

A harmful banana?

The purpose of this case study is to review the pathophysiology, preanalytical factors, and treatment for a condition.

By Michael Lazzari, MS, BS(CLS), MT(ASCP)

A morbidly obese 63-year-old, diabetic Caucasian woman was brought to the emergency room on May 17, 2007, after falling and hitting her head while taking a shower. At the time of the fall, she noted muscle weakness, heartburn, and a blood sugar of 80 with no loss of consciousness. She said she had no apparent signs or symptoms for the past few days. (For her past medical history, physical exam, and surgeries, see Table 1.)

Table 1.

Her past medical history showed:	Upon physical examination on May 17th, the doctor noted:	Here past surgical history included:
Mild chronic renal insufficiency	Morbidly obese without distress	Cardiac catheterization
Coronary artery disease	Normal vital signs	Right and left knee arthroplasty
Asthma	Clear lungs	Anterior cervical discectomy with fusion
Obstructive sleep apnea	Edema in lower extremities	Tonsillectomy
Morbid obesity		Adenoidectomy
Hypertension		Appendectomy
Diabetes mellitus		Benign tumor removal from bowel
Venous insufficiency		Umbilical hernia repair
Varicose veins		
Lymphedema in lower extremities		
Degenerative joint disease		
Anemia		
Depression		
Anxiety		
Gastroesophageal reflux disease		

The patient had no history of using illicit drugs or alcohol. She had been placed on a strict diet recently which included lots of fruit (apples, pears, and bananas) and vegetables (lettuce and tomatoes). She was, however, on 13 outpatient medications: Lantus, Singulair, aspirin, Niferex, Toprol, Lasix, Zestril, Lipitor, propoxyphene, Aldactone, Plavix, Prilosec, and Cardizem, as well as home oxygen. She cited allergies to codeine, Celebrex, sulfa, Vioxx, and penicillin. Upon admission as an inpatient, she was prescribed an additional 15 medications.

The patient's laboratory results are shown in Tables 2

and 3. The physician ordered a basic metabolic panel for six days, cardiac labs, and a complete blood count with differential on admittance. The results from the basic metabolic panel showed a decreased sodium, an increased potassium, and a decreased CO₂ on admittance. A decreased GFR, an increased glucose, creatinine, and BUN also were seen on admittance. The remaining tests showed normal cardiac labs and a normocytic/normochromic anemia. The sodium and potassium levels were repeated by a direct potentiometric method. Questions to consider were 1) what was the cause of this woman's fall and muscle weakness; 2) what conditions in her past could have lead to this present condition; and 3) what laboratory results presented support the diagnosis of this condition?

Table 2 - Important Basic Metabolic Panel Results

	Normal Values	May 17 th	May 18 th	May 19 th	May 20 th	May 21 st	May 22 nd
Sodium	135-145 (mmol/L)	130	137	138	138	138	139
Potassium	3.5-5.2 (mmol/L)	7.9	5.8	5.4	5.1	4.8	4.4
Chloride	95-107 (mmol/L)	103	108	109	109	108	108
CO ₂	21-31 (mmol/L)	18	19	21	23	22	18

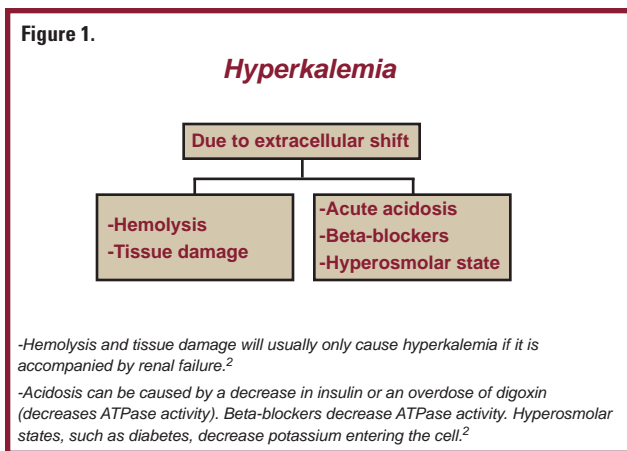
Table 3 - Miscellaneous Tests on May 17th

