Case Report

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Thyrotoxic Hypokalemic Periodic Paralysis: Case Report

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Abstract

Thyrotoxic Hypokalemic Periodic Paralysis (THPP) is a rare hereditary disorder which is characterized by thyroid hormone elevation, low blood potassium level and recurrent acute muscle weakness. Basic pathology is thought to be the increase in activity in the sodium-potassium pump (Na+/K+ATPase). Here we report the case of a 31-year-old male that presented with weakness in his legs, and inability to walk. The patient had elevated thyroid hormone levels (FT3 and FT4) and lower TSH levels, lower serum potassium levels, and recurrent acute muscle weakness. The diagnosis was made to be Thyrotoxic Hypokalemic Periodic Paralysis precipitated after intense physical activity. THPP is a reversible medical emergency. Early diagnosis, and rapid treatment is lifesaving. Although rare, THPP must be considered as a differential diagnosis in patients presenting with hypokalemia and paralysis.

Key Words: endocrine, thyrotoxic hypokalemic periodic paralysis, sodium potassium pump, hypokalemia

Introduction

Thyrotoxic Hypokalemic Periodic Paralysis (THPP) is a rare endocrinological entity, characterized by thyrotoxicosis, hypokalemia, and acute proximal muscle weakness. Its incidence has been reported to be 2% in Japanese and Chinese populations with thyrotoxicosis, and even lower (0.1-0.2%) in Caucasian populations[1]. Although hyperthyroidism is more frequently seen in women, THPP is more frequent among men. THPP can be triggered by physical activity, cold exposure, stress, heavy carbohydrate or alcohol intake. Pathophysiologic mechanism is when the catecholamine discharge increases secondary to hyperthyroidism, intracellular passage of potassium increases. This leads to an increase in the number and activity of Na+/K+ATPase, and it manifests as paralysis.[2,3]. Additionally, it may present with fatal hypokalemia, reversible muscle weakness, and attacks of paralysis[4]. Fortunately, THPP is a reversible medical emergency. To get serum potassium levels back to normal, beta-blockers can be initiated and euthyroidism can be ensured with hyperthyroidism treatment. Death can be easily prevented with accurate diagnosis and management. Therefore, here we report the case of a 35-year-old male patient with THPP.

Case Report

A 35-year-old male patient presented to our Emergency Department with weakness in his legs, and inability to walk. He worked as a dockworker, and he reported that he previously experienced episodes of muscle weakness a few times at night. Because these weaknesses were not severe, he didn't seek medical help. Two months ago, he consulted another medical center with complaints of dizziness and shivering. Propranolol and methimazole were initiated as treatment, however he didn't use these medications regularly. The patient's additional medical and family history was unremarkable. He had no history of smoking, alcohol substance or additional drug use.

At the time of his admission to our hospital, he was first evaluated by the Neurology Department. Cerebral and spinal imaging studies were clean, but his blood tests revealed the presence of hypokalemia. Hence, he was transferred to the Internal Medicine Department for further evaluation. Here the physical examination findings were as follows: TA: 120/70 mmHg; pulse rate: 90/min, rhythmic; body temperature: 36.9 C; respiratory rate: 16/min. His skin had a natural color but was sweaty. Thyroid gland manifested Stage 1b goiter. He had open consciousness with complete cooperation, full time and place orientation. He was agitated due to complaints of weakness. Muscle strengths were 4/5 and 5/5 for proximal and distal parts of upper; 3/5 and 5/5 for proximal and distal parts of lower extremities, respectively. Sensory loss was not detected. Babinski reflexes were negative, and bilateral DTRs were decreased. Rest of the examination was normal.

For diagnostic purposes, biochemical and hematologic tests were requested, and they revealed lower serum potassium levels of 2.1mmol/L (reference range: 3.5-5 mmol/L), creatinine levels of 0.38mg/ dL (reference range: 0.74-1.35

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mg/dL), hemoglobin levels of 13.5 gm/dL (reference range: 14.0-17.5 gm/dL). On the other hand, the patient's blood urea was higher with the level of 31 mg/dL (reference range: 6-24 mg/dL). His other biochemical findings were within the normal range. Arterial blood gas analysis, complete urinalysis and peripheral smear were unremarkable. ESR was higher than the reference range with a level of 30 mm/hr (reference range: 0-22 mm/hr), but iron /lipid panel, CRP and phosphorus levels were within normal limits. Posteroanterior chest X-ray, EMG, and ECHO did not demonstrate any abnormality. On EKG, a mildly elongated PR interval, and slight T- wave flattening were detected. Thyroid US, and scanning results were consistent with Graves' disease. Thyroid Function tests reported a lower TSH level of 0.01 miU/L (reference range: 0.2-5.5 miU/L), and higher levels of FT3: 45 pmol/L (reference range: 3.1-6.8 pmol/L) and FT4:57 pmol/L (reference range: 10-24.5 pmol/L). Anti-TPO and TSH receptor blocking antibody levels were higher with levels of 194 IU/mL (reference range: < 9 IU/mL) and 27 IU/L (reference range 0.79-3.47 IU/L) respectively.

