EM CASES SUMMARY

Episode 86 – Hyperkalemia

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General Approach to Emergency Management of Hyperkalemia

Place the patient on a cardiac monitor, establish IV access and obtain an ECG

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If the patient is stable, consider the cause and rule out pseudohyperkalemia (from poor phlebotomy technique, thrombocytosis or leucocytosis) and repeat the potassium to confirm hyperkalemia.

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Stabilize the cardiac membrane **with Calcium Gluconate 1-3 amps (or Calcium Chloride 1 amp if peri-arrest/arrest) if:**

a) K>6.5 or
b) wide QRS or
c) absent p waves or
d) peri-arrest/arrest

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Drive K into cells **with 2 amps D50W + Regular Insulin 10 units IV push followed by B-agonists 20mg by neb or 8 puffs via spacer if:**

a) K>5 with any hyperkalemia ECG changes or
b) K>6.5 regardless of ECG findings

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Eliminate K through the kidneys and GI tract while achieving euvolemia and establish good urine flow

Normal Saline IV boluses **if** *hypo*volemia
Furosemide IV only **if** *hyper*volemic
PEG 3350 17g orally for alert patients remaining in your ED for prolonged period of time
Dialysis for arrest, peri-arrest, dialysis patient or severe renal failure

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Monitor *rythym strip*, glucose at 30 mins, K and ECG at 60 mins and repeat as needed until the K is below 6, ECG has normalized and/or dialysis has been started

The ECG in Emergency Management of Hyperkalemia

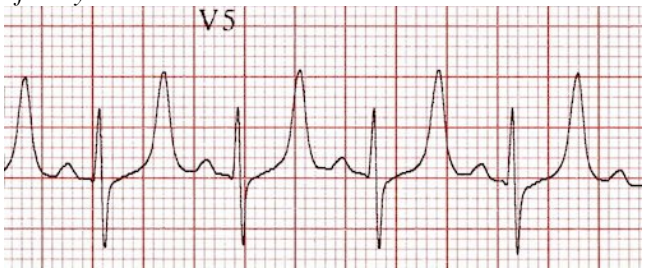
The ECG changes associated with hyperkalemia do not always happen in a step-wise fashion with predictable serum potassium levels. Although it is generally true that higher levels of potassium correlate with progressive ECG changes, the more acute the hyperkalemia the more likely the ECG changes occur. It is possible for a hyperkalemic patient to progress rapidly from a normal ECG to ventricular fibrillation.

The classic ECG progression in hyperkalemia

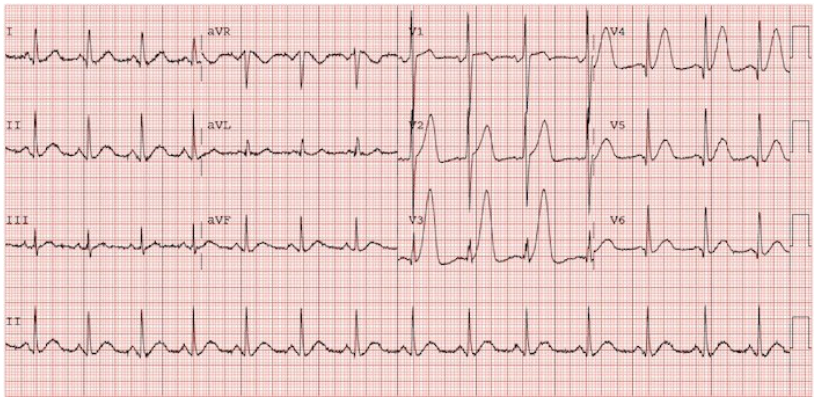
1. **Peaked T wave (K approx 5.5-6.5)**

Peaked T waves reflect faster repolarization of the myosite. A sensitive sign is if the *amplitude of the T exceeds the amplitude of the R*. This

distinguishes *peaked T waves of hyperkalemia* from *hyperacute T waves of early MI* which tend to have a broader base T wave.



