Case Report
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Thyrotoxic Hypokalemic Periodic Paralysis: Case Report

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Abstract

Thyrotoxic hypokalemic periodic paralysis is characterized by muscle paralysis and acute hypokalemia that secondary to hyperthyroidism, often detected in men and in Asians. The increase in Na-K ATPase activity as a result of increased beta adrenergic stimulation due to thyrotoxicosis causes to decrease intravascular potassium level resulting paralysis. In our study, we aim to discuss a case who applied with lower extremity paralysis in the emergency department and diagnosed with THPP.

Keywords: hypokalemia,paralysis,periodic,thyrotoxicosis

Introduction

Hypokalemic periodic paralysis is a neuromuscular disorder usually seen as familial autosomal dominant or sporadic with hyperthyroidism.In both forms, it presents with weakness in the lower extremity in the morning.[1] The mechanism of formation of thyrotoxic hypokalemic periodic paralysis (THPP) is an increase in beta adrenergic stimulation due to thyrotoxicosis, resulting in over-activation of Na-K ATPase pump, consequently in excessive passage of potassium into the cell . It presents with episodes, the duration of the attack can be shortened by K^+ supplement.[2] In this case, we wanted to talk about the steps of THPP detection and treatment via a case of male patient who had muscle weakness.

Case Report

A 44-year-old male patient presented with complaints of weakness in his legs and difficulty in moving in the emergency department. It was learnt from his history that after a heavy dinner, he tried to get out of bed towards morning and had difficulties to move. there was no known additional disease, 3 months ago he was admitted to the neurosurgery polyclinic with complaints of pain and weakness in his legs and his examinations were still continuing. In neurological evaluation; he was conscious, his cooperation and orientation was good, muscle strength in his bilateral upper extremities was 5/5, in his lower extremities was 3/5, sensory and reflex examination were normal. Other systemic examinations were natural and the patient with no family history of a disorder was taken to observation room. Muscle strength in the first hour of follow-up was 5/5 in the bilateral upper extremity and 1/5 in the lower extremity. Potassium (K^+) level was 1.9 mmol/L in the innitial tests and K^+ level was 1.3 mmol / L in the control blood sample, thereon the patient was replaced with 30 mEq K^+ at 10 mEq/h. Despite the replacement, K^+ value was 1.4 mmol / L and therefor the patient with a preliminary diagnosis of thyrotoxic hypokalemic periodic paralysis was performed a test for assessment of thyroid stimulating hormone (TSH). TSH value was <0.006 ulU / ml (0.27-4.2).

At the 15th hour of the patient's follow-up with K + replacement therapy, it was observed that the decrease in muscle strength was regressed and the muscle strength of the lower extremities was 3/5. The potassium level of the patient who had 70 mEq K^+ replacement therapy at 10 mEq / h was 4,7 mmol/L. After the 24th hour in the emergency department, it was learned that the patient who was transferred to the internal medicine department was diagnosed with autoimmune thyroiditis and was discharged with good health on the 5th day of hospitalization.

Discussion

THPP is a disease characterized by muscle paralysis, acute hypokalemia without any deficit of total body potassium and hyperthyroidism.[3] Failure in the awareness and treatment of

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this disease may lead to lethal cardiopulmonary complications due to low serum potassium[4]. Hyperthyroidism is one of the risk factors for THPP. Although the most common cause of hyperthyroidism is Graves' disease, any cause of thyrotoxicosis can cause THPP.[4] In our case; TSH value was <0.0006uIU /ml, free T4 value was 2.46 ng/dl and TSH receptor blocking antibody level was 3.30 IU/L. The patient was diagnosed with autoimmune thyroiditis (Graves' disease). Although thyrotoxicosis and thyroid diseases are more common in women, as opposed to this situation THPP is more common in men.[5] The patient who is mentioned in our case was also a man.

It is not known exactly that with what mechanism hyperthyroidism causes hypokalemic periodic paralysis, but the common view is that thyroid hormones increase the response of beta-adrenergic stimulation and cause an increase in Na-K ATPase activity. Potassium passes into the cell with increased Na-K ATPase activity and as secondary to hyperpolarization,a decrease occurs in muscle excitability. It is also thought that insulin resistance, which is accompanied by hyperinsulinemia, may also play a role in the pathogenesis. In the study conducted by Soonthornpun and colleagues, subjects with THPP had more severe insulin resistance than thyrotoxic patients without paralysis.[6] This may be explained by the synergistic action of insulin with thyroid hormones, thereby increasing Na-K ATPase activity. For this reason, paralysis attacks develop after an exercise and heavy carbohydrate consumption. In a patient with THPP, the clinic is fairly typical: a young man, the story of a high-carbohydrate meal or a heavy exercise, wakes up not being able to get out of bed because of lower extremity paralysis which is started after the midnight.[7] In history of our patient, there was a complaint of weakness in the legs after eating a heavy dinner, which had too much fat and carbohydrate content.

Attacks usually occur last hours of the night.[2] This can be explained by the fact that some hormones that affect the Na-K ATPase pump and increase the entry of K^+ into the cell have a diurnal rhythm. In our case, according to history of our patient, there was no loss of muscle strength while he was going to bed at night, but he could not get out of bed in the morning.

Treatment of THPP should include the management of hypokalemia and the treatment of the underlying hyperthyroid state. Oral or intravenous potassium administration is on the agenda to accelerate muscle building and prevent cardiopulmonary complications.[8] Doses required for K^+ replacement ranges from 10 to 200 mEq and if hypokalemic cardiopulmonary complications didn't develop, replacement should be done slowly. Non-specific beta-blockers such as propronolol are recommended as alternative therapy.[8] Administration of propranolol can be a more controlled option in cases of that strict evaluation of K^+ level. We anticipated that the K^+ level was going to continue to decrease and we started to K^+ replacement therapy under cardiac monitorization. In our patient whose post-replacement values improved and the paralysis regressed, no complications related to the replacement occurred during the follow-up.

Conclusion

We present a 44-year oldu male patient diagnosed with THPP in the emergency department and wanted to talk about treatment experience resulted positively to draw attention to the disease which is rarely seen and therefore difficult to be brought to mind by physicians.

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