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Case Report
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Thyrotoxic Periodic Paralysis: Case Report

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Abstract

Thyrotoxic Periodic Paralysis (TPP) represents an acquired form of Hypokalemic Periodic Paralysis (HPP) in which attacks of weakness occur, often precipitated by rest after strenuous exercise or a high-carbohydrate load. TPP is more prevalent in Asians than non-Asians. Despite a higher incidence of hyperthyroidism in females, over 95 percent of TPP occur in males. Any cause of hyperthyroidism can be associated with TPP. Graves' disease is the underlying disorder in most of cases with TPP as it is the cause in most of cases with hyperthyroidism. We report a 28-year-old male presenting TPP with very low potassium level.

Keywords: Thyrotoxic periodic paralysis; Hypokalemia; Hyperthyroidism

Case Report

A 28-year-old man was admitted to our emergency room with severe weakness in his lower limbs. The symptoms started three hours after a strenuous exercise. On physical examination, muscle strength of his upper limbs was normal while muscle strength of lower limbs was 0/5. Deep tendon reflexes and planter response were absent. There was no sensory deficit. He was anxious, had palpitation and had tremor on stretched hands. Cranial nerves were intact. After questioning, the patient reported that for a long time he had been suffering from palpitation, irritability, weight loss and heat intolerance that accepted them as normal signs of being stressful. In laboratory findings, initial potassium level was 1,54 meq/l and electrocardiogram showed hypokalemia features such as ST depression an U-Wave. Other laboratory findings were in normal ranges. The patient was monitored and administered intravenous potassium. After 60 meq potassium was administered within 6 hours, the symptoms totally resolved. İn order to make differential diagnosis we performed electromyography and it was normal. The patient's Thyroid function tests revealed that TSH was 0,005 mıU/L (Normal 0,27-4,2), Free T4 66,2 ng/dl (Normal 12-22) and Free T3 11,3 ng/dl (Normal 3,1-6,8). The patient's neck ultrasound and thyroid nuclear scan were compatible with Graves' disease. The patient was given propranolol and methimazole after endocrinology consultation and informed about repetition of hypokalemic paralysis and/or reactive hyperkalemia.

Discussion

TPP is a sporadic form of hypokalemic periodic paralysis that may occur in association with hyperthyroidism [1]. Any etiology of hyperthyroidism can be associated with TPP. Graves' disease is the underlying disorder in most cases with TPP, as it is the cause in most of cases with hyperthyroidism [2,3]. The mechanism by which hyperthyroidism can produce hypokalemic TPP is not well understood. Thyroid hormones increase tissue responsiveness to beta-adrenergic stimulation which increases sodium-potassium ATPase activity on the skeletal muscle membrane [4]. This drives potassium into cells, perhaps leading to hyperpolarization of muscle membrane and relative in excitability of muscle fibers. Thyrotoxic patients with TPP have been found to have higher sodium pump activity than those without paralytic episodes [5]. In this way, excess thyroid hormone may predispose to paralytic episode by increasing the susceptibility to the hypokalemic action of epinephrine and insulin [6]. Hypokalemic paralytic attacks can be precipitated by events such as strenuous physical activity, stress, high-carbohydrate meal, infection and cold exposure but in many cases no obvious precipitant is identified. In acute attack, TPP must be distinguished from other causes of acute quadriparesis such as myasthenic crisis, Guillain-Barre syndrome, acute myelopathy and botulism [2,7]. The finding of hypokalemia and recovery with treatment should alert the clinician in terms of the diagnosis of hypokalemic paralysis, in which the possibility of thyrotoxicosis must always be evaluated particularly in the absence of a family history of periodic paralysis.

Conclusion

TPP is a life-threatening condition, especially in terms of cardiac complications. Hence, early diagnosis and acute treatment with potassium administration is essential. In order to prevent recurrence of attacks and reactive hyperkalemia, euthyroid state

should be achieved as early as possible. Potassium level should be closely monitored and the dosage of propranolol and methimazole must be arranged according to patient's clinical condition. Familial periodic paralysis and other causes that make similar symptoms should be evaluated in differential diagnosis [8].

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