EM CASE OF THE WEEK.

BROWARD HEALTH MEDICAL CENTER
DEPARTMENT OF EMERGENCY MEDICINE

BROWARD HEALTH
Care Warriors

Author: Bansi Patel, OMS IV |

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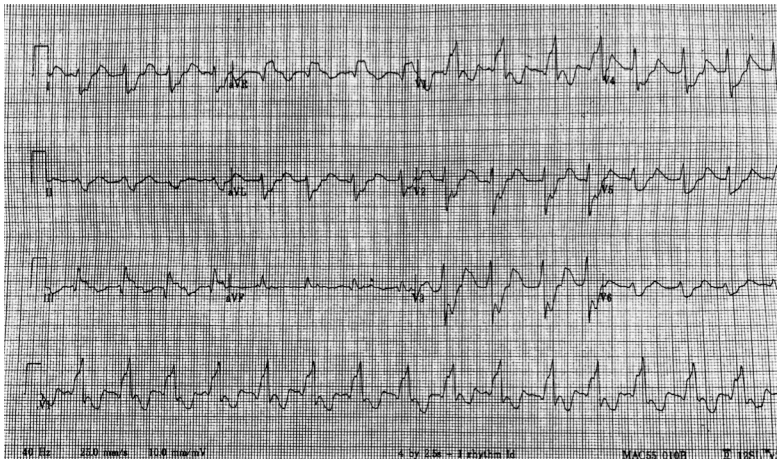
Hyperkalemia

A 57-year-old male with a medical history of hypertension and end stage renal disease on hemodialysis three times weekly presents to Broward Health North Emergency Department with chest pain for 4 hours. Pain was described as constant non-radiating 6/10 heaviness of the left chest that started suddenly while at home. The patient denied any aggravating or relieving factors. He has never had this type of chest pain prior. He denied any shortness of breath, diaphoresis, numbness or tingling, headache, or nausea/vomiting. The patient stated his last hemodialysis was sometime last week.

Patient was afebrile, but hypertensive at 175/108, tachycardic with heart rate of 110 and tachypnic with a respiratory rate of 21. On physical exam the patient was alert and oriented x3. Cardiac exam demonstrated regular rate and rhythm wit no murmurs and normal peripheral perfusion. Lungs were clear to auscultation bilaterally in all fields with equal, non-labored breath sounds. No JVD or focal neurologic deficits were appreciated.

EKG of the patient showed the following.



What is the classic EKG change associated with in hyperkalemia?

A. U waves

B. Peaked T waves

C. Increased QT segment

D. Down sloping of ST segments

E. Osborne waves

F. Delta waves

Answer: B – Peaked T waves

U waves are characteristic of hypokalemia. Lengthening of the QT segments is typically found with hypocalcaemia. Digoxin and myocardial infarction can result down sloping of ST segments. Osborn waves or J waves can be found in patients with hypothermia and delta waves in those with Wolf-Parkinson White Syndrome.

EM Case of the Week is a weekly "pop quiz" for ED staff.

The goal is to educate all ED personnel by sharing common pearls and pitfalls involving the care of ED patients. We intend on providing better patient care through better education for our nurses and staff.

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Department of Emergency Medicine
1625 SE 3rd Avenue
Fort Lauderdale, FL 33316

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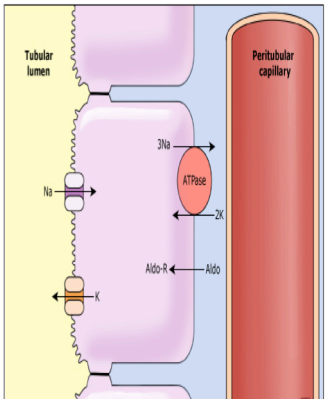
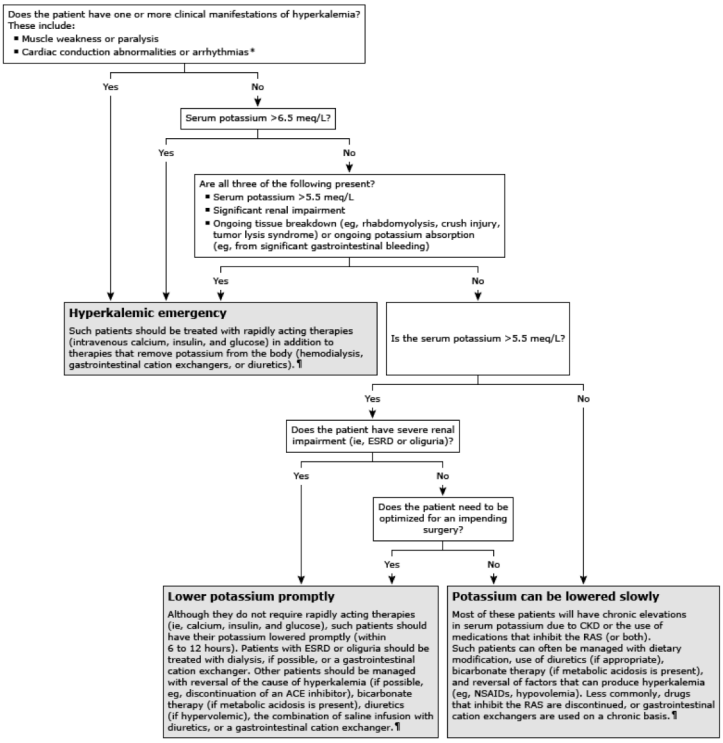
Discussion



