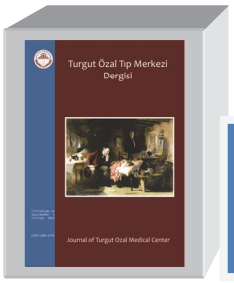

OLGU SUNUMU/CASE REPORT
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Ventricular Fibrillation due to Severe Hypokalemia in a Patient with Thyrotoxic Periodic Paralysis

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Abstract
Hypokalemic thyrotoxic periodic paralysis (TPP) is a rare complication of hyperthyroidism. It accompanies episodes of hypokalemia and muscle weakness. In general, hypokalemia, normokalemia, and hyperkalemia can all be observed. There are generally some precipitating factors such as stress, vigorous exercise, and high carbohydrate consumption all of which make way for the occurrence of attacks. The duration of attacks range from 2-36 hours and can be shortened by potassium supplementation. Except for attacks of paralysis, it is a disease that affects totally healthy people. A 31-year-old female patient was admitted to the emergency unit with the complaints of bilateral lower and upper extremity weakness and paralysis. The patient's history and laboratory findings were consistent with our diagnosis of thyrotoxic periodic paralysis. Thyrotoxic periodic paralysis is a rare but life-threatening clinical entity. We would like to present an uncommon case of thyrotoxic periodic paralysis and along with a review the literature.
Key Words: Thyrotoxicosis; Hypokalemia; Hypokalemic Periodic Paralysis.

Tirotoksik Hipokalemik Periyodik Paralizili Bir Hastada Hipokaleminin Tetiklediği Ventriküler Fibrilasyon

Özet
Tirotoksik hipokalemik periyodik paralizi (TPP) hipertiroidinin nadir bir komplikasyonu olup, hipokalemi ve kas güçsüzlüğü atakları ile seyretmektedir. Klinik pratikte hipokalemi, normokalemi ya da hiperkalemi saptanabilir. Genellikle atakların oluşmasını tetikleyen stres, aşırı egzersiz, karbonhidrattan zengin beslenme gibi presipitan faktörler vardır. Atakların süresi ortalama 2-36 saat kadar olup gerekli durumlarda potasyum replasmanı ile bu süre daha da kısalmaktadır. Paralizi atakları dışında kişinin tamamen sağlıklı olduğu bir hastalıktır. Acil polikliniğimize başvuran 31 yaşında kadın hastanın, bacaklarında ve kollarında güçsüzlük, yürüyememe şikayetleri mevcuttu. Anamnez ve laboratuvar incelemeleri ile hastaya tirotoksik periyodik paralizi tanısı kondu. Tirotoksik periyodik paralizi nadir ancak hayatı tehdit edebilen bir klinik durumdur. Bu olgu sunumunda nadir olması nedeniyle tipik tirotoksik periyodik paralizi tanılı bir hasta literatür eşliğinde gözden geçirilip sunulmuştur.
Anahtar Kelimler: Tirotoksikozis; Hipokalemi; Hipokalemik Periyodik Paralizi.

INTRODUCTION

Thyrotoxic hypokalemic periodic paralysis (TPP) is a rare disease that is often seen in East Asian males. In TPP, paralysis occurs in periodic attacks along with thyrotoxicosis and hypokalemia and the patient is completely healthy except for these paralysis attacks (1). As an infequent complication of hyperthyroidism, TPP has an incidence rate of 0.1-0.2%. While TTP is uncommon in western societies, it is more frequently observed in Asian countries including especially China and Japan (2). TPP's incidence is not known among the Caucasian nations, including Turkey, although a growing number of case series have been reported. A series with a collection of 40 cases from Turkey that has been published previously supports its rarity (3). TPP often takes place during the the recovery period after excessive exercise following intense carbohydrate food intake though it may also occur spontaneously. Paralysis attacks tend to come out at nights. Paralysis usually effects lower extremities and proximal muscles. In hyperthyroidism, there is an increase in Na-K ATPase activity. Treatment is achieved by the correction of serum potassium levels and taking control of hyperthyroidism (1,4).

CASE REPORT

A 31 year old female patient, who did not have any health issues prior to a plane trip during which she had to remain seated for a long time, presented with complaints of upper and lower extremity weakness at the emergency department. Our patient had a history of asthma and steroid inhaler use though her family history was uneventful. During the physical examination, blood pressure was 90/60 mm Hg, pulse was 105/mins and body temperature was 36.5 C, respectively. Her general condition was moderate; she was alert, oriented, and cooperative during the examination. The thyroid was non-palpable. The pupils were isochoric, eye movements were independent, and pupillary light reflex was bilateral

