

## > THE PATIENT

33-year-old man

## > SIGNS & SYMPTOMS

- Flaccid paralysis in arms and legs
- 30-lb weight loss in previous month
- Thyromegaly without nodules

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## > THE CASE

A 33-year-old Hispanic man with no significant past medical history presented to the emergency department with generalized flaccid paralysis in both arms and legs. Two days before, he had been working on a construction site in hot weather. The following day, he woke up with very little energy or strength to perform his daily activities, and he had pain in the inguinal area and both calves. He denied taking any medications or supplements.

The patient had complete muscle weakness and was unable to move his arms and legs. He reported dysphagia and an unintentional weight loss of 30 lb during the previous month.

On physical examination, the patient's vital signs were within the normal range, and mild thyromegaly without nodules was present. Neurologic examination revealed decreased deep tendon reflexes with intact sensation. Muscle strength in his arms and legs was 0/5.

Initial laboratory test results included a potassium level of 2.2 mEq/L (normal range, 3.5–5 mEq/L) and normal acid-basic status that was confirmed by an arterial blood gas measurement. Serum magnesium was 1.6 mg/dL (normal range, 1.6–2.5 mg/dL); phosphorus, 1.9 mg/dL (normal range, 2.7–4.5 mg/dL); and random urinary potassium, 16 mEq/L (normal range, 25–125 mEq/L). An initial chest x-ray was normal, and an electrocardiogram showed a prolonged QT interval, flattening of the T wave, and a prominent U wave consistent with hypokalemia.

## THE DIAGNOSIS

The initial clinical diagnosis was hypokalemic paralysis. The patient was treated with intravenous (IV) potassium chloride 40 mEq; however, his potassium level decreased further, to 1.8 mEq/L. Potassium chloride administration was continued and potassium levels were monitored. Normal saline 1 L was also administered, and other electrolyte abnormalities were corrected.

Evaluation of the patient's hypokalemia revealed the following: thyroid-stimulating hormone (TSH) level, < 0.01 microIU/mL (normal range, 0.27–4.2 microIU/mL); free T4 (thyroxine) level, 4.47 ng/dL (normal range, 0.08–1.70 ng/dL); total T3 (triiodothyronine) level, 17.5 ng/dL (normal range, 2.6–4.4 ng/dL).

The patient was diagnosed with hypokalemic periodic paralysis (HPP) secondary to thyrotoxicosis, also known as thyrotoxicosis periodic paralysis (TPP). His hyperthyroidism was treated with oral atenolol 25 mg/d and oral methimazole 10 mg tid.

Within a few hours of this treatment, the patient experienced significant improvement in muscle strength and complete resolution of weakness in his arms and legs. Serial measurements of potassium levels normalized.

Further workup revealed that the patient's thyroid-stimulating immunoglobulin (TSI) was 4.2 on the TSI index (normal, ≤ 1.3) and his thyroid peroxidase (TPO) antibody level was

133.4 IU/mL (normal, < 34 IU/mL). Ultrasonography showed decreased echogenicity of the thyroid gland, consistent with the acute phase of Hashimoto thyroiditis or Graves disease.

The patient was unaware that he had any thyroid disorder previously. He was a private-pay, undocumented immigrant and did not have a regular primary care physician. On discharge, he was referred to a local primary care physician as well as an endocrinologist. He was discharged on atenolol and methimazole.

## DISCUSSION

A rare neuromuscular disorder known as periodic paralysis can be precipitated by a hypokalemic or hyperkalemic state; HPP is more common and can be either familial (a defect in the gene) or acquired (secondary to thyrotoxicosis; TPP).<sup>1,2</sup> In both forms of periodic paralysis, patients present with hypokalemia and paralysis. Physicians need to look closely at thyroid lab test results so as not to miss the cause of the paralysis.

TPP is most commonly seen in Asian populations, and 95% of cases reported occur in males, despite the higher incidence of hyperthyroidism in females.<sup>3</sup> TPP can be precipitated by emotional stress, steroid use, beta-adrenergic bronchodilators, heavy exercise, fasting, or high-carbohydrate meals.<sup>2-4</sup> In our patient, heavy exercise and fasting likely were the triggers.

The pathophysiology for the hypokalemia in TPP is thought to involve the sodium/potassium-adenosine triphosphatase (Na<sup>+</sup>/K<sup>+</sup>-ATPase) pump. This pump activity is increased in skeletal muscle and platelets in patients with TPP vs patients with thyrotoxicosis alone.<sup>3,5</sup>

### ■ The role of Hashimoto thyrotoxicosis.

Most acquired cases of TPP are mainly secondary to Graves disease with elevated levels of TSI and mildly elevated or normal levels of TPO. In this case, the patient was in the acute phase of Hashimoto thyrotoxicosis (“hashitoxicosis”) with elevated levels of TPO and only mildly elevated TSI. Imaging studies to support the diagnosis, such as a thyroid up-

take scan or ultrasonography, are not necessary to determine the cause of thyrotoxicosis. In the absence of test results for TPO and TSI antibodies, however, a scan can be helpful.<sup>6,7</sup>

■ **Treatment of TPP** consists of early recognition and supportive management by correcting the potassium deficit; failure to do so could cause severe complications, such as respiratory failure and psychosis.<sup>8</sup> Because of the risk for rebound hyperkalemia, serial potassium levels must be measured until a stable potassium level in the normal range is achieved.

Nonselective beta-blockers, such as propranolol (3 mg/kg) 4 times per day, have been reported to ameliorate the periodic paralysis and prevent rebound hyperkalemia.<sup>9</sup> Finally, restoring a euthyroid state will prevent the patient from experiencing future attacks.

## THE TAKEAWAY

Few medical conditions result in complete muscle paralysis in a matter of hours. Clinicians should consider the possibility of TPP in any patient who presents with acute onset of paralysis. **JFP**

### CORRESPONDENCE

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**Failure to correct the potassium deficit in thyrotoxicosis periodic paralysis could cause severe complications, such as respiratory failure and psychosis.**