org/ipcc/hyperkalemia/)

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**The Podcast Episode**

Want to Download the Episode?

[Right Click Here and Choose Save-As](http://traffic.libsyn.com/ibccpodcast/IBCC_EP11_Hyperkalemia_Final.mp3)
To keep this page small and fast, questions & discussion about this post can be found on another page [here](https://emcrit.org/pulmcrit/hyperk/).

The following therapies should be avoided: kayexalate, hypertonic bicarbonate (bicarbonate ampules), normal saline.

Don't provide temporizing measures *without* definitive therapy (e.g. patient is given insulin/glucose but no other treatment). This will temporarily make the potassium look better, but the hyperkalemia will inevitably recur later on.

Insulin dosing errors: 10 units must be given *intravenously* (not subcutaneously).

**Going further:**

**General**
- [Treatment of severe hyperkalemia](https://emcrit.org/emcrit/hyperkalemia/) (Scott Weingart, EMCrit)
- [Updates in management of hyperkalemia](http://www.emdocs.net/emdocs-cases-updates-management-hyperkalemia/)(Brit Long and Justin Warix, EMDocs).
- [Hyperkalemia](https://lifeinthefastlane.com/hyperkalemia/) (Kane Guthrie) & [Hyperkalemia Management](https://lifeinthefastlane.com/ccc/hyperkalaemia-management/) (Chris Nickson, LITFL)
- [Hyperkalemia](http://coreem.net/core/management-of-hyperkalemia/) (Anand Swaminathan, CoreEM)
- [Management of life-threatening hyperkalemia](https://rst10em.com/hyperkalemia/) (First10EM)
- [Hyperkalemia management: preventing hypoglycemia from insulin](https://www.aliem.com/2015/04/hyperkalemia-management-preventing-hypoglycemia-from-insulin/) (Bryan Hayes ALiEM)

**EKG in hyperkalemia**
- [Critical hyperkalemia](https://emcrit.org/emcrit/critical-hyperkalemia/) (H Pendell Meyers, EMCrit; focus on EKG diagnosis)
- [Hyperkalemic EKG changes](https://litfl.com/hyperkalaemia-ecg-library/) (Edward Burns, LITFL)
- [ECG changes in hyperkalemia](http://rebelem.com/ecg-changes-hyperkalemia/) (Salim Rezaie, RebelEM)

**Kayexalate**
- [Is kayexalate useless?](https://emcrit.org/emcrit/is-kayexalate-useless/) (Scott Weingart, EMCrit)
- [Is kayexalate useful in treatment of hyperkalemia in the ED?](http://rebelem.com/ecg-changes-hyperkalemia/) (Salim Rezaie, RebelEM)

**References**


2. Penicillin comes in a variety of different forms. Intravenous penicillin G-potassium contains potassium.

3. For situations where there is no clear cause of hyperkalemia, further investigation may involve measurement of renin and aldosterone levels. These take forever to return and usually aren't helpful in the acute management phase.


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The Internet Book of Critical Care is an online textbook written by Josh Farkas (@PulmCrit), an associate professor of Pulmonary and Critical Care Medicine at the University of Vermont.

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