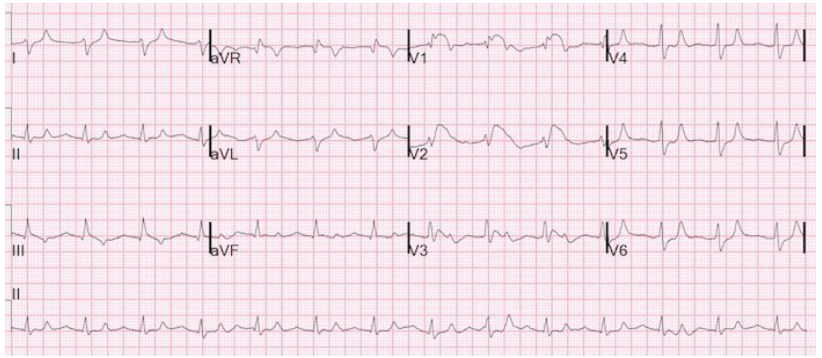
Peaked T waves of Hyperkalemia. Note the amplitude of the T exceeds the amplitude of the R. Care of Life in The Fast Lane blog.



Hyperacute T waves of early MI. Note the broad-based T waves in the anterior leads to help distinguish from peaked T waves of hyperkalemia. Care of Dr. Smith's ECG blog.

2. Prolonged PR interval and flattening or disappearance of the P wave (K approx 6.5-7.5)

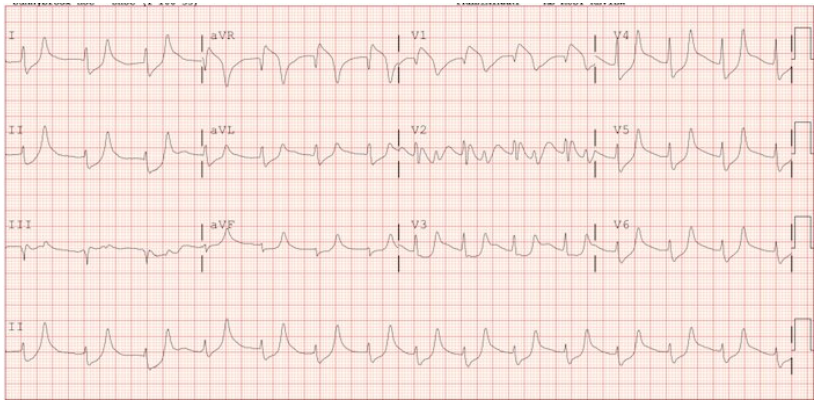
The resting potential of myosite becomes more positive which slows depolarization.



Example of bradycardia with absent or flattened p waves in hyperkalemia. Care of Dr. Smith's ECG blog.

3. Widening of the QRS

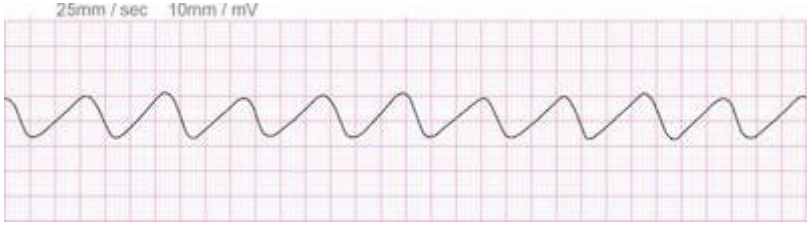
The increasingly positive membrane potential leads to progressively slowed depolarization and widening of the QRS.



Widened QRS in severe hyperkalemia (Care of Dr. Melanie Baimel)

4. Sine Wave: pre-terminal rhythm

As the depolarization slows, the widening QRS begins to merge with the T wave. This is a pre-terminal rhythm which can deteriorate rapidly into Ventricular Fibrillation.



Sine wave in severe hyperkalemia, a pre-arrest rhythm.

Hyperkalemia is the Great ECG Imitator

These ECG findings are not specific to hyperkalemia alone. Given the broad differential for these ECG changes, hyperkalemia has been dubbed the "Great ECG Imitator". It is important to consider the patient's presentation, clinical complaints and trends on the ECG.
PEARL: Hyperkalemia has been known to cause almost any dysrhythmia. Pay special attention to patients in "slow VT" and wide-complex bradycardia and consider treating them empirically as hyperkalemia.

Determine the Cause of Hyperkalemia

First rule out pseudohyperkalemia which accounts for 20% of hyperkalemia lab values.
Pseudohyperkalemia is caused by hemolyzed sample, poor phlebotomy technique leukocytosis or thrombocytosis.
Then treat the underlying cause:

- **Medications:** ACEi, Potassium sparing diuretics, B-Blockers, NSAIDs, Trimethoprim (Septra) and Non-prescription salt substitutes
- **Renal Failure**
- **Cell death:** Secondary to rhabdomyolisis, massive transfusion, crush or burn injuries.
- **Acidosis:** Consider Addisons crisis, primary adrenal insufficiency and DKA.

