Paroxysmal paralytic attacks secondary to excessive cola consumption

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Abstract:
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 work up 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 detailed history and broad differential diagnosis in identifying uncommon but reversible etiologies.

Introduction:
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. [1] Proportion of persons consuming these beverages has increased in all age groups [1]. Excessive consumption of these 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, 3, 4, 5, 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. During each presentation he was found to have hypokalemia. His symptoms improved promptly after replenishing serum potassium.

Case report:
65 year-old African American male presented with chief complaint of episodic paroxysmal lower limb paralysis, describing it as “both legs giving way”. He reported having multiple such episodes in the last 2-3 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. As per him none of his family members had similar manifestations. On presentation, the patient had mildly decreased tone and strength in his lower extremities bilaterally. His initial labs were significant for Potassium of 1.9 mmol/L & elevated creatine kinase (CK) levels up to 3800 Units/L. Patient’s EKG 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 antero-septal leads. (Figure: 1) He had normal complete blood count, liver function, renal function and thyroid function tests. Serum renin (2.1ng/ml) & aldosterone level (5.6ng/dl) were also in the normal range. MRI 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 (26meq/L/d) but a low trans-tubular 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 3-4 litres of cola daily over the last 2-3 years. He obtained most of his daily calories from chicken based foods and sugar sweetened beverages. Contents of cola based drink consumed by him included carbonated water, sugar, colour, phosphoric acid and caffeine. [7] None of his symptoms had preceded this heavy cola based soft drink consumption. Patient was admitted to the medicine floor with telemetry monitoring. He was given 120 meq of IV KCl, along with 120 meq of oral KCl solution in the first 24 hours, with Q 6 hour serum electrolyte check. This was followed by 100 meq oral potassium in the next 24 hours. He was also kept off cola based diet with sustained improvement in his potassium levels and his clinical symptoms. Dietary counselling was provided with emphasis on restriction of cola based drinks consumption. On follow up in clinic after three
months, he had cut down on daily cola consumption to approximately 200-300 ml per day. He remained asymptomatic and his serum potassium level was 3.9mmol/L.

Discussion:
Hypokalemia has been attributed 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 repolarisation after passing of an action potential. Decreased potassium level in the extracellular space causes hyperpolarisation of resting membrane potential resulting in requirement of greater than normal stimulus for membrane depolarization in order to initiate an action potential [8, 9, 10].

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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 GI infection (diarrhea/vomiting), renal potassium wasting syndromes (such as Gitelman/Barter syndrome) and familial hypokalemic periodic paralysis were considered [12]. Extensive work up showed normal serum magnesium, Thyroid Stimulating Hormone, T3, T4, aldosterone, renin levels. Renal function and liver function were also normal. Spinal cord imaging was unremarkable for any pathology. Low trans-tubular potassium gradient showed that there was not excessive urinary loss of potassium making conditions such as Barter or Gitelman syndrome unlikely. Due to late onset of symptoms in the sixties (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 importance of 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 al described the first case of this underappreciated etiology of hypokalemia. [15] Few of the case reports described hypokalemia secondary to cola consumption in pregnant females [15, 16]. Sharma et al reported a case of 58 year old male consuming 6-7 L of cola per day. He presented to the hospital with severe generalized weakness and significantly low serum potassium. [18]

Mechanism of hypokalemia secondary to excessive cola consumption can be attributed to increase renal wasting of potassium. This occurs due to caffeine induced diuresis and increased renin release. High caffeine content in cola causes downstream elevation of cAMP (Cyclic adenosine monophosphate) and intracellular potassium shifts. The stimulation of Beta 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 leads 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 gastro-intestinal tract leading to osmotic diarrhea subsequently causing potassium and other electrolyte losses. [17] Due to lack of 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 excessive potassium losses.

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.
References:


7. Coca cola. http://www.coca-cola.co.uk/


Table 1: Table comparing salient features of case reports on cola induced hypokalemia (N = value within normal range; not specified in the case report. M=male, F=female).

<table>
<thead>
<tr>
<th>References</th>
<th>Age (years)</th>
<th>Gender</th>
<th>Amount of cola consumed</th>
<th>Duration of cola consumption</th>
<th>K levels at the time of admission (mmol/l)</th>
<th>Urine K (mmol/l/d)</th>
<th>Renin (ng/ml/h)</th>
<th>Aldosterone (ng/dl)</th>
<th>f/u K after stopping/reducing cola (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present case</td>
<td>65</td>
<td>F</td>
<td>3-4L/d</td>
<td>2-3 years</td>
<td>1.9</td>
<td>26</td>
<td>2.1</td>
<td>5.6</td>
<td>3.8</td>
</tr>
<tr>
<td>Sharma et al [18]</td>
<td>58</td>
<td>M</td>
<td>6-7L/d</td>
<td>Several years</td>
<td>2.4</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>_</td>
</tr>
<tr>
<td>Packer et al [19]</td>
<td>52</td>
<td>M</td>
<td>&lt;1L/d</td>
<td>Several years</td>
<td>3.0</td>
<td>8.6</td>
<td>0.33</td>
<td>4.8</td>
<td>3.5</td>
</tr>
<tr>
<td>Kasap et al [20]</td>
<td>16</td>
<td>M</td>
<td>&gt;1L/d</td>
<td>2-3 yrs</td>
<td>2.2</td>
<td>31</td>
<td>2.1</td>
<td>15</td>
<td>N</td>
</tr>
<tr>
<td>Chaudhry S-P et al [21]</td>
<td>21</td>
<td>F</td>
<td>4L/d</td>
<td>8 months</td>
<td>2.2</td>
<td>11</td>
<td>_</td>
<td>_</td>
<td>_</td>
</tr>
<tr>
<td>Mudge et al [22]</td>
<td>44</td>
<td>M</td>
<td>4-10L/d</td>
<td>3 yrs</td>
<td>1.4</td>
<td>17.4</td>
<td>_</td>
<td>_</td>
<td>_</td>
</tr>
<tr>
<td>Appel et al [16]</td>
<td>24</td>
<td>F</td>
<td>6-7L/d</td>
<td>10 months</td>
<td>2.0</td>
<td>41.9</td>
<td>12.9</td>
<td>3.6</td>
<td>N</td>
</tr>
<tr>
<td>Rice et al [14]</td>
<td>21</td>
<td>F</td>
<td>8L/d</td>
<td>1-2 yrs</td>
<td>1.8</td>
<td>9</td>
<td>_</td>
<td>_</td>
<td>_</td>
</tr>
<tr>
<td>Lee et al [13]</td>
<td>52</td>
<td>M</td>
<td>4-9L/d</td>
<td>1.5 month</td>
<td>2.3</td>
<td>6.6</td>
<td>_</td>
<td>_</td>
<td>4.2</td>
</tr>
<tr>
<td>Matsunami et al [15]</td>
<td>21</td>
<td>F</td>
<td>3L/d</td>
<td>6 yrs</td>
<td>1.9</td>
<td>_</td>
<td>_</td>
<td>0.67 ng/dl</td>
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Figure 1: