Severe hypokalemic paralysis and rhabdomyolysis occurring after binge eating in a young bodybuilder

Case report

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Abstract

Rationale: Severe hypokalemia can be a potentially life-threatening disorder and is associated with variable degrees of skeletal muscle weakness.

Patient concerns: We report a case of severe hypokalemia paralysis and rhabdomyolysis in a 28-year-old bodybuilder. He was admitted to the emergency room due to progressive paralysis in both lower extremities, which had begun 12 hours earlier. He was a bodybuilder trainer and had participated in a regional competition 5 days earlier. He went on a binge, consuming large amounts of carbohydrates over 4 days, resulting in a gain of 10 kg in weight.

Diagnoses: He had no family history of paralysis and this was his first attack. He strongly denied drug abuse, such as anabolic steroids, thyroid and growth hormone, and diuretics. Neurological examinations revealed symmetrical flaccid paralysis in his lower extremities, but the patient was alert and his sensory system was intact. His initial serum potassium and phosphate level was 1.8 mmol/L and 1.4 mg/dL, respectively. The calculated transtubular potassium gradient (TTKG) was 2.02. His thyroid function was normal.

Interventions and Outcomes: Serum potassium levels increased to 3.8 mmol/L with intravenous infusion of about 50 mmol of potassium chloride over 20 hours.

Outcomes: His muscular symptoms improved progressively and he was discharged from the hospital 7 days after admission on foot. He was followed in our outpatient clinic, without recurrence.

Lessons: Physicians should keep in mind that large intakes of food during short periods can provoke hypokalemic paralysis and rhabdomyolysis, especially in bodybuilders.

Abbreviations: BUN = blood urea nitrogen, CPK = creatine phosphokinase, LDH = lactate dehydrogenase, TPP = thyrotoxic periodic paralysis, TTKG = transtubular potassium gradient.

Keywords: hypokalemia, paralysis, rhabdomyolysis

1. Introduction

Hypokalemia-related emergencies vary from simple muscle weakness to paralysis with or without respiratory muscle involvement and are referred to as hypokalemic paralysis.[1,2]

Rarely, severe hypokalemia may result in rhabdomyolysis, leading to acute kidney injury.[3-5] Hypokalemia can also induce electrocardiographic changes such as U wave, T-wave flattening, and arrhythmias.[6] The etiology of hypokalemic paralysis can be categorized generally into 2 groups: hypokalemic periodic paralysis, due to a shift of potassium into the intracellular space without an overall potassium deficit, and hypokalemic non-periodic paralysis, due to a large potassium deficit through gastrointestinal or renal loss.[7] Hypokalemic paralysis because of shifting of potassium may be associated with insulin administration, catecholamine excess, thyrotoxic periodic paralysis (TPP), and familial hypokalemic periodic paralysis.[8] Two cases of hypokalemic paralysis in bodybuilders have been reported.[9,10] We report an additional case of hypokalemic paralysis leading to rhabdomyolysis after binge eating in a bodybuilder.

2. Case report

A 28-year-old Korean male presented to the emergency room with progressive paralysis of the lower extremities, which had begun 12 hours earlier. He could not walk or lift himself off the bed upon admission to the hospital. He was a bodybuilder trainer and had participated in a regional competition 5 days earlier. He went on a binge, consuming large quantities of carbohydrates over 4 days, resulting in a gain of ∼10 kg in weight after the competition.
There was no family or personal history of periodic paralysis and this was the first attack. He also denied any underlying disease. In addition, the patient had no history of trauma. We asked him about intake of known toxins, drugs, dietary supplements, or nutraceuticals affecting serum potassium level several times, but he strongly denied taking them. He also did not suffer from polyuria or polydipsia, diarrhea, and vomiting.

His initial vital signs were as follows: blood pressure = 130/70 mm Hg, heart rate = 100 beats/min, respiratory rate = 20 breaths/min, and body temperature = 36.6°C. A neurological examination revealed a symmetrical decrease in motor power (Grade II/V) in his lower extremities, but the patient was alert and his sensory system was intact. Examination of the neck did not reveal an enlarged thyroid and there was no exophthalmos. We could not hear thyroid bruit. We could not find any features of Cushing disease, such as a moon face, buffalo hump, truncal obesity, or thinning of the skin. We also detected no diastolic bruit on abdomen examination.

