EM CASE OF THE WEEK

BROWARD HEALTH

Author: Trevor Smith | **Editor:** Dr. January Moore

October 2, 2022 | Vol 13 | Issue 1

Hyperkalemic Emergency

A 49-year-old male with past medical history of hypertension, heart failure with preserved ejection fraction, hyperlipidemia, chronic kidney disease and obesity presented to the ED with nausea, vomiting, and diarrhea for 2 days. He denies any chest pain, shortness of breath, fever, chills, or abdominal pain. Patient is afebrile on exam with blood pressure of 170/90 mmHg and heart rate 78 bpm. On physical exam, patient is alert with regular rate and rhythm, clear to auscultation bilaterally, and a soft, nondistended abdomen. EKG showed peaked T waves and prolonged QRS complexes.

Initial CMP revealed the following: Sodium: 132, Potassium: 8.2, Chloride: 93, Anion Gap: 29
BUN: 232, Creatinine 25.1, GFR of 2, Lipase: 248.
Repeat CMP to confirm accuracy showed the following: Sodium: 132, Potassium 7.7, Chloride 94, Anion Gap: 29, BUN: 238, Creatinine 24.9, GFR of 2.

Which of the following is the most appropriate next step of management?

A: Loop diuretics

B: Calcium gluconate, Insulin, D50, and Nephrology consult

C: Patiromer

D: Calcium gluconate and D50

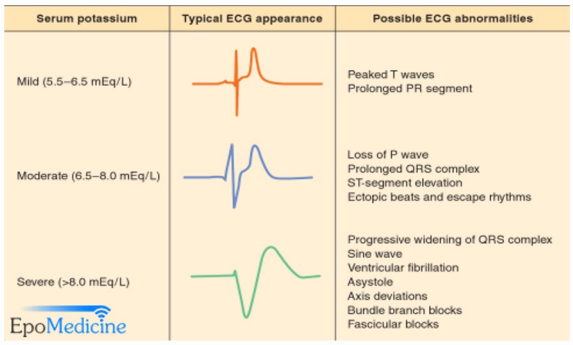

Figure 1. EKG Rhythm in Hyperkalemia. https://epomedicine.com/emergency-medicine/ecg-changes-hyperkalemia/

Clinical Pearl

Emergent EKG can show conduction abnormalities due to hyperkalemia, however up to 50% of patients with hyperkalemia will have no indications on EKG.

Department of Emergency Medicine
1600 S. Andrews Avenue
Fort Lauderdale, FL 33316

BROWARD HEALTH MEDICAL CENTER
Emergency Department

The correct answer is B. The patient is presenting with hyperkalemic emergency with potassium of 8.2 and 7.7 on repeat. For patients with severe kidney disease, nephrology consult is warranted to discuss candidacy for emergent hemodialysis.
A. Loop diuretics are not appropriate monotherapy for a patient with hyperkalemic emergency.
C. Patiromer is a cation exchanger that increases potassium excretion via feces. This is not an appropriate monotherapy.
D. Calcium gluconate must be given for hyperkalemic emergency. However, glucose alone is not recommended as the body's endogenous release of insulin is variable. Insulin and glucose are needed together to drive potassium into cells while preventing hypoglycemia.

Discussion

Hyperkalemic emergency is defined by a serum potassium > 6.0 mEq or a sudden increase in potassium 1.0 mEq above 4.5 in 24 hours.[1] Associated symptoms include neuromuscular weakness, loss of deep tendon reflexes, and/or cardiac arrhythmia.

Hyperkalemia can be caused by a variety of factors. Common causes of hyperkalemia include acute and chronic kidney disease, potassium sparing diuretics, ACE inhibitors, Bactrim. Endocrine disorders such as hypocortisolism and hypoaldosteronism can lead to an increase in potassium. Type 4 renal tubular acidosis can precipitate hyperkalemia, in addition to hyperchloremic metabolic acidosis with normal anion gap.[2]

Diagnosis is confirmed with laboratory studies. EKG should be performed promptly to evaluate for any changes in cardiac conduction. EKG follows a typical progression: tall peaked T waves, short QT interval, lengthening of PR interval, loss of p waves, widening of the QRS complex, and eventual sine wave.[3-4] The sine wave is an EKG finding indicating worsening cardiac function as a result of the fusion of the QRS complexes and T waves with loss of P waves.

Possible conduction abnormalities seen with hyperkalemia include bundle branch blocks and AV blocks. Hyperkalemia can also lead to varying arrhythmias, which can include sinus bradycardia, ventricular tachycardia, fibrillation, and even asystole.[5]

Potassium level (mmol/L)	Mechanism	ECG changes
5.5 – 6.5	Repolarisation abnormalities	Peaked T waves
6.5 – 7.0	Progressive atrial paralysis	P wave widening/flattening PR prolongation P waves eventually disappear
7.0 – 9.0	Conduction abnormalities	**Bradyarrhythmias:** Sinus bradycardia; high-grade AV block with slow junctional and ventricular escape rhythms; slow AF Conduction blocks (bundle branch block, fascicular blocks) Prolonged QRS interval with bizarre QRS morphology
> 9.0	All of above	Development of sine wave appearance (pre-terminal rhythm) Asystole Ventricular fibrillation PEA with bizarre, wide complex rhythm

Figure 2. Potassium Level and Effect on EKG. Courtesy of LITFL. https://litfl.com/hyperkalaemia-ecg-library/

Treatment

First step in treatment is giving calcium gluconate 1000 mg or calcium chloride 500-1000 mg IV over a few minutes. The role of calcium in hyperkalemia is to stabilize the cardiac membranes.