PEARL: If hyperkalemia cannot be explained by any other cause and the patient has unexplained hypotension, draw a random cortisol and ACTH level and give 100 mg IV solucortef for presumed adrenal insufficiency.

Medications in the Emergency Management of Hyperkalemia

Three main principles

1. Stabilize cardiac membrane
2. Shift potassium intracellularly
3. Eliminate potassium

There are specific treatments geared at targeting each of these three main principles, which we will discuss below. Unfortunately, there is no clear evidence to guide exactly when to initiate specific treatments for hyperkalemia. Our experts recommend using two factors to guide your management:

1. Serum potassium level and
2. ECG

with the following indications for immediate treatment of hyperkalemia in the ED:

K>6.5mmol regardless of ECG OR K>5, <6.5 with ECG changes
Drive K into cells and stabilize cardiac membrane

K>5, <6.5 and no ECG changes
Enhance excretion and ensuring good urine output

Principle 1: Stabilize the cardiac membrane

Indications for administering calcium
K > 6.5mmol
ECG changes (P wave or QRS changes)

There is no good literature to help guide whether *calcium gluconate* or *calcium chloride* is better for stabilizing the cardiac membrane in hyperkalemia. The most important difference to remember is that *calcium chloride* has 3 times more elemental calcium than *calcium gluconate* (6.8 mEq/10 mL vs 2.2 mEq/10 mL) and has greater bioavailability. However, *calcium gluconate* has less risk of local tissue necrosis at the IV site. Therefore, if you decide to give *calcium gluconate*, ensure you are giving sufficient doses of calcium since one amp may not be enough. **Three amps of calcium gluconate are often required to start to see the ECG changes of hyperkalemia resolve**. Remember that calcium does not lower the potassium level.

Our experts recommend using *calcium chloride* through a *large well-flowing peripheral IV or central line* in the arrest or peri-arrest patient. Calcium gluconate is recommended for all other patients given it's lower risk for local tissue necrosis.

Routine use: 1 gram of 10% calcium gluconate (i.e. 10 ml mixed with 100cc of D5W or NS in a mini-bag) over 5-10 minutes. Repeat as needed to achieve QRS <100ms and p waves re-appear.

Arrest or Pre-arrest: Push 1 amp (1 gram) of 10% calcium chloride through a large bore well-running peripheral IV or central line (preferable). Repeat as needed to achieve QRS <100ms and p waves re-appear.

In patients taking digoxin the traditional teaching is that calcium is contraindicated. The so-called "stone heart" from the administration of calcium in patient with digoxin toxicity has been largely debunked. A small case-controlled study found no mortality differences between 23 patients with hyperkalemia and digitalis toxicity who were treated with calcium and 136 patients who were not.

If digoxin toxicity is suspected in the setting of severe hyperkalemia, our experts recommend giving calcium cautiously, at a slower rate than usual: 1 gram of 10% calcium gluconate in 100 cc of D5W or NS over 15-30 minutes (rather than 5 minutes).

Principle 2: Shifting potassium into cells:

The choices for shifting potassium into cells include intravenous insulin and glucose, beta-agonists and bicarbonate. The indications for starting insulin and glucose include a K>5 mmol ***with*** ECG changes or a K> 6.5 mmol regardless of ECG changes. Observational studies have shown that many patients treated with insulin and glucose for hyperkalemia become hypoglycemic when given 1 amp of D50W followed by 10 units of humulin R. Therefore, based on a recent systematic review our experts recommend the following approach to initiating insulin and glucose therapy:

2 amps of D50W followed by 10 units IV humulin R (rapid injection)

Monitor glucose q30 minutes

Repeat K at 60 minutes

Beta agonists are also useful to rapidly shift potassium into cells. They act synergistically with insulin and can lower serum potassium by 1.2 mmol in an hour. Paradoxically, one third of patients will not have the predicted drop in serum potassium, and observational data has shown a very transient initial rise in potassium up to 0.4 mmol after administration of beta agonists. Therefore, B-agonists should NOT be used as mono-therapy and insulin/glucose be given first. The doses of beta agonists for hyperkalemia are generally higher than what you would use in asthma:

Salbutamol 8 puffs by aerochamber or 20mg nebulized

Insulin and beta agonists will start to take effect within 15min with their peak effect being at 60min.

