Clinical Challenge: Hyperkalemia in the Heart Failure Patient

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DISCLOSURE INFORMATION:
There are no relationships to disclose related to this presentation.

Objectives

• 1. Describe the precipitating factors and the dangers of hyperkalemia in the HF patient.

• 2. Contrast the mechanism of actions of organic polymer resins and new therapies for hyperkalemia.

• 3. Identify potassium containing foods and the importance of understanding their dietary role.

Epidemiology

• Hyperkalemia in the healthy population is rare.
• < 5% of general population worldwide\(^1\)
• Hospitalized patients range 1 – 10%\(^1\)
• 75% cases are drugs related\(^2\)
• Other Predisposing Factors\(^2\)
  - Frenal function
  - Severe diabetes
  - Poly-pharmacy

Incidence
Heart Failure increases the potential for hyperkalemia
• SOLVD Trial
  - 6% (K⁺ ≥ 5.5 mmol/L)
  - 1% (K⁺ ≥ 6.0 mmol/L)
• TIME-CHF
  - 13.4% (K⁺ ≥ 5.5 mmol/L)
  - 4.9% (K⁺ ≥ 6.0 mmol/L)
• Retrospective study of patients on ACEi
  - 20% (K⁺ ≥ 5.5 mmol/L)

Reported Hyperkalemia in Heart Failure Trials
• Definition criteria ≥ 5.5 or 6.0 mmol/L
• Timing of sampling
• Type of sample
• Level of renal function
• Hyperglycemia
• Control of diet
• Drug use and dosing range
• Diuretic therapy adjustments

“The kidney presents in the highest degree the phenomenon of sensibility, the power of reacting to various stimuli in a direction which is appropriate for the survival of the organism; a power of adaptation which almost gives one the idea that its component parts must be endowed with intelligence.”
E. Starling - 1909
Potassium Handling in the Nephron

- Proximal Tubule
  - Reabsorption K+ 90-100%
  - Filtered K+ 100%

- Loop Henle
  - Reabsorption K+ 0-10%
  - Excretion K+ Variable

Potassium

- Major intracellular cation
- Concentration maintained by the Na⁺/K⁺ pump
- Intracellular concentration 98%
- Extracellular concentration 2%
- Transmission and conduction of nerve impulses, normal cardiac rhythms, and skeletal and smooth muscle contraction.

- Potassium
  - The passive outward diffusion of K⁺ is the most important factor that generates the resting membrane potential.
  - Maintenance of steady state requires K⁺ ingestion = K⁺ excretion
  - Nearly all regulation of renal K⁺ excretion and total body K⁺ balance occurs in the distal nephron, via principal cells
  - Potassium balance is regulated by the kidney, aldosterone, insulin secretion and changes in pH.

Classification of Hyperkalemia

- Normal: 3.5 to 5.1 mmol/L
- Mild: > 5.5 to 6.0 mmol/L
- Severe: Levels of 7.0 mmol/L or greater

Functions of Potassium

- Maintain the osmotic integrity of cells
  ▫ Osmotic pressure in ICF
- Maintain acid-base balance
  ▫ Through potassium-hydrogen exchange
- Contribute to the reactions that take place in cells
  ▫ Transform carbohydrates into energy
  ▫ Convert amino acid to protein
  ▫ Change glucose into glycogen
- Play a critical role in the excitability of skeletal, cardiac, and smooth muscle.

Transcellular Shifts

- Sodium-potassium ATPase
  ▫ Both insulin and epinephrine increase the activity of sodium-potassium pump.
  (An increase in potassium level stimulates insulin release. ↔
  a feedback mechanism)
- Potassium channels
  ▫ ECF osmolality↑→H₂O leaves cell→ICF K⁺↑→K⁺ moves out of cell through K⁺ channels →ECF K⁺
  ▫ Exercise
- Potassium-hydrogen exchange to maintain electrical neutrality
  ▫ Acidosis
HYPERKALEMIA CAUSES

I. Shifting of K into extracellular space
   A. Tissue (lots of cells) damage: burns, crush injury, rhabdomyolysis
   B. Acidosis
   C. Hyperosmolar states
   D. Insulin deficiency
   E. Extreme exercise or seizures

II. Impaired Renal Excretion (↑ total body K)
   A. Renal insufficiency/failure
   B. Endocrine: adrenal insufficiency, ↓ renin, ↓ aldosterone, pseudohypoaldosteronism