Test	Test Results	Normal Range
Total CK	40 mU/mL	30-150 mU/mL
CK-MB	2.6 ng/mL	<5.0 ng/mL
CK-MB Index	1.8	<2.3
Troponin I	<0.1 ng/mL	<0.3 ng/mL
B-Type Natriuretic Peptide	27 pg/mL	<100 pg/mL
GFR	30	45-104
Creatinine	1.8 mg/dL	0.7-1.5 mg/dL
Glucose	123 mg/dL	65-110 mg/dL
BUN	97 mg/dL	8-22 mg/dL
CBC with differential	Normocytic/ Normochromic anemia	
Iron Studies	Normal	

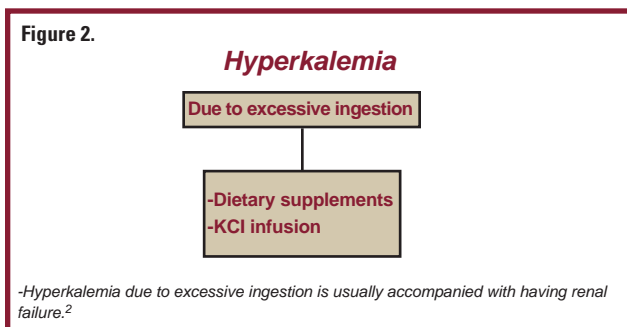
Importance of potassium in the body

Potassium is important for cell maintenance, cellular-membrane potential, and homeostasis of cell volume.¹ Potassium is eliminated from the body through the gastrointestinal tract and kidneys. Renal elimination is passive through the glomeruli of the kidney, while there is active

reabsorption of potassium, along with sodium and chloride, in the proximal tubule and ascending limb. There is excretion of potassium in the distal tubule and collecting duct, which is controlled by aldosterone.² Other factors that regulate distal tubular secretion of potassium include intake of sodium and potassium, water flow rate in the distal tubule, and acid-base balance.³



There are two mechanisms for potassium regulation in the human body. One course is that ingested potassium enters the circulation, stimulating the pancreas to release insulin. Elevated insulin levels cause transport of potassium into cells via Na⁺/K⁺ ATPase. This pump, by active transport, moves three sodium ions out of the cell for every two potassium ions that move into the cell. If increased potassium is present in the circulation, another route involves the kidneys releasing renin. This stimulates hepatic activation of angiotensin I that is converted to angiotensin II in the lungs. Angiotensin II stimulates the adrenal gland to secrete aldosterone, which causes renal cortical collecting ducts to excrete potassium and retain sodium.⁴

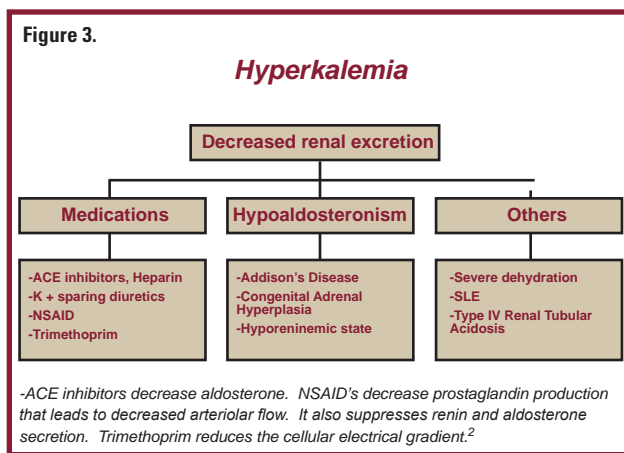


Pathophysiology

Serum/plasma potassium levels are only an approximate indicator of total body potassium. Hyperkalemia is a concentration of serum potassium above the reference limit of 5.0 mmol/L. This can be caused by redistribution (shift of potassium out of the cells), increased potassium intake, or increased retention (reduced renal potassium excretion). Figures 1, 2, and 3 outline each cause of hyperkalemia. Hyperkalemia can be a life-threatening condition, and its diagnosis should be made from a clinical history, re-

view of medications, and a physical exam. Most of these conditions are seen in premature babies and the elderly.³ Three mechanisms causing the condition of hyperkalemia are reduced aldosterone and response, renal failure, and reduced distal delivery of sodium.²

