

Hyperkalemia Case Review

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Case Number Eight

Background Information

Potassium abnormalities are common problems in emergency medicine. Most of a patient's potassium is contained intracellularly, with only a small fraction remaining in the extracellular fluid. Hyperkalemia is defined as a serum potassium level greater than 5.5 mEq/L and generally occurs either due to decreased excretion of potassium or to increased potassium secretion into the extracellular space. Potassium excretion may be impaired by renal failure, renal tubular acidosis, hypoaldosteronism, and by medications such as potassium sparing diuretics, NSAIDs, and ACE inhibitors. Events that increase secretion of potassium into the intracellular space include increased potassium ingestion, blood cell breakdown due to hemolysis, burns, tumor lysis, muscle breakdown due to rhabdomyolysis, and redistribution of potassium from the intracellular space due to such things as acidosis or medications.

Patients with hyperkalemia are often asymptomatic. When patients develop symptoms, the symptoms are often nonspecific, including weakness, fatigue, or palpitations.

Diagnosis of hyperkalemia is usually based on laboratory studies, although the ECG may contain changes suggestive of hyperkalemia. Typical ECG findings in hyperkalemia progress from "peaked" T waves and a shortened QT interval to lengthening PR interval and loss of P waves, and then to widening of the QRS complex culminating in a "sine wave" morphology and death if not treated.

Treatment of hyperkalemia involves stabilizing the myocardium to prevent arrhythmias, shifting potassium back into the intracellular space and removing excess potassium from the body.

- Intravenous insulin increases intracellular resorption of potassium for approximately 4 hours. Concomitant administration of intravenous dextrose will prevent hypoglycemia.
- Nebulized beta agonists also increase cellular uptake of potassium. Five to twenty milligrams of nebulized albuterol will lower the serum potassium level for approximately 2-3 hours.
- With more pronounced hyperkalemia, intravenous calcium is often recommended to prevent arrhythmias by antagonizing the effects of potassium on the myocardium. Case reports of sudden death when intravenous calcium is given to digoxin toxic patients have led some sources to recommend against giving calcium to patients with potential digoxin toxicity. Magnesium is an acceptable alternative if calcium is unavailable or contraindicated.
- Intravenous sodium bicarbonate will lower serum potassium levels for approximately 2 hours and is still an acceptable treatment for hyperkalemia, although it has fallen out of favor except in the treatment of known acidosis.
- Binding resins such as sodium polystyrene, (Kayexelate) exchange potassium for sodium in the GI system and remove potassium from the body. Such resins should be administered in combination with other methods of managing hyperkalemia since the onset of action of binding resins is 1-2 hours.
- Finally, hemodialysis is the definitive treatment for removing potassium from the blood. Subsequent re-equilibration of potassium may cause "rebound" hyperkalemia and dialysis may not be readily available under many circumstances.

Facts of the Case

The case submitted to the Standard of Care Review Panel included the following facts:

A 65-year-old male came to the emergency department (ED) with a "poorly characterized history of generalized chest pain" that was present 30 minutes prior to arrival. In addition, the patient also complained of generalized body pain, generalized weakness, bilateral lower extremity pain, weakness to his arms and legs, low back pain, and difficulty walking. According to the triage notes, the patient described the pain in his arms and legs as 10 on a 1-10 scale. Review of systems was also significant for blurred vision, shortness of breath, and dizziness. The patient had a past medical history of coronary artery disease, hypertension, chronic renal failure, stroke, and diabetes. The patient was scheduled to have dialysis the day prior to his ED visit, but there is no indication on the medical records whether or not the patient went to this dialysis session.

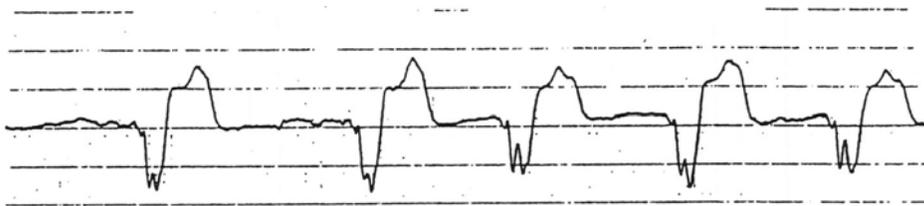
The patient had been in the ED two weeks prior to his presentation also complaining of weakness. A rhythm strip done on that visit is shown below.



During his prior visit, his lab tests were normal and he was discharged to follow up with his nephrologist.

The patient was taking many medications including Amiodarone, Prevacid, Coumadin, Synthroid, Zaroxolyn, allouprinol, aldactone, Renagel, Zetia, and Epogen.