III. Iatrogenic
   A. Dietary intake
   B. Potassium supplement
   C. Medications: NSAIDS, ACE inhibitors, K sparing diuretics, digitalis and heparin

PSEUDOHYPERKALEMIA

Falsey elevated serum K due to K movement out of the cells during or after a blood draw.
   - Lysis of RBC
   - Specimen deterioration (cooling, prolonged storage)
   - ↑WBC, ↑Plt
   - Drawing blood downstream from a vein into which K is infusing
   - Trauma: forcible expression of blood (milking a heel stick)
   - Exerciser: fist clenching with blood draws

Hyperkalemia Risk Factors in Heart Failure

- Increased age ≥ 75 yrs
- Male gender
- Renal dysfunction
- NYHA Class III – IV
- Diabetes
- Atrial fibrillation
- Loop diuretics
- Guideline Directed Medical Therapy (GDMT)

Hyperkalemia in Heart Failure

Potassium excretion is diminished by two primary mechanisms:¹

• Reduction in GFR (despite normal creatinine value)
• Reduction from Pharmacotherapy² (decrease in aldosterone production or interference)
  - Introduction or during chronic therapy of ACEi
  - Volume contracting illness (diarrhea, poor intake, over diuresis)


Hyperkalemia in Heart Failure

The Hyperkalemia Dilemma

• Cardiotoxicity and consequences requiring prompt treatment/intervention
• Clinical relevance requiring modifications to HF drug regimens

Hyperkalemia in Heart Failure

• Aldosterone Receptor Antagonist:¹

<table>
<thead>
<tr>
<th>NYHA</th>
<th>II - IV</th>
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<tbody>
<tr>
<td>LVEF</td>
<td>≥50% or less</td>
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<tr>
<td>Creatinine</td>
<td>≥2.0 mg/dL (Men)</td>
</tr>
<tr>
<td>eGFR</td>
<td>&gt;60 ml/min/1.73m²</td>
</tr>
<tr>
<td>Potassium</td>
<td>≤5.0 mEq/L or less</td>
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</table>

• Careful follow up is needed for early recognition and treatment of hyperkalemia

Hyperkalemia in Heart Failure

- Potassium supplement discontinued or reduced
- Counsel patient regarding avoid foods high in potassium
- Avoidance of NSAIDs
- Addition or increase in dosage of RAAS medications result in new cycle of monitoring

Renal & Potassium Assessment

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Interval</th>
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<tbody>
<tr>
<td>2-3 days following initiation</td>
<td>7 days</td>
</tr>
<tr>
<td>Monthly for 3 months</td>
<td>Every 3 months</td>
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Co-trimoxazole and sudden death in patients receiving inhibitors of renin-angiotensin system: population based study


- Co-trimoxazole and sudden death in patients receiving inhibitors of renin-angiotensin system: population based study
  - Retrospective chart review
  - Ontario, Canada 1994-2012
  - ≥ 66 yrs
  - Commonly prescribed for UTI
  - Structured similarly to potassium sparing amiloride
  - Blocks sodium channel in distal nephron, impairing renal potassium elimination
  - In combination with ACE inhibitor or ARB 7-fold increase risk hyperkalemia
  - Relative to amoxicillin, co-trimoxazole was associated with increased risk of sudden death, (adjusted odds ratio 1.38, 95% CI 1.09 – 1.54)
  - Consider alternate antibiotic or limit dosing and duration
  - Close monitoring of potassium

Signs & Symptoms

- Many patients with hyperkalemia are asymptomatic
- Symptoms are nonspecific
- Weakness and fatigue most common
- Mild
  - Hypopolarized membrane, causing neuromuscular irritability
  - Tingling of lips and fingers, restlessness, intestinal cramping, and diarrhea
- Severe
  - The cell is not able to repolarize, resulting in muscle weakness, loss of muscle tone, fascicul paralysis and hypoventilation
Reduction of Chemical Gradient

The loss of the concentration gradient in hyperkalemia inhibits repolarization of the cardiac muscle. The muscle is then paralyzed in the absence of a greater stimulus.

Hyperkalemia ECG Changes

- **Mild**: Narrowing and peaking of the T waves occurs. Shorten QT
- **Moderate**: PR interval is increased. The P waves are smaller and may be absent. Widening QRS and amplified R wave.
- **Severe**: Ventricular conduction system is impaired and the QRS complex widens. Finally, at near-fatal levels, the QRS complexes continue to widen, and a large undulating sine wave appears.

