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A cola-induced hypokalemic rhabdomyolysis with electromyographic evaluation: A case report

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Abstract

Objective: To report a rare case of hypokalemic rhabdomyolysis induced by the heavy and prolonged ingestion of cola-based beverages, and its uneventful recovery after kalemia normalization.

Methods: We report a 38-year-old Caucasian male presented in our emergency room with a recent and progressive weakness of the lower limbs proximal muscles.

Results: A dietary history revealed a prolonged ingestion of cola-based beverages. Blood tests showed severe hypokalemia and marked increase in serum creatine phosphokinase. The analysis of cerebrospinal fluid resulted normal. Electromyography was suggestive for a myopathy. The clinical, laboratory and neurophysiological data were evocative for a cola-induced hypokalemic rhabdomyolysis. After kalemia normalization, the improvements of the electromyographic findings paralleled the clinical recovery.

Conclusion: Chronic consumption of large amount of cola-based soft drinks may result in severe symptomatic hypokalemia, eventually leading in turn to myopathy. To our knowledge, this is the first description of the electromyographic findings of the cola-induced hypokalemic rhabdomyolysis. An early diagnosis and a prompt treatment appear to be crucial for a benign clinical course.

Keywords

Coca cola, hypokalemic rhabdomyolysis, electromyography

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Introduction

There is an assumption that the intake of large amount of cola-based drinks may result in severe symptomatic hypokalemia.[1,2] Multiple pathophysiological mechanisms have been hypothesized: glucose-promoted osmotic diuresis and kaliuresis, chronic caffeine toxicity, reactive hyperinsulinemia and fructose-induced osmotic diarrhea.[3,4]

A characterization of electromyographic findings of cola-induced hypokalemic rhabdomyolysis is lacking. Here, we describe a rare case of hypokalemic rhabdomyolysis induced by excessive cola consumption, its recovery after kalemia normalization and the electromyographic evaluation before and after the treatment.

Case report

A 38-year-old Caucasian male presented in our emergency room with a 10-day history of low back pain followed by progressive weakness of proximal muscles of the lower limbs associated with muscle pain, cramps and inability to walk. No vomiting, diarrhea or weight loss were present. The patient reported a past history of alcohol abuse treated with sodium oxybate and a daily consumption of approximately 6 L of cola soft drink for the past 3 years. There was no

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Table 1. EMG findings at baseline and after potassium replacement therapy and recorded 1 month after the cessation of cola consumption.

Muscle	Before treatment				After treatment			
	MUP amplitude	MUP duration	Fibrillation potentials and/or positive sharp waves	Recruitment pattern	MUP amplitude	MUP duration	Fibrillation potentials and/or positive sharp waves	Recruitment pattern
Right tibialis anterior	Normal	Normal	+	Interference, reduced amplitude, early recruitment	Not performed			
Left tibialis anterior	Normal	Normal	Absent	Interference, reduced amplitude, early recruitment	Not performed			
Right vastus medialis	Normal	Normal	+	Interference, reduced amplitude, early recruitment	Lower limit	Lower limit	Absent	Interference, reduced amplitude, early recruitment
Left vastus medialis	Normal	Normal	+	Interference, reduced amplitude, early recruitment	Lower limit	Lower limit	Absent	Interference, reduced amplitude, early recruitment
Right biceps brachii	Normal	Normal	+	Interference, reduced amplitude, early recruitment	Lower limit	Lower limit	Absent	Interference, reduced amplitude, early recruitment
Right deltoid	Normal	Normal	+	Interference, reduced amplitude, early recruitment	Not performed			

EMG: electromyography; MUP: motor unit potential.

familial history of muscle diseases. The neurological examination revealed a symmetrical lower limbs proximal weakness (grade 2/5 of Medical Research Council grading system), specifically in the quadriceps femoris muscle and iliopsoas muscle, bilaterally. Muscle strength in the remaining lower limbs muscles and in all upper limbs muscles was normal. Blood tests showed severe hypokalemia (1.06 mEq/L), marked increase in serum creatine-phosphokinase level (CPK) (3763 U/L) and a slight elevation in aspartate aminotransferase (77 U/L) and in alanine aminotransferase (67 U/L). The other serum electrolytes levels, thyroid, renal and liver function tests were normal. A slight respiratory alkalosis (arterial pH 7.50) was detected. Urinary potassium level was 7 mmol/L. Total-body computed tomographic (CT) scan was negative. The cytomorphological and chemical analysis of cerebrospinal fluid (CSF) resulted normal as well as immunofixation. The polymerase chain reaction for neurotropic viruses, *Treponema pallidum* research and CSF bacteriological culture were negative. Autoimmune and thrombophilia screening were unremarkable.

A nerve conduction study with disposable bipolar rectangular surface electrodes (Neuroline 70.010-K/C/12, Ambu®) was performed. According to the normative data of our laboratory, the examination revealed normal motor and sensitive nerve conduction velocities and normal amplitudes of both compound muscle action potential (cMAP) and sensitive action potential (SAP), with the exception of reduced cMAP amplitude of the right median nerve (1.9 mV). A needle electromyography (EMG) (Neuroline concentric needle, 38 × 0.45 mm, 1.5″ × 26G, Ambu) was performed on tibialis anterior muscle and vastus medialis muscle of both sides and on right biceps brachii and deltoid muscles. An abnormal spontaneous activity (fibrillation potentials and positive sharp waves) was found in five of these six muscles, along with motor unit potentials (MUPs) within normal limits for amplitude and duration (Table 1). The maximal voluntary contraction showed an interference pattern of reduced amplitude and an early recruitment of motor units.