in the neurologic examination. The muscle strength was 4/5 in the proximal upper limb, 3/5 in the proximal lower limb, and 2-3/5 in the distal. Suffering from shortness of breath and palpitations, the patient had a respiratory arrest during the examination upon which we applied endotracheal intubation and started to monitor the patient. During the follow-up, the patient had ventricular tachycardia. For this, the department of cardiology performed an electrical cardioversion. After an hour, due to ventricular fibrillation, we administered defibrillation and cardiopulmonary resuscitation. Then the patient was taken tointensive care unit (ICU) for further tests. The routine laboratory test results were as follows: glucose 91 mg/dL, urea 34 mg/dL, creatinine 0.8 mg/dL, aspartate aminotransaminase (AST) 25 U/L, alanine aminotransferase (ALT) 30 U/L, sodium (Na) 140 mmol/L, potassium (K) 1,4 mmol/L (3.5-5.1), chlorine (Cl) 101 mmol/L, calcium (Ca) 9.8mg/dl, phosphate (P) 2.6 mg/dL, creatine phosphokinase (CPK) 180 U/L (0-190), CK-MB 15 U/L, and troponin T was negative. Because we identified hipopotasemia in the patient, we started intravenous potassium replacement. The thyroid function tests we conducted due to palpitations showed the following results: TSH: 0,017 microU/ml (0.4 to 4), sT4: 2.32 pg/ml (0.93-1.71), sT3: 6.27 pg/ml (1.8-4.6), Anti-TPO (anti-microsomal antibody): 1000 IU/ml (0-34), and Anti-TG (anti-thyroglobulin antibodies): 128 IU/ml: (0-115). The thyroid ultrasound carried out in the intensive care unit displayed that the right thyroid lobe dimensions were 17×21×51 mm, the left thyroid lobe dimensions were 15×22×55 mm, the isthmus size was 5 mm. The results also showed that the thyroid parenchyma was highly blooded; there were fibrous bands that were widely spread; there was a highly heterogeneous appearance, and these were consistent with the thyroiditis. In the Tc-99m pertechnetate thyroid scintigraphy, we detected diffused hyperplasia in both lobes. Having diagnosed the patients with thyrotoxic hypokalemic periodic paralysis, we started propylthiouracil 4×150 mg/day, propranolol, 3×20 mg/day, and for the respiratory distress issues, we applied a single dose of (60 mg) methylprednisolone. As the respiratory functions improved in the ICU, the tracheal tube was removed. The K levels reached normal limits in the follow-up and the paralysis gradually improved. After an oral therapy of propylthiouracil 4×150 mg/day and propranolol 3×20 mg/day, the patient was discharged.

DISCUSSION

Thyrotoxic hypokalemic periodic paralysis shares similarities with familial hypokalemic periodic paralysis in terms of clinical features although its pathogenesis is still unknown. The most common underlying cause of TTP is Graves' disease. TPP's incidence is about 2% in hyperthyroid patients (5,6). Although hyperthyroidism is more common in females, thyrotoxic periodic paralysis is common in males with a 95% of incidence rate and 80% of these patients are between the ages of 20-39 (7). In a study of 135 patients conducted over 10 years, the male/female ratio was 130/5. The study shows that 70% of the paralytic attacks took place in summer and in the morning; moreover, in only 34% of these patients the precipitating factors were high carbohydrate intake, high salt diet, and infection (8). In the pathogenesis, because mechanisms responsible for the growth of the response of thyroid hormone increase to beta-adrenergic stimulation in the tissue, the potassium is taken into the cell and brings about the paralysis as a result of the hyperpolarization of the muscle cell membrane. It is known that hypokalemia in familial periodic paralysis type is a result of defects in the ion channels. However, ion channel mutations common in familial forms such as calcium channel alpha-1 subunit (Cav1.1: CACNA1S) and voltage-gated potassium channel (Kv3.4: KCNE3) could not be assessed in relation to TPP (9, 10). A number of studies have illustrated the relationship between human leukocyte antigen haplotypes (HLA Bw46, B51) and TPP (11). Hypokalemia may sometimes be life-threatening and therefore it is included in endocrinological emergency cases. Therefore, although the paralysis in TPP is a clinical condition that can resolve without sequels, it actually requires rapid diagnosis and treatment due to hypokalemia-related risks it carries. Indeed, there are reported deaths due to ventricular fibrillation and respiratory failure in the literature (12). The goal of the treatment should be fixing the paralysis and preventing attacks by providing an euthyroid state (13). To this end, nonselective beta-blockers reduce the frequency and severity of attacks. Attacks can be prevented by treating the underlying cause (14). In the literature, there are studies that claim that hypokalemia can be induced in patients using methylprednisolone; however, these cases should be under close monitoring in terms of hypokalemia (15). Although TPP is more common in men, the fact that our case was a female patient with a history of prolonged inactivity as opposed to exercise as the common precipitating factor, and that the patient developed respiratory failure and ventricular fibrillation make our study a notable case.

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