Evaluation

- No predisposition or ECG changes repeat blood test
- CBC
- Metabolic profile
- Urine potassium, sodium and osmolality
- Renal insufficiency (eGFR)
- Depending upon clinical findings:
  - Glucose level
  - Digoxin level
  - ABG (if acidosis is suspected)
Treatment of Hyperkalemia

1. **Determine Approach** – if emergent or not.
   Frequently management of elevated K+ can occur before it becomes dangerously high.
   However, potential fatal hyperkalemia occurs with K+ > 7.5 and associated with profound weakness.
   THIS CANNOT BE COUNTED ON; cardiac toxicity does not consistently correlate well with plasma K+ concentration.
   MUST OBTAIN STAT ECG.

2. **Determine underlying cause(s)** once patient treated maintain stability
   If K+ high and ECG normal, consider pseudohyperkalemia
   Usually, chronic hyperkalemia is due to impaired K+ excretion
   Review medications, oral and all IV therapies
   Evaluate effective circulating volume
   Patients with kidney disease are highest risk of developing hyperkalemia.

### Hyperkalemia Treatment

**I. Do no harm**
A. Remove any K containing fluids
B. Remove any medications that could be contributing

**II. Stabilize cell membranes: IV calcium**

**III. Drive K back into cells**
A. Insulin and glucose
   B. Albuterol

**IV. Remove excess K from the body**
A. Loop diuretics
B. Cation exchange resin: Sodium polystyrene sulfonate (Kayexalate)
C. Hemodialysis

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
<th>Onset</th>
<th>Length of Effect</th>
<th>Mechanisms of action</th>
<th>Cautions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium gluconate</td>
<td>10 to 20 mL of 10 percent</td>
<td>Immediate</td>
<td>30 minutes</td>
<td>Protects myocardium from toxic effects of calcium</td>
<td>Can worsen Digoxin toxicity</td>
</tr>
<tr>
<td>Insulin</td>
<td>10 units IV with 50 mL of 50 percent glucose</td>
<td>15 to 30 minutes</td>
<td>Two to six hours</td>
<td>Shifts potassium out of the vascular space and into the cells; no effect on total body potassium</td>
<td>Glucose unnecessary if blood sugar elevated above 250 mg per dL</td>
</tr>
<tr>
<td>Albuterol</td>
<td>10 to 20 mg by nebulizer over 10 minutes (use concentrated form, 5 mg per mL)</td>
<td>15 to 30 minutes</td>
<td>Two to three hours</td>
<td>Shifts potassium into the cells, additive to the effect of insulin; no effect on total body potassium</td>
<td>May cause a brief initial rise in serum potassium</td>
</tr>
<tr>
<td>Furosemide</td>
<td>20 to 40 mg IV, give with saline if volume depletion is a concern</td>
<td>15 minutes to one hour</td>
<td>Four hours</td>
<td>Increases renal excretion of potassium; Only effective if adequate renal response to loop diuretic</td>
<td></td>
</tr>
<tr>
<td>Sodium polystyrene sulfonate</td>
<td>Oral: 50 g in 30 mL of sorbitol solution; Rectal: 50 g in a retention enema</td>
<td>One to two hours (rectal route is faster)</td>
<td>Four to six hours</td>
<td>Removes potassium from the gut in exchange for sodium; May cause bowel necrosis and sodium retention</td>
<td>Hollander-Rodriguez J et al. Am Fam Physician. 2006 Jan 15;73(2):283-290.</td>
</tr>
</tbody>
</table>
Binding Resins

- Sodium Polystyrene Sulfonate: Promote the exchange of K+ for Na+ in the GI tract.
- Lower K+ within 1-2 hours of administration with effects lasting 4-6 hours.
- Kayexalate 25-50g mixed with 100 ml Sorbitol PO or PR. Multiple doses usually necessary.

Kayexalate/sorbitol should never be given to someone with hypoactive bowel sounds because fluid shifts can lead to bowel necrosis.

Hemodialysis

- Sometimes the best solution for hyperkalemia, especially in patients with known renal failure, is dialysis. This process simulates the chemical gradient within healthy kidneys and draws out excess potassium.
- Most hemodialysis patients presenting with elevated K+ require dialysis for other electrolyte imbalances as well i.e.: elevated urea, creatinine.