Initial laboratory tests were as follows: sodium = 142 mmol/L (135–145 mmol/L), potassium = 1.8 mmol/L (3.3–5.1 mmol/L), chloride = 106.6 mmol/L (98–110 mmol/L), blood urea nitrogen (BUN) = 14.6 mg/dL (6–20 mg/dL), creatinine = 0.99 mg/dL (0.6–1.2 mg/dL), glucose = 129 mg/dL (70–110 mg/dL), calcium = 9.2 mg/dL (8.6–10.2 mg/dL), phosphate = 1.4 mg/dL (2.7–4.5 mg/dL), creatine phosphokinase (CPK) = 1580 U/L (0–171 U/L), lactate dehydrogenase (LDH) = 252 U/L (140–171 U/L), myoglobin = 313 ng/mL (23–72 ng/mL), magnesium = 2.2 mmol/L (1.5–2.5 mmol/L), white blood cells (WBCs) = 10,240 mm3/L (4000–10,000 mm3/L), hemoglobin = 16.0 g/dL (13.3–17.0 g/dL), hematocrit = 50% (39–52%), and platelet count = 263,000 mm3/L (130,000–400,000 mm3/L). An arterial blood gas analysis revealed a pH of 7.39 (7.35–7.45), pCO2 = 46 mm Hg (32–46 mm Hg), and HCO3 = 23 mmol/L (23–29 mmol/L). The calculated transtubular potassium gradient (TTKG) was 2.02, serum osmolality was 290 mOsm/kg, (276–300 mOsm/kg), urine osmolality was 799 mOsm/kg, urine sodium was 74.2 mmol/L, and urine potassium was 10 mmol/L. An initial electrocardiogram (ECG) revealed a prolonged corrected QU interval, ST-segment depression, inverting of the T wave, and prominent U waves (Fig. 1). These changes were all reversed after restoration of his serum potassium level (Fig. 1). A thyroid function test showed that his serum thyroid stimulation hormone (TSH) was 1.71 mIU/L (0.27–4.2 mIU/L), T3 was 63.98 ng/dL (80–200 ng/dL), and free T4 was 0.89 ng/dL (0.93–1.70 ng/dL). We could not measure serum and urine toxin and drug level because it is impossible in our institution.

The patient received intravenous replacement of 50 mmol potassium chloride during the initial 20 hours of treatment, after which his serum potassium level had increased to 3.8 mmol/L (Fig. 2) and his muscular power improved slowly (Gr. III/V). His muscle enzyme increased during potassium replacement and hydration was provided with normal saline infusion (Fig. 3). He could ambulate on the ward 72 hours after admission and was discharged from the hospital 7 days after admission with no apparent complication. His serum potassium and muscle enzyme were restored to normal ranges. He was followed in our outpatient clinic without recurrence of the attack for 8 months.

3. Discussion

We have described hypokalemic paralysis and rhabdomyolysis induced by binge eating in a bodybuilder. This report is meaningful in that life-threatening hypokalemia can occur in patients with weight gain over a short period via binge eating of large quantities of carbohydrate foods; this should be considered as another etiology of hypokalemia.

Two other cases of hypokalemic paralysis in bodybuilders have been reported in the English-language literature to our
In preparing for competition, one had a strict diet and misused different pharmacological substances, such as anabolic steroids, human growth hormone, thyroid hormone, and fast-acting insulin for an intended anabolic effect.\[^9\] Differing from our case, he gained weight with these measures, and hypokalemic paralysis occurred just after a competition; he also took furosemide 24 to 48 hours before the competition. Thus, his hypokalemia may have resulted from various complex etiologies. The other patient had taken agents of an unknown nature for weight control and his paralysis also occurred at the end of a competition without weight gain.\[^10\] Our patient strongly denied taking any of the pharmacological substances mentioned previously, nutraceutical or diuretics, and had gained 10 kg in weight over 5 days after a competition by binge eating.

The 2 most common forms of hypokalemic paralysis are thyrotoxic and familial periodic hypokalemic paralysis.\[^1,11–13\] The pathophysiology of the paralysis remains unclear. Hypokalemia is usually not an absolute deficiency of total body potassium but, rather, a rapid and exaggerated intracellular shifting of potassium into muscles during attacks. It is known that many predisposing factors, including high carbohydrate load, strenuous exercise, and emotional stress may be associated with paralysis.\[^1,11–13\] Our patient may have suffered from life-threatening hypokalemia due to a shift of the potassium from the extracellular to the intracellular space through stimulating endogenous insulin release by his large carbohydrate intake with hypophosphatemia.\[^11,19\] Judging from restoration of serum phosphate level to normal value after only potassium replacement, not phosphorus, hypophosphatemia in our case also might be originated from intracellular shifting although urine phosphate level was not measured.

In conclusion, with the recent, increasing numbers of new fitness centers and the increasing numbers of youngsters participating in bodybuilding, physicians should be able to recognize possible causes of sudden severe hypokalemia in these patients, with large amounts of muscle mass to treat, and advise the patients appropriately.

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