

CLINICAL VIGNETTE

Hypokalemia – Unusual Presentations in Primary Care

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Case 1

A 39-year-old female with no significant past medical history presented for urgent evaluation of left arm numbness and muscle weakness for one day. She denied recent trauma or similar symptoms in the past. She was not taking any medications and has no known drug allergies. Social history is negative for tobacco, alcohol, or illicit drug use, and family history was non-contributory.

The vitals included blood pressure 126/80, pulse 63, temperature 98°F (36.7°C), respirations 17 per minute, oxygen saturation 97%, and BMI 22.5. Physical exam was remarkable only for bradycardia and decreased sensation in C5-C6 distribution of the left arm. She had normal muscle bulk. EKG showed sinus bradycardia with rate of 48, normal axis, and normal intervals (Figure 1). Neck X-ray was unremarkable; she was scheduled for a NCS/EMG with neurology.

Her initial laboratory tests included sodium 143, potassium 2.8, chloride 105, CO₂ 27, BUN 8, creatinine, 0.5, GFR > 89, and glucose 96. The patient was started on potassium supplementation. Repeat testing one week later showed potassium had increased to 3.0 with normal magnesium of 1.7.

Upon further questioning, the patient disclosed that she was drinking a beverage consisting of green tea and taking garcinia cambrosia supplements in order to lose weight. After the patient stopped taking these supplements for only several days, her potassium improved to 3.4. The differential diagnosis for her hypokalemia includes caffeine-induced hypokalemia.

Case 2

A 32-year-old male with past medical history significant for Hodgkin's Lymphoma diagnosed 7 years ago, currently in remission, presented to establish care. At the time of initial visit, he had no acute complaints and was not taking any medications, either prescription or over the counter. He reported drinking two to three beers per weekday, possibly more on weekends; however, he denies CAGE symptoms. He also denies tobacco use or other illicit/recreational drug use. Family history is notable for his father with sick sinus syndrome requiring pacemaker and paternal grandfather with diabetes mellitus type 2 and myocardial infarction at age 67.

His vital signs included blood pressure 120/80, temperature 98.3°F, respirations 18 per min, and oxygen saturation 98%. His BMI is 26.6. Physical exam was unremarkable.

Initial laboratory data revealed an anion gap acidosis and metabolic alkalosis with sodium 144, potassium 2.9, chloride 96, CO₂ 32, BUN 17, creatinine 0.9, GFR >89, glucose 102, and magnesium 1.5. (Anion gap was 12.) Repeat labs were similar with EKG showing normal sinus rhythm, no T-wave abnormalities, and normal intervals.

Upon further questioning, he revealed he had a prior diagnosis of chronic hypokalemia, which was treated with spironolactone. According to the patient, an extensive evaluation was performed but no etiology found. The spironolactone was discontinued due to hypotension. He did not follow-up and never took potassium supplements.

His caffeine intake includes about 2-4 cups of regular coffee per day. He was advised to limit his intake of caffeinated beverages. With aggressive potassium and magnesium supplementation, discontinuation of all caffeinated beverages, and reduction of his alcohol intake, his potassium did increase but never surpassed 3.2. He has since then been evaluated by nephrology. The differential diagnosis for his hypokalemia and hypomagnesemia are Gitelman and Bartter syndrome.

Discussion

The most common causes of hypokalemia in the primary care setting are gastrointestinal or urinary losses due to vomiting, diarrhea, or diuretic therapy. Another cause is redistributive hypokalemia in which potassium transiently enters the cells (for example, with the administration of insulin or a non-selective beta adrenergic agent). Hypomagnesemia must also be excluded as a cause. Manifestations of hypokalemia include severe muscle weakness, cardiac arrhythmias, renal abnormalities, and glucose intolerance.

Cardiac arrhythmias and ECG abnormalities can be present in those with hypokalemia. Most commonly these include premature atrial and ventricular contractions, sinus bradycardia, paroxysmal atrial, junctional tachycardia, atrioventricular block, and ventricular tachycardia or fibrillation. On ECG, there may be a depression of the ST

segment, decrease in amplitude of the T wave, prolongation of the QT interval, and an increase in the amplitude of the U wave, which occurs at the end of the T wave. U waves are often seen in leads V4-V6.

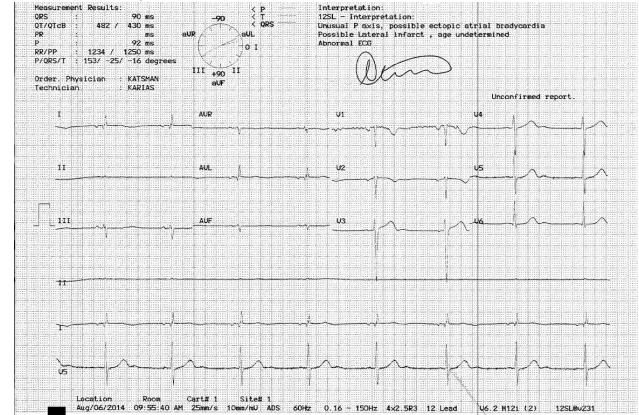
Severe hypokalemia from excessive tea or caffeine consumption is rare. A literature review yielded two case reports of severe hypokalemia from excessive drinking of green tea extract,¹ and hypokalemia associated with herbal tea consumption.² Another case reported a woman developing life-threatening arrhythmia from severe hypokalemia after consuming tea with licorice.³ Discontinuing excessive tea consumption resulted in the correction of the hypokalemia in these cases.

Gitelman syndrome and Bartter syndrome are rare autosomal recessive disorders with a characteristic set of metabolic abnormalities, which include hypokalemia, metabolic alkalosis, elevated plasma renin, hyperaldosteronism, and, in some patients, hypomagnesemia.⁴ The incidence of Gitelman syndrome is 1 in 40,000 people and the incidence of Bartter syndrome is 1 in 1,000,000 people. The tubular defects in sodium chloride transport are almost identical to that seen with chronic ingestion of a loop diuretic (mimicking Bartter syndrome) or a thiazide diuretic (mimicking Gitelman syndrome). The hyperaldosteronism associated with Bartter and Gitelman syndrome is a secondary form of hyperaldosteronism that results from a volume contraction induced increase in renin. These patients are not hypertensive. Treatment of both Gitelman syndrome and Bartter syndrome include NSAIDs (non-steroidal anti-inflammatory drugs), potassium-sparing diuretics, ACE inhibitors, and potassium and magnesium supplementation.

In the primary care setting, hypokalemia is common as it is often a manifestation of diuretic therapy or resulting from an acute gastrointestinal or genitourinary condition. When these common causes are ruled out, one must explore for the more rare causes and ask about supplement and caffeine intake. Once all other causes are ruled out, then one must consider

genetic causes of potassium loss in the kidneys, as with Gitelman and Bartter syndromes.

Figure 1: EKG showing sinus bradycardia with rate of 48, normal axis, and normal intervals.



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