Key Points

- Renal and gastrointestinal pathways are responsible for preservation of potassium homeostasis
- Hyperkalemia is not an uncommon finding in HF trials. Reports vary based on multiple factors.
- HF patients on GDMT can develop hyperkalemia
- Risk Factors such as, increased age, NYHA, renal dysfunction and diabetes
- Ongoing assessment of K+ levels and renal function is critical
Novel Therapy for Hyperkalemia in Persons with HF

<table>
<thead>
<tr>
<th>Novel Agent</th>
<th>Company</th>
</tr>
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<tbody>
<tr>
<td>Cross-linked polyelectrolyte (CLP-1001) Investigation halted</td>
<td>Sorbent Therapeutics, Inc</td>
</tr>
<tr>
<td>Patiromer calcium (RLY 5016) Veltassa®</td>
<td>Relyas, Inc</td>
</tr>
<tr>
<td>FDA approved late 2015 Zirconium cyclosilicate (ZS-9) Sodium Under FDA review</td>
<td>ZS Pharma, Inc</td>
</tr>
</tbody>
</table>


Cross-Linked Polyelectrolyte

- Polyelectrolyte polymers
- Administered orally
- Absorbs both water and electrolytes in the GI tract (potassium and sodium) with elimination in the feces
- Double-blind, randomized, placebo-controlled study
- **Primary Endpoint:**
  - Change in serum K+ from baseline
- **Secondary Endpoints:**
  - Effect on fluid overload, functional capacity, quality of life and blood pressure

Cleland JGF. Late Breaking Clinical Trials. 2014 HFSA


CLP Study Results

**Primary Endpoint:**
- After 8 weeks, no difference in serum K+ between groups
- At Week 4 patients eligible to increase spironolactone was similar between groups
- (64.4% CLP vs. 73.1% Placebo)

**Secondary Endpoints:**
- Weight loss and functional capacity were significantly improved in the CLP group
Patiromer Calcium (RLY 5016)
- Non-absorbed potassium binding organic polymer
- Binds primarily in the colon
- Administered orally
- PEARL-HF Study
- 4 week multi-site, prospective double-blind randomized placebo-controlled pilot study
- **Primary Endpoint:**
  - Mean change of serum K+ from baseline to end of study
- **Secondary Endpoints:**
  - Proportion of patients with serum K+ >5.5 mEq/L at any time
  - Proportion of patients whose spironolactone dose could be increased to 50mg/day


Patiromer (RYL 5016)

**Two-phase trial n=237 OPAL-HK**
- Hyperkalemic Patients with and without HF
  - Randomized Patiromer or placebo
  - Dose was dependent on potassium level (2 dosing levels: 4.2 g or 8.4 g)
  - Sustained decrease in both drug treated patient groups
  - HF (p<0.001) and Without HF (p<0.001)
  - End of treatment phase: 76% of patients had normal K+ level

**Second phase n=107**
- Patients receiving RAASi (K+ >5.5 to <6.5)
  - Reduced K+ 3.8-5.5
  - Randomized and treated for 8 weeks
  - 60% Placebo group K+ >5.5mmol/l, compared to 15% treated group (p< 0.001)


Patiromer (Valtassa®)
- Approved by FDA in late 2015.
- Indication Non critical hyperkalemia (delayed onset of action.
- MOA Non absorbed cation exchange polymer that contains a calcium–sorbitol counter ion.
- Can bind to other medications so must be taken 6 hours pre or post other medications.
- Dose 8.4 grams once daily (can up titrate to 16.8 and 25.2).
- Presumed to have no effect on fetus or breastmilk as not absorbed.
- No renal dosing. (3% of subjects had CKD)
- Caution in patients with decreased gastric motility.
Patiromer (Valtassa®) Side Effects

- Constipation: 7.2%
- Hypomagnesemia: 5.3%
- Diarrhea: 4.8%
- Hypokalemia: 4.7%
- Nausea: 2.3%
- Abdominal discomfort: 2.0%
- Flatulence: 2.0%
- Edema of lips: 0.3%

Zirconium Cyclosilicate (ZS-9)

- Insoluble, non-systemic inorganic cation exchanger currently under review by the FDA

- Preferentially entraps $k^+$ over other divalent ($Ca^{2+}, Mg^{2+}$) and other monovalent ($Na^+$) cations in the GI tract

