Bilateral Lower Extremity Paralysis in a Caucasian Male Presenting to the Emergency Department

Yicheng K. Bao, Vishwanath C. Ganesan, Richard Rapp, and Shunzhong S. Bao

1 University of Missouri-Kansas City School of Medicine, USA
2 Little Rock Diagnostic Clinic, USA

Correspondence should be addressed to Shunzhong S. Bao; sbao@lrdc.com

Received 20 November 2017; Accepted 17 April 2018; Published 15 May 2018

1. Case

A 39-year-old Caucasian male presented to the emergency department with sudden onset bilateral lower extremity paralysis, with normal sensation. He denied any abnormalities in his upper extremities, speech, or vision, and he also denied headache, fever, nausea, or vomiting. The patient consumed large carbohydrate meals with alcohol the night before at a Halloween party, and the patient reported stress from losing his job and separating from his wife. In further questioning, he reported anxiety, tremors, with weight loss of 9.1 kg in past 6 months. He did not take any medications and denied illicit drug use.

Physical examination shows an anxious man with BMI 29.27 kg/m², blood pressure 153/79 mmHg, temperature of 36.8°C, and respiratory rate 18/min. The patient had normal S1 and S2 with irregular beats, normal cranial nerve function, and normal upper extremities. Muscle strength of bilateral lower extremity is 3/5 with normal sensation. Tremors are positive in the upper extremity. Initial BMP revealed K 1.7 mEq/L (normal 3.5–5.1 mEq/L), Na 140 mEq (136–146 mEq), Cl 108.7 (98–107 mEq), CO₂ 22 mEq/L (22–31 mEq/L), glucose 147 mg/L (75–110 mg/L), Ca 9.8 mg (normal 8.4–10.2 mg/L), Creatinine 0.65 mg/dl (0.75–1.25 mg/dl), Magnesium 1.66 (1.3–2.1 mEq/L), total CK 330 (30–200 IU/L).

Initial EKG revealed ventricular heart rate of 66 BPM, atrial rate of 234 BPM, atrial flutter with variable A-V premature ventricular beats and aberrantly conducted complexes, prolonged QT of 474 ms, QTc 496 ms. Brain CT, MRI, and lumbar puncture were obtained and found to be normal.

Thyroid function was obtained: TSH < 0.003 (0.35–4.94 uIU/ml), T3 uptake 37 (15–50%), total T4 (4.87–11.72 ug/dl), T7 (Ft4) 4.29 (1.65–4.07 ug/dl), TSI 465 (<140%). Thyroid ultrasound revealed normal thyroid size, but with hypervascular heterogeneous parenchyma, suggesting hyperthyroidism. No nodules were identified.

The patient was treated with intravenous potassium and metoprolol initially, then propranolol and weakness gradually recovered in 12 hours. For the patient’s hyperthyroidism, he was treated with methimazole and then had total thyroidectomy.

2. Discussion

Thyrotoxic periodic paralysis is rare in Caucasians and typically presents in middle aged Asian men with recurring episodes of sudden onset bilateral lower extremity weakness that can range from weakness to complete paralysis [1]. The upper extremities and muscles controlled by cranial nerves are generally not affected. Decreased muscle tone with
hyporeflexia or areflexia is typical [2]. Sensory function is
typically normal, and there is no bowel or bladder control
dysfunction. Respiratory muscles are usually not affected, but
in severe cases they can be paralyzed, causing respiratory
failure [3]. Hypokalemia can also cause severe ventricular
arrhythmia, A-V block, and ventricular fibrillation [4]. Com-
mon triggers include high carbohydrate diet and alcohol,
with episodes most often occurring in the morning [4]. In
clinic reports, excessive exercise, trauma, emotional stress,
acute upper respiratory infection, exposure to cold, use of
drugs such as corticosteroids, epinephrine, and NSAIDs are
common precursors to thyrotoxic periodic paralysis [5, 6].