For treatment, potassium replacement was started. Initially, infusion of 40 mEq/L KCL in 1000 cc isotonic saline was delivered within 4 hours. In the third hour of treatment, the patient's complaints were reduced; and in the fifth hour, the patient was able to walk. After he received a total of 80 mEq/L KCl, his potassium levels normalized and was 4.5 mmol/L (reference range: 3.5-5 mmol/L). The patient was also started on daily doses of 3*10 mg p.o. Methimazole, and 2*40 mg p.o. Propranolol treatment. He was advised about the importance of quitting smoking, using non ionized salt, and complying to drug therapy. His general health state improved without any additional complaints and he was discharged with recommendation of ambulatory control in the Endocrinology Outpatient Clinic. During his 2-month long follow-up, he didn't have any recurrent episodes of the condition. His control tests revealed significant improvement.

Discussion

Thyrotoxic Hypokalemic Periodic Paralysis (THPP) is a rare hereditary disorder which is characterized by thyroid hormone elevation, low blood potassium level and recurrent acute muscle weakness. Ion channel defects in the familial paralysis tables, and relevant family histories are not seen in this disease. THPP can be differentiated from other entities with concomitant presence of hyperthyroidism, and hypokalemia. The basic pathology in THPP is thought to be due to the disequilibrium between intra and extracellular potassium values. This condition is related to increased number, and activity of Na/K/ATPase pump secondary to thyrotoxicosis with resultant increase in intracellular potassium inflow[5]. Case presented here is consistent with the definition of THPP since the patient had elevated thyroid hormone levels FT3: 45 pmol/L (reference range: 3.1-6.8 pmol/L) and FT4:57 pmol/L (reference range: 10-24.5 pmol/L), lower serum potassium levels of 2.1mmol/L (reference range: 3.5-5 mmol/L) and recurrent acute muscle weakness.

Among factors precipitating the attacks, higher amounts of carbohydrate intake, trauma, upper respiratory tract infection, emotional stress, alcohol intake, and heavy exercise have been reported[6,7]. Heavy intake of a diet rich in carbohydrates develops hyperinsulinemia and induces intense beta-adrenergic activity which speeds up intracellular inflow of potassium. Similarly, higher rates of THPP developed in men are associated with the androgenic effect induced increase in Na+/K+ATPase. In our case, the patient was male and an androgenic effect might be present. He presented to the Emergency Department after a work day where he does intense physical activity. It might also be possible that the THPP attack was precipitated after a heavy exercise.

Among etiologic factors of hypokalemia, diuretic use, renal and gastrointestinal causes should be priorly considered, and inquired. One should be attentive about cardiac arrhythmias and respiratory problems in the cases of hypokalemia. Our patient, although had hypokalemia, presented with only mild changes in the ECG and no abnormalities in respiration. In the case of reversible paralysis, it's important to remember possible cardiac and respiratory complications of THPP.

In most of the cases with THPP, Grave's disease was detected; while other reported etiologies include toxic nodular goiter, thyroiditis, TSH-secreting adenoma, and exogenous thyroid hormone intake[8,9]. Here our case was in parallel with the previous literature as the Anti-TPO and TSH receptor blocking antibody levels: 194 IU/mL (reference range: < 9 IU/mL) and 27 IU/L (reference range 0.79-3.47 IU/L) respectively, thyroid US, and scanning results were consistent with Grave's disease.

THPP is a reversible medical emergency that can be treated in two parts: treatment of hypokalemia and treatment of hyperthyroidism. Treatment of hypokalemia can be instituted based on the clinical manifestations of the patient through oral route or intravenous route in compliance with relevant protocols. In the literature, development of rebound hyperkalemia has been reported in 40 % of the patients who received IV KCl at a rate of 10 mmol/hour. Therefore, slow replacement with IV KCL is recommended for these cases[10]. That's why we did not see rebound hyperkalemia in our case. For hypokalemia treatment, non-selective beta-blockers such as Propranolol should be initiated, which will lead to correction of Na+/K+ATPase, hence a marked improvement in hypokalemic state. Our case was consistent with this information as well. For long-term effective treatment in cases with THPP, euthyroidism should be ensured with hyperthyroidism treatment that normalizes the function of the pump. Based on the underlying thyroid pathology, medical treatment, radioactive iodine, or surgery can be preferred.

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Conclusion

Early and accurate diagnosis, and rapid treatment in THPP is lifesaving. Although rare, THPP must be considered as a differential diagnosis in patients presenting with hypokalemia and paralysis.

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