Hypocalemic Myopathy Due to Excessive Cola Consumption

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SUMMARY
A 25-year-old man was admitted to our emergency department (ED) with progressive paralysis in both upper/lower extremities. His serum potassium level was very low. Laboratory testing ruled out renal potassium wasting. A dietary history revealed that he had been consuming 2-4 liters of cola per day for six years. We present a case of hypokalaemic myopathy induced by chronic cola consuming.

Key words: Cola; hypokalemia; myopathy.

Case Report
A 25-year-old man was admitted to our emergency department (ED) with significant worsening of muscle weakness and progressive paralysis in both upper/lower extremities for 2 days. He had no vomiting or diarrhea during that time. Patient history revealed that he had been consuming 2-4 liters of cola per day for the past six years. The patient was alert and oriented, and his skin was warm and dry. His vital signs were as follows: body temperature 36.7 ºC, heart rate 107 beats/min regular, with normal hearth sounds, arterial blood pressure 120/70 mmHg and respirations 15 per minute. There was muscle weakness to grade 2/5. Besides, deep tendon reflexes were reduced in both lower extremities. The patient’s electrocardiography (ECG) showed widespread ST depression, most prominent in leads V2-6 (Fig. 1).

Laboratory results include serum potassium 1.8 mmol/L and...
other biochemical parameters, CBC count and arterial blood gas analysis were normal. His urine potassium level was 6.4 mmol/L and urine osmolality was 224 mosm/kg. Serum aldosterone was 5.6 ng/dL (normal 4–31 ng/dL) and the plasma renin activity was 0.38 ng/mL/hr (normal 1.31-3.96 ng/mL/hr upright, 0.15–2.33 ng/mL/hr supine). Spot urine potassium was 8.6 mEq/L, urine sodium was < 10 mEq/L, and urine chloride was 26 mmol/L. His thyroid function test was normal. There were no explanation for the hypokalemia, other than excessive consumption of cola. The patient’s cola intake was stopped and, oral and intravenous (i.v.) potassium replacement therapy was started. A total of 120 mEq of KCl was initially ordered (60 mEq i.v. KCl over 6 hours in a dextrose-free normal saline solution and 60 mEq KCl orally). A 50 mEq i.v. KCl was also given within 4 hours after the 24 hours of initial potassium therapy.

As electrolyte replacement proceeded, the patient’s neurological symptoms and signs gradually improved over the next 32 hours. His serum potassium level was reached from 1.8 mmol/L to 4.4 mmol/L and he had 5/5 muscle strength in all extremities. The patient was discharged from the ED with the suggestion of ending excessive cola consumption and eating foods rich in potassium.

**Discussion**

The first report of cola-induced hypokalemia was on 1993 by Matsunami et al. Several years later, Appel and Myles reported on another pregnant woman who presented with ascending muscular weakness and very low serum potassium levels. The main ingredients of cola are high-fructose corn syrup, sugar, colorings, phosphoric acid, caffeine, citric acid, and natural flavors. There is approximately 110.4 g of high-fructose corn syrup per liter of regular cola, so it follows that this patient was consuming approximately 220-440 grams of high-fructose corn syrup per day. High-fructose corn syrup is 90% fructose and 10% glucose, which calculates to a daily fructose intake of 198-396 grams. Fructose is absorbed in limited quantities (only about 40% as compared with glucose) by a facilitated transport mechanism in the small intestine. Therefore, a large amount of unabsorbed fructose passed into the colon, causing an osmotic diarrhea and chronic potassium depletion. In the present case, because of the patient had no diarrhea before, current hypokalemic situation could not explained by osmotic diarrhea. The quantities of cola consumed in these case studies varied from 2 to 9 L per day, whereas the most common complaints were muscular in origin and ranged from mild weakness to profound paralysis. The normal plasma renin activity, normal serum aldosterone, and low urine potassium suggest that this patient’s hypokalemia was not caused by renal potassium wasting.

There are several potential mechanisms by which caffeine may produce hypokalemia. Perhaps by increasing renin release, caffeine may increase renal excretion of potassium. Redistribution of potassium into cells by elevation of intracellular cyclic adenosine monophosphate levels may occur. Caffeine induces catecholamine release, probably by means of adenosine antagonism; excessive β-adrenergic stimulation may mediate hypokalemia. Caffeine-induced hyperven-
tilation with respiratory alkalosis is another possible mechanism. In our patients hyperventilation wasn’t seen. The most significant factor underlying hypokalemia in our patient may have been excessive caffeine intake.

It is known that an oral intake of only 180–360 mg caffeine can provoke serious hypokalemia. Cola contains 130 mg caffeine per liter and our patient thus had consumed approximately 260-520 mg caffeine per day for more than 6 years. In most of the cases, caffeine intoxication was thought to play the most important role for hypokalemia. In support to this assumption, several other cases of hypokalemia have been described in individuals consuming large amounts of caffeine-containing preparations (such as tea or coffee) that do not contain glucose of fructose. Probably our patient had consumed plenty of tea and coffee drinks as well as.

**Conclusion**

Chronic consumption of large amounts of cola soft drinks may adversely affect potassium homeostasis and result in potentially severe conditions, such as hypokalaemic myopathy.

When encountered with unexplained hypokalemia, patients should be asked to provide a thorough history of caffeine intake, such as cola, coffee, cocoa and oriental tea.

**Conflict of Interest**

The authors declares no conflict of interest related to this work.

**References**