Potassium replacement therapy (13.50 g of intravenous potassium administered over 72 h) and cessation of cola consumption resolved hypokalemia. The CPK levels fell to normal value (61 U/L) within 20 days. During the same period, the patient experienced a progressive improvement of the proximal muscles lower limbs weakness, in parallel with the normalization of CPK levels. Clinical, laboratory and neurophysiological data suggested a cola-induced hypokalemic rhabdomyolysis. The EMG was repeated after 1 month and did not show any pathological spontaneous activity in the previously examined muscles. Duration and amplitude of MUPs, during mild voluntary contraction, were at the lower limit of normality on the right biceps brachii muscle and on the vastus medialis muscle of both sides (Table 1).

Discussion

Here, we report a rare case of hypokalemic rhabdomyolysis induced by the heavy and prolonged ingestion of cola-based

beverages. To the best of our knowledge, this is the first report of the electromyographic pattern that such disease exhibits. The myopathic EMG findings were substantially mild in degree and consisted of signs of muscle fibers damage, reduced amplitude and early recruitment of the interference pattern. The presence of an abnormal spontaneous activity, such as fibrillation potentials and positive sharp waves, may indicate muscle membrane irritability, which could be secondary to toxic, inflammatory, dystrophic, congenital or metabolic muscular disorders.[5] This abnormal spontaneous activity is non-specific and simply represents the chronic nature of the underlying process.[6] The reduced interference pattern is likely to be related to the mechanical muscular fibers damage,[6] as a consequence of hypokalemia. Finally, the early recruitment of motor units could indicate the presence of many active motor units with high firing frequency, which are necessary to optimize and increase motor strength.[6] Together with these findings, we found MUPs within normal limits for amplitude and duration. This is understandable, since modifications in MUPs generally follow the presence of abnormal spontaneous activity.[6] Of note, we also found reduced cMAP amplitude of the right median nerve. This is consistent with the literature data, which show nerve conduction abnormalities during episodes of hypokalemic paralysis in the presence of weakness.[7,8]

The myopathic EMG findings improved after potassium replacement therapy, signaling the full recoverability of the disease when its causes are early discovered and promptly treated. Not surprisingly, there was a change in the MUPs from "normal" before treatment to "lower limit" after treatment in three of the explored muscle groups, despite of weakness resolution and serum CPK normalization. This probably reflects the long-standing muscle damage occurred during the disease course. Severe hypokalemia following large ingestion of cola-based beverages can develop through various mechanisms.[4] First, the large amount of glucose present in the beverages can induce both an osmotic diuresis with kaliuresis and a secondary hyperinsulinemia, which leads to potassium redistribution between extra- and intracellular compartments. Second, the large amount of fructose present in the beverages passes almost unabsorbed into the colon where it promotes osmotic diarrhea with potassium depletion. Third, hypokalemia can develop as result of caffeine toxicity. This latter seems the most plausible explanation for the clinical pattern. Our patient had consumed 0.5–0.7 g of caffeine per day during the previous 3 years. Symptoms of caffeine toxicity may occur in adult at doses of as little as 500–600 mg/day. The consumption of moderate quantities of caffeine may result in severe hypokalemia because of potassium redistribution into the cells and its increased renal excretion.[4] The inhibition of phosphodiesterase, which results in augmented levels of intracellular cyclic adenosine monophosphate (cAMP), along with the caffeine-induced respiratory alkalosis and adrenergic stimulation, possibly represent the main mechanisms underlying the shift of potassium into the cells. Increased levels of cAMP enhances the Na^+/K^+-ATPase activity. Severe serum potassium depletion can be in turn responsible for rhabdomyolysis,[9] characterized by muscle pain, weakness, dark urine, fever, tachycardia, nausea with vomiting and marked elevation of serum CPK (five times the upper limit of normal). Hypokalemia likely leads to rhabdomyolysis by disrupting the Na^+/K^+-ATPase and Ca^{2+}-ATPase activity, which in turn results in cellular Na^+ and Ca^{2+} influx and muscle cells death. Although it is conceivable that rhabdomyolysis is mainly responsible for the patient's weakness, alternative explanations cannot be excluded. A low extracellular K^+ concentration alters channel gating and reduces the conductance of some K^+ channels.[4] Moreover, the reduction in serum K^+ concentration shifts the potassium equilibrium potential (and in turn the resting membrane potential) to more hyperpolarized values, according to the Nernst equation ($E_K = -58$ mV $\times \log[(K)_{in}/(K)_{out}]$). Consequently, the transmission of action potentials will be disrupted, and the result can be generalized weakness.

In conclusion, we reported a rare case of hypokalemic rhabdomyolysis induced by the prolonged intake of large amount of cola-based beverages. The myopathic features of the disease, ascertained by electromyographic investigation, are described for the first time. The improvements of these myopathic features after therapy paralleled the clinical course of the disease.

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Informed consent

Written informed consent was obtained from the patient for their anonymized information to be published in this article.

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