Approximately 98 percent of the body's potassium is intracellular. The Na-K-ATPase on the cell membranes pumps 3 sodium molecules out for 2 potassium in to maintain resting membrane potentials crucial for propagating action potential for neural and muscle function.

In otherwise healthy individuals, potassium excretion is determined by three main factors in the principal cells of the distal tubule: an increase in plasma potassium concentration or intake, increased aldosterone secretion, and increased delivery of sodium and water to distal secretory sites. Hyperkalemia therefore rarely occurs in healthy individuals as potassium adaptation takes place. With increased extracellular potassium levels, there is an aldosterone independent increase in activity of apical secretory potassium channels, increase Na-K-ATPase activity, and increased activity of apical epithelial sodium channels to create electrogenic driving force for potassium excretion. All three of these mechanisms work to maintain electrochemical gradients in the body.

Therefore persistent hyperkalemia requires some form of impairment in urinary potassium excretion, whether through pseudohyperkalemia, decreased excretion or increased release from cells. Pseudohyperkalemia, or falsely elevated potassium levels typically result from hemolysis or repeated fist clenching with tourniquet placement. Renal failure and low aldosterone levels due to type IV renal tubular acidosis, Addison's disease,



Treatment

The best initial treatment of hyperkalemia takes into account three main mechanisms. Calcium Chloride is crucial to antagonize the membrane action potentials of potassium. The effect of calcium is short acting however and must be combined with other therapies to reduce potassium load. To drive excess potassium into cells, insulin with glucose, beta-2 agonists or sodium bicarbonate, if acidotic, are helpful. Lastly, the potassium can be removed the body with loop or thiazide diuretics, cation exchange resins like kayexelate, or hemodialysis. Loop diuretics should not be used in patients with severe renal impairment. Hemodialysis is preferred over cation exchangers in patients with severe renal impairment if it can be performed within 6 hours.

For a list of educational lectures, grand rounds, workshops, and didactics please visit ***BrowardER.com*** and **click** on the ***"Conference"*** **link**.

All are welcome to attend!

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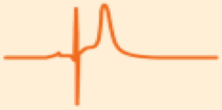
Progression of EKG Changes in Hyperkalemia

Tall peaked T waves with shortened QT intervals are usually the first findings. As levels become more severe, there is lengthening of PR interval and QRS waves. P waves may disappear as QRS continues to widen into a sine wave pattern. If untreated, asystole can occur confirming the complete lack of cardiac activity.

Serum potassium	Typical ECG appearance	Possible ECG abnormalities
Mild (5.5–6.5 mEq/L)		Peaked T waves Prolonged PR segment
Moderate (6.5–8.0 mEq/L)		Loss of P wave Prolonged QRS complex ST-segment elevation Ectopic beats and escape rhythms
Severe (>8.0 mEq/L)		Progressive widening of QRS complex Sine wave Ventricular fibrillation Asystole Axis deviations Bundle branch blocks Fascicular blocks

EpoMedicine

Despite these ominous EKG changes, the progression and severity of the EKG findings do not correlate well with serum potassium concentrations. Peaked T waves can be seen in other conditions such as acute myocardial infarction, making them a nonspecific finding. Therefore, serial measurements of serum potassium rather than EKG findings are helpful to guide treatment efficacy.

High potassium levels can also produce a type I Brugada pattern in critically ill patients with potassium levels > 7.0 mEq/L. EKG will demonstrate a pseudo-right bundle branch block, persistent ST elevations in two consecutive precordial leads, absent p waves, widened QRS complexes and abnormal QRS axis.

Take Home Points

- Na-K-ATPase is crucial for keeping adequate serum potassium levels to maintain proper membrane gradients.
- The body has innate mechanism to preserve acceptable serum potassium levels. These mechanisms are unregulated when extracellular potassium levels rise to increase urinary excretion of potassium primarily via principal cells in the distal tubules. Therefore persistent hyperkalemia requires impairment in urinary potassium levels.
- Pathognomonic EKG changes of hyperkalemia include peaked T waves, and widened QRS interval that can become sinusoidal.
- Typical treatment involves calcium to antagonize potassium induced membrane potentials, insulin with glucose to drive potassium into the cells and kayexelate to increase potassium excretion.

This month's case was written by Bansi Patel. Bansi is a 4th year medical student from NSU-COM. She did her emergency medicine rotation at BHMC in October 2017. Bansi plans on pursuing a career in Anesthesiology after graduation.

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