The patient consumed large amounts of carbohydrates
with alcohol the night before, which is a common precursor
of thyrotoxic periodic paralysis. If a patient presents with
sudden onset weakness and with severe hypokalemia, thyroid
function should be obtained even when there are no obvious
symptoms of hyperthyroidism.

2.1. Differential Diagnosis. The differential diagnosis should
focus on hypokalemia and the cause of hypokalemia. Thyro-
toxic periodic paralysis is not a condition of net potassium
deficiency; rather it is a condition of transcellular shift of
potassium [4, 7]. In this condition, the physician should exer-
cise caution not to overzealously replace potassium, because
this can cause hyperkalemia. Other causes for transcellular
shift are drugs such as tocolytics, theophylline toxicity, chlo-
roquine toxicity, insulin overdose, beta agonist overdose,
and familial hypokalemic periodic paralysis. In this case,
the emergency physician should also consider refeeding
syndrome in the differential diagnosis. In refeeding syn-
drome, high intake of carbohydrates and alcohol could cause
increases in insulin secretion, which leads to glycocen, fat,
and protein synthesis [8]. These processes could consume
cofactors such as thiamine and disturb electrolytes, resulting
in intracellular potassium shift (hypokalemia) and thiamine
deficiency, which could manifest themselves as paralysis, car-
diac arrhythmias, and acute Wernicke's encephalopathy [8].

In our case, the potassium was properly replaced and
monitored, but he was excessively worked up with a head CT,
head and spine MRI, lumbar puncture, and electromyogram.
In a presentation of hypokalemia and paralysis, the diffe-
rential diagnosis should focus on hypokalemia; the extensive
workup on paralysis should be held until potassium levels
recover.

2.2. Pathophysiology. The Na-K ATPase channel has been
implicated in TPP, because thyroid hormone increases the
Na-K ATPase and shift K into the cells [7]. High carbohydrate
meals and alcohol increase insulin levels, which stimulates
Na-K ATPase activity [4, 7]. Catecholamines can also increase
Na-K ATPase activity in skeletal muscles; therefore stress can
also facilitate the attack [4]. Our case has typical triggers, such
as stress from job loss and separation from a spouse, as well
as a large high carbohydrate meal and high alcohol intake the
night before the episode.

Androgens have been suggested to increase the expres-
sion of the Na-K ATPase and explain the male to female
prevalence to have been reported to be from 20 to 44:1 [5, 9],
although the prevalence of hyperthyroidism of male to female
is 1:10. It also suggested that the high level of testosterone
catecholamines in the morning may be responsible for
the higher rate of TPP in the morning [4]. Our patient had
attack early in the morning which is typical.

3. Treatment

Acute treatment requires correction of hypokalemia and con-
rol of hyperthyroidism; intravenous potassium with normal
saline is recommended. In correction for hypokalemia, atten-
tion should be paid to avoid overcorrection with rebound
hyperkalemia. Close monitoring is very important, especially
when cardiac arrhythmia is present.

Nonspecific beta blockers like propranolol are very effec-
tive and should be initiated immediately. Some specialists
recommend just using nonspecific beta blocker without
potassium supplement to avoid potassium rebound. In this
case, high dose (3-4 mg/kg orally) has been reported to be
used successfully.

Thyroid function control has been achieved with con-
tventional treatment with medication, surgery, or radioactive
iodine. Patients need to be consulted to avoid a high carbohy-
brate diet, high alcohol intake, or excessive physical exertion
even after hyperthyroidism has been controlled.

4. Conclusion

TPP is most commonly occurs in middle aged Asian men,
but we presented a case of Caucasian man. His hyperthy-
roidism was not previously diagnosed, and this episode was
preceeded by high carbohydrate and alcohol. In the ED,
typical presentations of paralysis and profound hypokalemia
should prompt the physician to check thyroid function. The
differential diagnosis should be focused on hypokalemia
instead of paralysis.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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