Answer

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The correct diagnosis is D. In this case, a routine electrolyte screen* revealed a serum potassium of 1.7 mmol/L (normal = 3.5–5.0) and a serum phosphate of 0.39 mmol/L (0.80–1.60). Calcium, magnesium, creatine kinase and ESR (erythrocyte sedimentation rate) were normal.

Thyrotoxic periodic paralysis (TPP) is an uncommon disorder characterized by thyrotoxicosis, hypokalemia and paralysis.[1] It occurs primarily in Asians but has been described in many ethnic groups. Features are identical to those of familial periodic paralysis (FPP), except that TPP attacks occur in the thyrotoxic state and can rarely be precipitated in euthyroid patients. (Note, however, that the features of hyperthyroidism may be subtle or subclinical in patients with TPP.) "Paralytic" attacks may be triggered by carbohydrate loading, strenuous activity followed by rest, trauma, cold exposure, infection, menses and emotional stress.[2] Attacks are usually self-limited and last from 3 to 36 hours, but may recur in susceptible patients.

In TPP and FPP, episodes are usually abrupt in onset with varying degrees of weakness. They typically affect the lower limbs more than the upper limbs and proximal muscles more than distal muscles. Reflexes are diminished or absent, but cranial and respiratory muscles are almost always spared and the sensory examination is usually normal. Patients may experience a variety of dysrhythmias,[3] and the ECG will often exhibit hypokalemic changes, including U waves, T-wave flattening, ST-segment depression, and QT prolongation.

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Hypokalemia in TPP reflects intracellular potassium shifting rather than total body depletion. The mechanism for this is poorly understood, but hyperthyroidism (through beta-2 adrenergic stimulation) is thought to increase Na+/K+ ATPase activity. This increased membrane pump activity would cause movement of potassium into cells. In addition, hyperthyroidism may have direct membrane effects favouring potassium influx.[4] The resulting high intracellular potassium levels alter depolarization and hamper muscle contraction. Hypophosphatemia may also contribute to in TPP-related muscle weakness.[5]

TPP treatment involves avoiding trigger factors and correcting the hyperthyroid state. In addition, potassium replacement is important to alleviate paralysis and prevent fatal dysrhythmias. In stable patients, oral potassium (20 mEq every 2 hours until muscle strength begins to recover) is usually sufficient, but doses exceeding 90 mEq per day may cause rebound hyperkalemia.[1] Propranolol has been shown to decrease the frequency and severity of paralytic attacks, whereas metoprolol (a beta-1 selective antagonist) has not.[6]

This patient received potassium 80 mEq (40 mEq intravenously and 40 mEq orally) and propranalol 50 mg orally. His symptoms resolved within 6 hours.

References

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***Editor's note:** There is no such thing as a routine electrolyte screen. Clever emergency physicians realize that disorders of potassium, magnesium and phosphate can cause muscular weakness. They also realize that serum magnesium levels do not reflect body magnesium stores and that normal serum magnesium levels are meaningless. [G.I.]

For the Challenge, see page 164.

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