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Case Report

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Sudden Paralysis of Lower Extremities Due to An Underlying Endocrinopathy

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Abstract

Thyrotoxic periodic paralysis (TPP) is a rare but serious complication of hyperthyroidism characterized by episodes of muscle weakness and paralysis [1]. Here, we present a case report of a 44-year-old male patient who presented with acute muscle weakness and was diagnosed with TPP in the setting of Grave's disease [1]. The patient's clinical presentation, diagnostic workup, management, and outcome are discussed in detail.

Keywords: Thyrotoxic Periodic Paralysis; Grave's Disease; Muscle Weakness; Sclerosis; Autoimmune Disease

Introduction

Thyrotoxic periodic paralysis (TPP) is a rare manifestation of hyperthyroidism, characterized by episodes of muscle weakness and paralysis. It affects predominantly young Asian males with hyperthyroidism, particularly those with Graves' disease. The exact pathophysiology of TPP remains unclear, but it is believed to involve abnormalities in potassium utilization in skeletal muscles induced by excessive thyroid hormone levels [2]. Punctual recognition and treatment of TPP are essential to prevent life-threatening complications such as respiratory failure and cardiac arrhythmias.

Case Presentation

A 44-year-old Hispanic male with a past medical history of restless leg syndrome, eczema, anxiety, depression, nicotine dependence on no home medications presents for one-week onset of bilateral lower extremity weakness and pain. The patient reports that one week prior to admission, he was experiencing episodes of leg weakness and diffuse muscular pain in the middle of the night, largely localized to his thighs. He presented to the emergency department due to the persistence of his symptoms. He was found to be hypokalemic to 3.3 which was repleted and his symptoms abated and he was discharged home. He was able to ambulate well throughout the day. The patient went to sleep in the night however when he awoke in the middle of the night, the patient was suddenly unable to walk easily and needed to shuffle to his bathroom. Over this time, the patient also noticed pain in his shoulders and palpitations. He presented to the emergency department again as his lower extremity weakness worsened to the point that he needed to use his arms to move his lower extremities and was crawling on the floor to his door. The patient denied nausea, vomiting, diarrhea, saddle anesthesia, urine or bowel incontinence, retention, seizures, neck weakness, double vision, swelling, numbness, tingling, shortness of breath, chest pain, headaches, weight changes, heat or cold intolerances, cough nasal congestions, fever or recent illnesses. He reported that this was the first time he experienced these symptoms. He reported a family history of multiple sclerosis in his cousin and systemic lupus erythematous in his sister. He reported smoking half a pack for the past two decades as well as

daily marijuana use but denied alcohol or intravenous drug use.

In the emergency department, the patient was sinus tachycardic to 110 beats per minute, afebrile and oxygen saturation within normal limits on ambient air. On exam, the patient was in no acute distress with intact strength, sensation and reflexes in all extremities as well as intact cranial nerves. Labs were significant for hypokalemia to 2.5 mmol/L, hypomagnesemia to 1.5 mg/dL, hypophosphatemia to 2.3 mg/dL, and CK mildly elevated to 607 U/L. A CT abdomen and pelvis performed for bilateral hip pain only demonstrated splenomegaly. His electrolytes were repleted and his symptoms resolved.

The initial differential for the patient's presentation included malnutrition, thyrotoxicosis, hypokalemic periodic paralysis, renal tubular acidosis, Guillan Barre Syndrome, transverse myelitis, and multiple sclerosis. Endocrinology and neurology were consulted for further workup.

An iron panel demonstrated ferritin 269 ng/mL, iron concentration 77 ug/dL, iron binding capacity 223 ug/dL, transferrin 178 mg/dL, and percent saturation 35%. A Thyroid Stimulating Hormone level was sent and was significantly decreased to <0.005 µUL/mL. Free T4 and total T3 were elevated to 4.28 ng/dL and 569 ng/dL, respectively. Thyroglobulin antibody was 26 IU/mL (normal < or = 1 IU/mL), thyroid peroxidase antibodies 788 IU/mL (normal < 9 IU/mL), TRAb Antibody 34.45 IU/L (normal <= 2.00 IU/L), and thyroid stimulating immunoglobulin (TSI) 435% baseline (normal < 150% baseline). Urine electrolytes were within normal limits except for random magnesium to creatine ratio elevated to 417 mg/gmCr, and urinalysis was significant for positive urobilinogen.

Overall presentation and findings were concerning for Grave's disease and thyrotoxic periodic paralysis with electrolyte abnormalities. The patient was initiated on methimazole 20 mg BID and propranolol 20 mg BID which was tolerated. Daily electrolytes were monitored and normalized, and the patient was able to ambulate without complication and his pain resolved. On further questioning, it was found that the patient's diet consisted of excessive carbohydrate meals. He was educated to avoid an excessive carbohydrate diet. He was discharged with scheduled follow up with endocrinology for potential thyroid ultrasound and further Grave's disease management.

Discussion

Thyrotoxic periodic paralysis (TPP) is a rare but potentially life-threatening complication of hyperthyroidism. While the patient in this case did not exhibit signs or symptoms indicative of a thyroid storm, prompt recognition and management are crucial to prevent recurrent attacks and serious complications such as respiratory failure, cardiac arrhythmias, and persistent neurologic paralysis. Treatment involves correction of hyperthyroidism with anti-thyroid medications, control of sympathetic overactivity with beta-blockers, and aggressive potassium replacement [1,2]. Patients should be educated about the importance of medication compliance and regular follow-up to prevent recurrence.

Conclusion

Thyrotoxic periodic paralysis (TTP) should be considered in the differential diagnosis of an acute muscle weakness and bilateral muscle pain especially in a patient with family history of autoimmune disease. Initial recognition and appropriate management are essential to prevent life-threatening complications and to ensure a favorable outcome.

References

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