- Administered orally in doses of 2.5, 5, 10mg

- Two Phase 3 trials showed ZS-9 significantly decreased serum potassium levels among patients with hyperkalemia

Phase 3: Acute Phase

Subjects:
- Hyperkalemia with HF, CKD, DM and on RAASi
- N=753
- Receive [1 of 3] ZS-9 doses for 48hrs
- ZS-9 TID decreased K+ during the first 48 hours regardless of severity of baseline levels
- Serum potassium level reduction was greatest in patients with the highest baseline K+ level
- Mean K+ levels normalized at 4.6 mmol/L in the highest dosing group, compared 5.1 mmol/L placebo
- Diarrhea was the most common adverse event


Effect of ZS-9 withdrawal

Packham DK et al NEJM 2014
ZS-9 Extended Treatment of Hyperkalemic Patients

- N= 258, Phase III, HARMONIZE Trial
- Received ZS-9 10g TID for 48 hours
- Patients whose K+ normalized were randomized to [1 or 3] ZS-9 doses
  or placebo daily for 28 days
- 98% treated with ZS-9 achieved normal potassium levels in 48 hours
- End of the maintenance period 83% of patients receiving (high
dose/15g) had normal K+ levels vs. 48% of patients receiving placebo
- Adverse events were comparable between groups


ZS-9 Extended Treatment of Hyperkalemic HF Patients with 3 Dosing Levels

- N= 94, Phase III, DB, PC Trial
- Received ZS-9 10g TID for 48 hours HF patients who normalized K
  were then randomized to Placebo or doses of 5, 10, or 15 mg ZS-9
  for 28 days of treatment
- K+ was 5.6 prior to treatment but was 4.4 at 48 hours
- End of the maintenance period 83, 89, and 92% of patients
  receiving 5, 10, and 15 mg doses had normal K+ levels vs. 40% of
  patients receiving placebo
- Adverse events were comparable between groups and edema was
  most common.

Anker et al, Eur J HF 2015: 17;1050-1056

Normalization of Serum K+ with ZS-9 in Patients with HF

- 154 HF and HK patients pooled from prior studies
- Subjects received 10 mg tid for 48 hours
- RAAS meds were kept at stable dose for this duration.
- 90% achieved normokalemia 24 hrs and 99% by 48 hours.
- Average time to normokalemia was 2 hours.

Deedwania et al. Journal of Cardiac Failure Vol. 21 No. 8S August 2015
Dietary Counselling: A Role for Self Care in Hyperkalemia

- Healthy adult intake recommendation is 4500 mg daily but most people have considerably less

Foods High in Potassium

- Avocado
- Banana
- Potatoes
- Spinach
- Tomatoes
- Citrus juices
- Fish

Dietary Counseling in Hyperkalemia

- Patients are challenged with dietary restrictions
- Review the role of potassium
- Low potassium 60 mEq or ~2000mg/day
- Obtain a dietary consult
- Recommend/Consider:
  - Smaller portions
  - Avoid salt substitutes
  - Consider food alternatives lower in potassium
  - Soak foods in water or overcook vegetables
Dietary Potassium

- Winter squash, cubed, 1 cup, cooked: 896 mg
- Sweet potato, medium, baked with skin: 604 mg
- Potato, medium, baked with skin: 610 mg
- White beans, canned, drained, half cup: 595 mg
- Yogurt, fat-free, 1 cup: 579 mg
- Halibut, 3 ounces, cooked: 490 mg
- 100% orange juice, 8 ounces: 490 mg
- Broccoli, 1 cup, cooked: 457 mg
- Cantaloupe, cubed, 1 cup: 431 mg
- Banana, 1 medium: 422 mg
- Pork tenderloin, 3 ounces, cooked: 382 mg
- Lentils, half cup, cooked: 366 mg
- Milk, 1%, low fat, 8 ounces: 366 mg
- Salmon, farmed Atlantic, 3 ounces, cooked: 326 mg
- Pistachios, shelled, 1 ounce, dry roasted: 295 mg
- Raisins, quarter cup: 250 mg
- Chicken breast, 3 ounces, cooked: 218 mg
- Tuna, light, canned, drained, 1 ounces: 201 mg