Pitfall: If B-agonists are given *before* insulin/glucose they may cause a transient *rise* in the serum potassium level. Always give B-agonists *after* insulin/glucose.

Bicarbonate is also known to shift bicarbonate into cells. Our experts do ***not*** recommend the routine use of bicarbonate in the treatment of hyperkalemia. It may have a role to play in a small subset of patients who also have a *non-anion gap metabolic acidosis* such as those patients with renal tubular acidosis.

Principle 3: Eliminate Potassium

The kidneys are the main route for eliminating of potassium. Ensuring euvolemia and appropriate urine output is the mainstay of treatment. Inserting a foley catheter will allow you to monitor urine output. Many patients will be hypovolemic and will need fluid resuscitation with crytalloid. If you need to volume resuscitate your patient, the *initial* fluid of choice is Normal Saline even though with huge doses hypercholoremic metabolic acidosis can occure. Ringer's Lactate contains 4mmol/L of potassium, which poses obvious risks of increasing serum potassium if appropriate renal elimination has not started.

There is no role for diuretics in the routine management of hyperkalemia ***unless*** the patient is hypervolemic.

Regarding potassium binding agents such as Kayexalate, a 2005 Cochrane review did not show any evidence that they improve potassium levels. There have also been case reports of Kayexelate causing GI necrosis and perforation. Our experts conclude that there is no role for kayexcelate in the ED.

Consider PEG 3350 orally to help eliminate potassium through the GI tract if the patient is likely to stay in your ED for a prolonged period of time. Given that most patients with hyperkalemia will have some element of renal insufficiency it is important to remember that milk of magnesia and fleet enemas are both contraindicated as they will cause magnesium and phosphate toxicity, respectively.

Hyperkalemia in Cardiac Arrest

Based on the principles of treatment and indications discussed above, our experts recommend the following approach to suspected hyperkalemia (based on patient history and rhythm strip) or confirmed hyperkalemia (based on a point of care blood gas) in cardiac arrest in addition to usual ACLS measures:

Push 1 amp calcium chloride in well running peripheral IV or central line and repeat until the QRS is <100ms

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Epinephrine 5-20 mcg q2-5 minutes (shifts K intracellularly)

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Sodium Bicarbonate 1 amp IV (if suspect severe acidosis)

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Bolus IV NS

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Shift potassium with Insulin and Glucose followed by B-agonist

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Dialysis

Rebound Hyperkalemia

In cases of cardiac arrest due to hyperkalemia, perform CPR until the hyperkalemia is corrected. This may be a much longer time than usual. When ROSC is achieved, it will be primarily due to the effects of calcium rather than decreased potassium levels. The effect of calcium can last 20-30min. Since the stabilizing effects of calcium will wear off, you must promptly work on shifting the potassium and enhancing its elimination as described above. Consider repeating the calcium bolus if there are any worsening ECG changes. Repeat serial potassium measurements to monitor for rebound hyperkalemia, which occurs more often than we'd like.

PEARL: the patient in cardiac arrest with hyperkalemia should not be pronounced dead until their potassium level is normalized

Intra-arrest Dialysis

In cardiac arrest, case reports have demonstrated successful ROSC and good neurologic outcomes despite prolonged arrest when dialysis is initiated *during* CPR to correct hyperkalemia.

Future Directions in Emergency Management of Hyperkalemia

A new potassium binding drug, *ZS-9* shows promise in the acute treatment of hyperkalemia and may make it possible to avoid or postpone the most effective therapy, emergency hemodialysis.

Other FOAM Resources for Hyperkalemia:
Rebel EM on **kayexalate** and **ECG changes in hyperkalemia**
EMBasic on **hyperkalemia**
Life in the Fast Lane on **hyperkalemia management**
Academic Life in EM on **preventing hypoglycemia from insulin** in hyperkalemia
First10EM on **initial management of hyperkalemia**
Dr. Smith's ECG blog on **ECG changes with hyperkalemia**

Dr. Etchelles, Dr. Bailel, Dr. Helman & Dr. Kilian have no conflicts of interest to declare.

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