On physical examination, the patient's vital signs were temperature 96.7, pulse 61, respiration 16, blood pressure 105/73, oxygen saturation 97% on room air. The patient was awake and alert in moderate distress due to his pain complaints. His lungs had normal breath sounds. His heart had an irregular rhythm and a grade 2 systolic murmur. His extremities were normal. No other abnormalities were noted on his physical examination. Capillary blood glucose was normal at 82. A chest x-ray showed clear lungs, normal cardiac silhouette. An EKG showed atrial fibrillation with rate of 60, left bundle branch block, and generalized ST segment changes. A rhythm strip is shown below.



The patient was seen by the physician approximately 20 minutes after his arrival. Nursing notes show that the patient was continuing to complain of chest pain 30 minutes after he arrived. His blood pressure had decreased to 98/69. He was given Demerol 50 mg slow IV push.

At this point the patient was signed over to another emergency physician at shift change.

One hour after the patient arrived, the lab technician was drawing blood and the patient stated that he was having some relief of chest and leg pain. A repeat blood pressure was 89/65. One hour and 15 minutes after he arrived, the patient complained of feeling sweaty. He was repositioned to recheck his blood pressure and became unresponsive. A code was called and CPR was initiated.

During the code, the patient was given epinephrine, atropine, and a total of 4 amps of calcium chloride. ACLS was performed for 20 minutes with no response. The patient was pronounced dead approximately 1 hour and 45 minutes after his arrival. Just as the code ended, the lab called the ED to notify them of the patient's critical 8.2 potassium level.

Autopsy showed severe atherosclerotic heart disease with 90% compromise of the right coronary artery and 100% blockage of the left coronary arteries. The patient also had right ventricular hypertrophy, dilated cardiomyopathy, congestive heart failure, cirrhosis, and 1950 cc of fluid in the peritoneum.

Statements Made By Experts

The physicians were sued by the patient's family.

During depositions, the defendant physician who initially evaluated the patient stated that he did not suspect that the patient may have had high potassium after reviewing the EKG because he thought the patient had a left bundle branch block. Instead, he suspected that the patient may have an "electrolyte imbalance," stating that the patient "could have had low potassium as easy as high potassium" and that the patient may have been "overdialyzed]. He also stated that physicians should not automatically treat a patient in chronic renal failure for hyperkalemia because if the patient has low potassium, the treatments could be potentially lethal.

The defendant physician who received "sign out" also testified that he did not suspect hyperkalemia based upon the patient's EKG findings. He interpreted the EKG as a left bundle branch block with nonspecific ST changes and an occasional PVC.

The management of the patient was criticized because the physicians chose to wait for lab results to come back prior to initiating treatment for hyperkalemia. Given the patient's history, lack of dialysis for the previous 24 hours, EKG findings, and falling blood pressure, the expert believed that the physicians should have treated the patient for hyperkalemia prior to knowing the patient's potassium level. The expert disagreed with the physician's statement that it was prudent to wait for the results of laboratory studies before initiating treatment for hyperkalemia since the treatments for hyperkalemia may cause a lethal imbalance of other metabolites.

According to the expert's statements, waiting for labs to return was deemed "substandard and lethal."

Findings of the Standard of Care Review Panel

After reviewing the submitted materials, it was the opinion of the Standard of Care Review Panel that:

- The patient's 9mmHg drop in systolic blood pressure was not likely due to the patient's high potassium level. In fact, the patient had been discharged with a lower blood pressure on his previous visit.
- A lack of dialysis for 24 hours should not raise the suspicion for a patient to have hyperkalemia. Chronic renal failure patients are generally dialyzed three times per week and routinely go 48 to 72 hours without dialysis.
- ECG changes were present between the patient's two visits to the ED. Although those changes may have been due to elevated potassium, other etiologies for the patient's ECG changes, including effects from amiodarone (one of the patient's medications), hypothyroidism (the patient was taking Synthroid) and hypocalcemia (common in patients with renal failure), or a combination of several factors, must also be considered.
- A general statement that physicians should wait for laboratory tests to return before initiating treatment is inaccurate. Emergency physicians routinely initiate treatment prior to receiving test results for complaints such as chest pain and for diseases such as meningitis. Instead, a decision whether to wait for laboratory testing prior to initiating treatment must be viewed on a case-by-case basis.

- Based on the information contained within the submitted materials and upon pertinent literature, the Standard of Care Review Panel believed that the physicians involved in this case did not breach the standard of care.

In addition, the Standard of Care Review Panel emphasized that the standard of care is a “reasonableness” standard. In other words, a physician is judged by what another reasonable physician would do under given circumstances, not by what the most astute physician would do under those circumstances.

References

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