Hyperkalemic emergency patients should receive insulin and glucose in addition to the calcium gluconate/chloride. A base regimen is 10 units of insulin and D50 infused over 5 minutes. The role of insulin is to shift extracellular potassium into the cells. The glucose functions to prevent subsequent hypoglycemia due to the administration of insulin. Sodium bicarbonate is another option for shifting potassium intracellularly and is used for concurrent metabolic acidosis. Albuterol, a beta-2 adrenergic agonist, also functions to shift potassium into the cells. The use of sodium bicarbonate and albuterol is at clinician discretion.

Removing potassium from the body can be accomplished in a few ways. Loop or thiazide diuretics can be used if the patient has stable kidney function. A gastrointestinal cation exchanger like Patiromer can be used to promote excretion of potassium in the feces. The final method of removal is hemodialysis. This is the preferred method in patients with severe hyperkalemia and chronic kidney disease or severely impaired kidney function.

Treatment (continued)

Trialysis catheter placement is indicated for venous access for dialysis. Ultrasound is used for identification of the anatomy, with caution given to a femoral artery overlying the vein. Once identified, a guidewire is inserted into the vein. Multiple skin dilators are used to enlarge the opening so the catheter will slide over the guide wire. Once in place, the catheter is sutured to the skin. Some contraindications to the procedure include Infection, obstruction and site-specific trauma.[6]

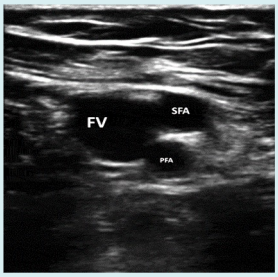

Figure 3. Ultrasound image of femoral artery and vein anatomy. https://www.structuralheartjournal.org/article/S2474-8706%2822%2900191-9/fulltext

Our patient was diagnosed with hyperkalemia secondary to AKI on chronic kidney disease and pancreatitis. He was given calcium gluconate, insulin and D50. A trialysis catheter was placed in the ED for emergent dialysis and the patient was admitted. He was discharged a week later with resolution of hyperkalemia after three hemodialysis sessions.

Take Home Points

- **Hyperkalemic emergency is a serious and life-threatening condition.**
- **Prompt diagnosis and lab evaluation should be performed.**
- **EKG may show cardiac conduction changes.**
- **Prompt treatment with calcium gluconate, insulin, and D50.**
- **Consult nephrology for severe cases or cases of hyperkalemia in kidney disease patients.**
- **Bedside catheter placement in ER is a practical and safe procedure when utilized with ultrasound guidance in preparation for emergent dialysis.**

About the Author

The author of this case is Trevor Smith. Trevor is a 4th year medical student from NSU-KPCOM. He did his emergency medicine rotation at BGMC August-September 2022. He plans on pursuing a career in Emergency Medicine after graduation.

References

1. Montford, J. R., & Linas, S. (2017). How Dangerous Is Hyperkalemia? Journal of the American Society of Nephrology, 28(11), 3155–3165. https://doi.org/10.1681/asn.2016121344
2. Schambelan, M., & Sebastian, A. (1985). [Type IV renal tubular acidosis: pathogenetic role of aldosterone deficiency and hyperkalemia]. Nephrologie, 6(3), 135–137. https://pubmed.ncbi.nlm.nih.gov/3908957/#:~:text=Type%20IV%20renal%20tubular%20acidosis%20(RTA)%20is%20a%20syndrome%20
3. Diercks, D. B., Shumaik, G. M., Harrigan, R. A., Brady, W. J., & Chan, T. C. (2004). Electrocardiographic manifestations: electrolyte abnormalities. The Journal of emergency medicine, 27(2), 153–160. https://doi.org/10.1016/j.jemermed.2004.04.006
4. Levis J. T. (2013). ECG diagnosis: hyperkalemia. The Permanente journal, 17(1), 69. https://doi.org/10.7812/TPP/12-088
5. Mattu, A., Brady, W. J., & Robinson, D. A. (2000). Electrocardiographic manifestations of hyperkalemia. The American journal of emergency medicine, 18(6), 721–729. https://doi.org/10.1053/ajem.2000.7344
6. Beecham, G. B., & Aeddula, N. R. (2021, December 25). Dialysis Catheter - StatPearls - NCBI Bookshelf. Dialysis Catheter - StatPearls - NCBI Bookshelf. https://www.ncbi.nlm.nih.gov/books/NBK539856/#:~:text=Tunneled%20cuffed%20catheters%20are%20inserted,15cm%20in%20length%20are%20appropriate.
7. Epomedicine. ECG changes in Hyperkalemia. Epomedicine; 2013 Nov 9 Available from: https://epomedicine.com/emergency-medicine/ecg-changes-hyperkalemia/.
8. Buttner, R., Burns, E., & and Ed Burns, R. B. (2018, August 1). Hyperkalaemia ECG changes • LITFL • ECG Library. Life in the Fast Lane • LITFL. https://litfl.com/hyperkalaemia-ecg-library/
9. Soud, M. (2018, July). Should Ultrasound Guidance Be Routinely Used for Femoral Artery Access. Structural Heart Journal. https://www.structuralheartjournal.org/article/S2474-8706%2822%2900191-9/fulltext

@browardem

Faculty Editors | Meena Hierholzer, MD & Jason Mansour, MD
Editor-in-Chief | Matthew Holme, MD
Managing Editor | January Moore, DO

BROWARD HEALTH MEDICAL CENTER
Emergency Department