Paroxysmal Paralytic Attacks Secondary to Excessive Cola Consumption

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We report a rare case of cola-induced hypokalemia presenting as recurrent attacks of lower extremity weakness and falls. Excessive consumption of cola-based drinks has been associated with dental enamel erosion, obesity, and diabetes. There are very few published cases depicting the correlation between cola drinks and hypokalemic manifestations. In our patient an extensive workup was performed, and common causes were ruled out before making the diagnosis of cola-induced hypokalemia. Improvement in the patient's symptoms and electrolyte levels after reducing the consumption of cola-based drinks further confirmed our diagnosis. This case also emphasizes the importance of obtaining a detailed history and consideration of broad differential diagnoses in identifying uncommon but reversible etiologies.

Keywords: cola; hypokalemia; paralysis; rhabdomyolysis

Consumption of cola-based soft drinks has been increasing over the last few years. Per capita consumption of cola-based drinks is nearly twice the per capita consumption of milk, and the proportion of persons consuming these beverages has increased in all age groups.¹ Excessive consumption of these cola-based drinks has been associated with multiple adverse effects including erosion of dental enamel, bone demineralization, formation of kidney stones, increasing trends in obesity, and diabetes mellitus.²⁻⁶ Here, we describe a case of an elderly male presenting with paroxysmal attacks of weakness for the last few years. He had multiple hospitalizations in the past for similar complaints, and during each presentation he was found to have hypokalemia. His symptoms improved promptly after replenishing his serum potassium.

Case Report

An African American male, aged 65 years, presented with the chief complaint of episodic paroxysmal lower limb paralysis, describing it as “both legs giving way.” He reported having multiple such episodes in the last 2 to 3 years, which lasted around an hour with no specific precipitating or relieving factors identified by him. He denied any other associated symptoms. He did not have any other significant medical conditions in the past and was not taking any prescribed, over the counter, or herbal medication. He did not recall any of his family members having similar manifestations. On presentation, the patient had mildly decreased tone and strength in his lower extremities bilaterally. His initial laboratory tests were significant for potassium of 1.9 mmol/L and elevated creatine kinase (CK) levels up to 3800 U/L. His electrocardiogram on admission was remarkable for ventricular rate of 61 beats per minute, prolonged corrected QT interval, QRS widening, and U waves merging with T waves in anteroseptal leads (figure 1). He had normal complete blood count, liver function, renal function, and thyroid

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Figure 1. EKG performed at patient's admission to the hospital.

function tests. Serum renin (2.1 ng/mL) and aldosterone level (5.6 ng/dL) were also in the normal range. Magnetic resonance imaging of the spine revealed no compression, demyelination, or any other significant pathology. Evaluation of urine electrolytes was done within 24 hours of admission, which revealed normal urine potassium (26 meq/L/d) but a low transtubular potassium gradient of 2.7. In absence of other obvious etiologies for his presentation, a detailed dietary history was obtained which revealed that the patient had been drinking up to 3 to 4 liters of cola daily over the last 2 to 3 years. He obtained most of his daily calories from chicken-based foods and sugar-sweetened beverages. The contents of the cola-based drink he normally consumed included carbonated water, sugar, color, phosphoric acid, and caffeine.⁷ None of his symptoms had preceded this heavy cola-based soft drink consumption. The patient was admitted to the medicine floor with telemetry monitoring. He was given 120 meq of intravenous potassium chloride (KCl), along with 120 meq of oral KCl solution in the first 24 hours (his serum electrolytes were checked every 6 hours). This was followed by 100 meq of oral potassium over the next 24 hours. He was also kept off cola-based drinks with sustained improvement in his potassium levels and his clinical symptoms. Dietary counseling was provided with emphasis on restriction of cola-based drinks consumption. On follow up in the clinic after 3 months, he had reduced his daily cola consumption to approximately 200 to 300 ml per day. He remained asymptomatic, and his serum potassium level was 3.9 mmol/L.

