

# Hypocalemic Myopathy Due to Excessive Cola Consumption

## Aşırı Kola Tüketimine Bağlı Gelişen Hipokalemik Miyopati

Halil KAYA, Mehmet Tahir GÖKDEMİR, Özgür SÖĞÜT, Levent ALBAYRAK

Department of Emergency Medicine, Harran University Faculty of Medicine, Şanlıurfa, Turkey

### 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.

### ÖZET

Acil servisimize 25 yaşında bir erkek hasta her iki alt/üst ekstremitesinde yaygın kas güçsüzlüğü ile başvurdu. Hastanın serum K<sup>+</sup> düzeyi çok düşük olarak tespit edildi. Böbrek testleri ile böbrekten K<sup>+</sup> kaybı ekarte edildi. Diyet anamnezinde hastanın son 6 yıldır günde ortalama olarak 2-4 litre kola tükettiği ortaya çıktı. Bu yazıda, kronik kola tüketiminin neden olduğu hipokalemik miyopati bir olguyu sunduk.

**Anahtar sözcükler:** Kola; hipokalemi; miyopati.

### Introduction

The consumption of soft drinks has increased considerably during the last decades.<sup>[1]</sup> There are a few case reports of severe chronic hypokalemia due to long-term, excessive cola consumption.<sup>[2]</sup> Important concerns have been raised about the effects of cola on human health. Complications described in the reports include nephrogenic diabetes insipidus, hypokalemic myopathy and hypokalemic nephropathy. Cola contains large amounts of caffeine. It is well known that caffeine intoxication induces neuro-psychiatric, cardiovascular, respiratory, gastrointestinal and metabolic abnormalities. Metabolic complications include hyponatremia, metabolic acidosis, hyperglycemia, respiratory alkalosis and hypokalemia.<sup>[3]</sup>

We report a case of hypokalemic myopathy developed in a patient following large amount of cola consumption.

### 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 regular cola consumption, 2 to 4 L 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 °C, heart rate 107 beats/min regular, with normal heart 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

Presented at the 7th Turkish Congress on Emergency Medicine (TATKON 2011) (October 13-16, 2011, Trabzon, Turkey).

Submitted (Geliş tarihi): 10.11.2011 Accepted (Kabul tarihi): 11.01.2012 Published online (Online baskı): 05.03.2012

Correspondence (İletişim): Dr. Halil Kaya. Harran Üniversitesi Tıp Fakültesi Acil Tıp Anabilim Dalı, Şanlıurfa, Turkey

e-mail (e-posta): drhalilkaya@gmail.com



**Figure 1.** All leads of ECG consisted widespread ST depression most prominent in leads V2-6.

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.<sup>[4]</sup> Several years later, Appel and Myles reported on another pregnant woman who presented with as-

ending muscular weakness and very low serum potassium levels.<sup>[5]</sup> 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.<sup>[6]</sup> Fructose is absorbed in limited quantities (only about 40% as compared with glucose) by a facilitated transport mechanism in the small intestine.<sup>[7]</sup> 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.<sup>[8]</sup> 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.<sup>[9]</sup> Redistribution of potassium into cells by elevation of intracellular cyclic adenosine monophosphate levels may occur.<sup>[9]</sup> Caffeine induces catecholamine release, probably by means of adenosine antagonism; excessive  $\beta$ -adrenergic stimulation may mediate hypokalemia. Caffeine-induced hyperven-

tilation with respiratory alkalosis is another possible mechanism.<sup>[10]</sup> 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.<sup>[11]</sup> 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 or 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

1. Nielsen SJ, Popkin BM. Changes in beverage intake between 1977 and 2001. *Am J Prev Med* 2004;27:205-10.
2. Takami M, Imuta N, Hara H, Abe Y, Oguro R, Shimaoka I, et al. Hypokalemic myopathy with long-term heavy Cola consumption. [Article in Japanese] *Nihon Naika Gakkai Zasshi* 2005;94:132-4. [Abstract]
3. Stavric B. Methylxanthines: toxicity to humans. 2. Caffeine. *Food Chem Toxicol* 1988;26:645-62.
4. Matsunami K, Imai A, Tamaya T. Hypokalemia in a pregnant woman with long-term heavy cola consumption. *Int J Gynaecol Obstet* 1994;44:283-4.
5. Appel CC, Myles TD. Caffeine-induced hypokalemic paralysis in pregnancy. *Obstet Gynecol* 2001;97:805-7.
6. High-fructose corn syrup. [http://en.wikipedia.org/wiki/High\\_fructose\\_corn\\_syrup](http://en.wikipedia.org/wiki/High_fructose_corn_syrup).
7. Rumessen JJ. Fructose and related food carbohydrates. Sources, intake, absorption, and clinical implications. *Scand J Gastroenterol* 1992;27:819-28.
8. Packer CD. Chronic hypokalemia due to excessive cola consumption: a case report. *Cases J* 2008;1:32.
9. Arnaud MJ. The pharmacology of caffeine. *Prog Drug Res* 1987;31:273-313.
10. Benowitz NL. Clinical pharmacology of caffeine. *Annu Rev Med* 1990;41:277-88.
11. Passmore AP, Kondowe GB, Johnston GD. Caffeine and hypokalemia. *Ann Intern Med* 1986;105:468.