Dietary Potassium Content

<table>
<thead>
<tr>
<th>Foods Low in Potassium</th>
<th>Foods High in Potassium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Applesauce</td>
<td>Baked Potato</td>
</tr>
<tr>
<td>Green beans &amp; Peas</td>
<td>Cod, halibut</td>
</tr>
<tr>
<td>Watermelon</td>
<td>Spinach</td>
</tr>
<tr>
<td>Blueberries</td>
<td>Milk</td>
</tr>
<tr>
<td>Oatmeal</td>
<td>Lima beans</td>
</tr>
<tr>
<td>Rice white or brown</td>
<td>Brussel sprouts</td>
</tr>
<tr>
<td>Spaghetti</td>
<td>Broccoli</td>
</tr>
<tr>
<td></td>
<td>Cantaloupe</td>
</tr>
</tbody>
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For more information: [www.nkdep.nih.gov](http://www.nkdep.nih.gov)
Academy of Nutrition and Dietetics

Diet Question

Menu
- Cod 3 oz
- Baked potato w/ skin (medium)
- Cantaloupe ½ cup

- Would you recommend this selection?
- Which of the foods are high in potassium
- Total potassium 1620 mg
Summary

• HF patients are susceptible to hyperkalemia
• The risk of hyperkalemia can limit the ability to prescribe and optimize the life saving RAASi therapy.
• Early detection and treatment of hyperkalemia among HF patients receiving RAASi therapy is warranted.
• Novel oral cation exchange therapy could potentially improve both acute and chronic management of hyperkalemia in HF.

Question 1. Muscle weakness and cardiac arrhythmias are symptoms that can be seen with

A. hyperkalemia
B. hypokalemia
C. both hyperkalemia and hypokalemia
D. neither hyperkalemia nor hypokalemia
Question 2. Which of the following concerning EKG changes associated with hyperkalemia should worry you THE MOST:

A. Sine wave pattern
B. Tall peaked T waves
C. Loss of P wave with tall peaked T waves
D. Widened QRS

Question 3. What is your first step to assess the following patient scenario

Mr. B is a HF patient NYHA II who has been on optimal therapy for the past 12 months. He has not experienced any change in treatment and reports feeling great. He obtains a blood draw in his PCPs office for routine follow up. His Potassium is reported to be 6.5. What are your next steps?

A. Call patient to come in for IV Calcium
B. Increase his Lasix dose
C. Repeat blood draw
D. Order a Kayexalate enema

Clinical Scenario

- A 52-year-old man with HFpEF and diabetes complains of weakness, nausea, and a general sense of illness, that has progressed slowly over 3 days. His medications include metformin, furosemide, and Lisinopril.
- On examination, he appears lethargic and ill. His BP is 154/105 mm Hg, HR 70bpm, temperature 98.6°F, and respiratory rate 22 breaths/min.
- Physical examination reveals moderate jugular venous distension, some minor bibasilar rales, and lower extremity edema. He is oriented to person and place but is unable to give further history. The ECG shows a wide complex rhythm.
- Laboratory studies performed are significant for K+ 7.8 mEq/L, BUN 144 mg/dL & creatinine 10.5.
ECG Changes of Hyperkalemia

- Easily Distinguished ECG signs:
  - peaked T wave.
  - prolongation of the PR interval
  - ST changes (which may mimic myocardial infarction)
  - very wide QRS, which may progress to a sine wave pattern and asystole.

- Patients may have severe hyperkalemia with minimal ECG changes, and prominent ECG changes with mild hyperkalemia.

- Diagnosis: Hyperkalemia

- Treatment Plan

Serum Potassium Cutoff Values to Define Hyperkalemia Vary Widely in Studies and Guidelines

The upper limit of normal (ULN) for serum K+ levels varies across guidelines and publications. Serum K+ levels of 5.5, 5.0, or 6.0 mEq/L are commonly used cutoffs for hyperkalemia.
Guidelines Recommend RAASi Dose Modifications With Increasing Serum K+

Serum K+ (mEq/L)

< 5.5

Most conservative

Most aggressive

ESC HFA, K/DOQI:
Reduce dose of/stop ACEi/ARB, MRA if >5.5

ACC/AHA HF1:
MRA not recommended >5.0

HFSA HF3: MRA not recommended >5.0

NICE5: don't start RAASi if >5.0

Stop RAASi if >6.0

Consider Other Cases

- What can be done for the patient with moderate HK?
- What can be done for long term therapy to enable patients to reach adequate doses of RAASI?