Discussion

Hypokalemia has been shown to affect muscle, nerve, and

cardiac physiology. Electrochemical gradient of potassium between the intracellular and extracellular space is essential for muscle and nerve function. Potassium influx plays an important role in cell membrane repolarization after passing of an action potential. Decreased potassium levels in the extracellular space causes hyperpolarization of resting membrane potential, resulting in the requirement of greater than normal stimulus for membrane depolarization to initiate an action potential.⁸⁻¹⁰ Rhabdomyolysis caused by low serum potassium levels has been attributed to loss of effect of potassium in increasing blood flow to the muscles.¹¹

Due to the peculiar presentation in this case, multiple differentials including thyrotoxic hypokalemic paralysis, adrenal pathology leading to hypokalemia, drug/diuretic induced hypokalemia, hypokalemia secondary to recent gastrointestinal infection (diarrhea/vomiting), renal potassium wasting syndromes (such as Gitelman/Barter syndrome), and familial hypokalemic periodic paralysis were considered.¹² Extensive work up revealed normal serum magnesium, thyroid stimulating hormone, triiodothyronine 3, triiodothyronine 4, aldosterone, and renin levels. Renal function and liver function were also normal. Spinal cord imaging was unremarkable for any pathology. Low transtubular potassium gradient showed that there was not excessive urinary loss of potassium making conditions such as Barter or Gitelman syndrome unlikely. Due to the late onset of symptoms, in his 60s, (consistent with the time of initiation of excessive cola consumption) and absence of any pertinent family history, genetic testing for familial periodic paralysis was deferred. Clinical improvement with reduction in consumption of cola-based drinks was a testament to the

Table 1. Comparison of salient features of case reports on cola induced hypokalemia.

Author	Age (years)	Gender	Amount of cola consumed (L/day)	Duration of cola consumption	K levels at the time of admission (mmol/l)	Urine K (mmol/l/dl)	Renin (ng/ml/h)	Aldosterone (ng/dl)	Followup K after stopping/reducing cola (mmol/l)
Present case	65	M	3-4	2-3 years	1.9	26	2.1	5.6	3.8
Sharma et al ¹⁸	58	M	6-7	Several years	2.4	-	-	-	N
Packer et al ¹⁹	52	M	4	Several years	3.0	8.6	0.33	4.8	3.5
Kasap et al ²⁰	16	M	>1	2-3 years	2.2	31	2.1	15	N
Chaudhry et al ²¹	21	F	4	8 months	2.2	11	-	-	-
Mudge et al ²²	44	M	4-10	3 years	1.4	17.4	N	N	N
Appel et al ¹⁶	24	F	6-7	10 months	2.0	41.9	12.9	3.6	N
Rice et al ¹⁴	21	F	8	1-2 years	1.8	9	-	-	N
Lee et al ¹³	52	M	4-9	1.5 month	2.3	6.6	N	N	4.2
Matsunami et al ¹⁵	21	F	3	6 years	1.9	-	-	0.67	N

N = value within normal range; not specified in the case report. K=Potassium; M=male, F=female

importance of a detailed history and clinical approach to the diagnosis.

Excessive cola consumption has been associated with both hypokalemia and myopathy in previous studies.^{13,14} Matsunami et al¹⁵ described the first case of this underappreciated etiology of hypokalemia. Few of the case reports described hypokalemia secondary to cola consumption in pregnant females.¹⁵⁻¹⁷ Sharma et al¹⁸ reported a case of 58-year-old male consuming 6 to 7 liters of cola per day. He presented to the hospital with severe generalized weakness and significantly low serum potassium.

The mechanism of hypokalemia secondary to excessive cola consumption can be attributed to increased renal wasting of potassium. This occurs due to caffeine-induced diuresis and increased renin release. High caffeine content in cola causes downstream elevation of cyclic adenosine monophosphate (cAMP) and intracellular potassium shifts. The stimulation of β -adrenergic receptors by caffeine leads to respiratory alkalosis, causing intracellular potassium migration. It has also been postulated that elevated glucose levels secondary to excessive cola consumption lead to osmotic diuresis, leading to loss of potassium in urine. Hyperinsulinemia subsequent to hyperglycemia also causes intracellular migration of potassium. The fructose load from the cola drinks acts as an osmotic agent in the gastrointestinal tract, leading to osmotic diarrhea, subsequently causing potassium and other electrolyte losses.¹⁷ Due to lack of a balanced diet (lack of consumption of fruits or other dietary ingredients rich in potassium) in our patient, there was insufficient repletion of potassium along with the excessive potassium loss.

Conclusion

In patients presenting with hypokalemia without any obvious cause, a detailed history of consumption of caffeinated or other hyperosmolar soft drinks must be obtained to determine this uncommon but reversible etiology.

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