This patient had chronic renal failure, was taking prescription drugs (Zestril is an ACE inhibitor, Toprol is a beta-blocker, and Aldactone inhibits aldosterone), ate a large volume of food that contained potassium (bananas), and had diabetes. All these conditions contributed to her hyperkalemia. Her increased serum potassium level on admittance supports her diagnosis of hyperkalemia. Her creatinine, GFR, and BUN results support her condition of renal failure.



When a patient has hyperkalemia, the signs and symptoms that are usually present include fatigue, weakness, tingling, numbness, paralysis, palpitations, difficulty breathing, and mental confusion.⁵ Cardiac effects can be seen due to bradycardia and on the ECG there will be a prolonged PR and QRS intervals with peaked T waves.³ The patient in this case had fatigue and muscle weakness but did not undergo an ECG.

Pre-analytical causes

Prior to running a specimen for a potassium level, there are causes that can create a factitious or pseudohyperkalemia reading, it is important to recognize these factors. These include leukocytosis (>50,000/mm³), thrombocytosis (>10⁶/mm³), and erythrocyte (RBC) hemolysis (Note: The samples in this case were not hemolyzed.) When these cells break down, they release potassium into the circulation, thus increasing the serum potassium level (make sure rhabdomyolysis is not the underlying cause). Pre-analytical causes can also be due to improper collection (e.g., excessive fist clenching, incorrect order of draw [K⁺ EDTA may be back flushed into the serum separator tube] and collecting above an IV line of a potassium infusion). Sample processing errors may include centrifuging (more than once) and refrigeration.^{6,7}

Treatment

In this case, the patient was treated with dextrose, insulin, and Kayexalate. The dextrose and insulin temporarily shift

potassium back into the cells, and the Kayexalate exchanges sodium for potassium and binds it in the large intestine. Other options for treatment include calcium supplementation (decreases myocardial excitability), bicarbonate therapy (stimulates Na⁺/K⁺ ATPase), Salbutamol (B2-selec-

tive catecholamine which promotes K⁺ into cells), dialysis, diuretic, diet low in potassium, and discontinuation of certain medications.⁵

Summary and case resolution

Hyperkalemia is a serum potassium concentration greater than 5.0 mmol/L.

The following causes contributed to this patient's hyperkalemia: chronic renal failure, prescription drugs (Zestril, Toprol, and Aldactone), increased potassium in food, and diabetes. Zestril is an ACE inhibitor that decreases aldosterone synthesis. Toprol is a beta-blocker that decreases Na⁺/K⁺ ATPase activity. Aldactone inhibits binding of aldosterone to receptors in the renal tubule.

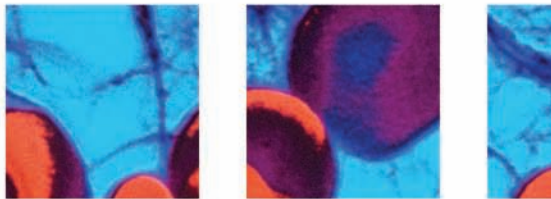
The patient had no history of using illicit drugs or alcohol. She had been placed on a strict diet recently which included a lot of fruit (apples, pears, and bananas) and vegetables (lettuce and tomatoes).

Pre-analytical causes include factitious (pseudohyperkalemia), collection errors, or sample processing errors. Hyperkalemia can be physiologically caused by redistribution, increased potassium intake, or increased retention. This condition is usually treated with dextrose/insulin, Kayexalate, calcium, bicarbonate, diuretic, low potassium diet, dialysis, or discontinuation of medication. □

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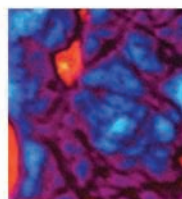
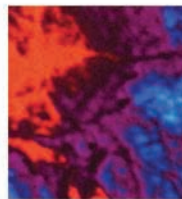


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