Thyrotoxic Periodic Paralysis: Case Report

Tirotoksik Periyodik Paralizi

Çiğdem TURA BAHADIR,[a]
Elif KILIÇ KAN,[a]
Feyzi GÖKOSMANOĞLU,[a]
Hulusi ATMACA[a]

[a]Division of Endocrinology and Metabolism,
Ondokuz Mayıs University
Faculty of Medicine, Samsun

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Yazışma Adresi/*Correspondence:*
Çiğdem TURA BAHADIR
Ondokuz Mayıs University
Faculty of Medicine,
Division of Endocrinology and Metabolism, Samsun,
TÜRKİYE/TURKEY
cigdemtura@hotmail.com

ABSTRACT Thyrotoxic periodic paralysis (TPP) is a rare manifestation of hyperthyroidism. Any form of thyrotoxicosis can cause TPP. Typically TPP presents in Asian males of 20-40 ages. It has a incidence of 1.9% in Japan and 0.01-0.02% in USA. It courses with attacks and remission. Attacks usually occur at night or early morning. Early semptoms are usually pain, cramp and stiffness of the muscles. If respiratory, ocular and bulbar systems are affected, it may lead to lethal consequences. Here we report a patient with thyrotoxic periodic paralysis that was formerly diagnosed with periodic paralysis one year ago. His symptoms were recurring following consumption of chocolate and soft drinks and long-lasting standing. He had a few recurrences due to delay in diagnosis of thyrotoxicosis. After antithyroid treatment there was no attack during patient's follow up.

Key Words: Paralysis; thyrotoxicosis

ÖZET Hipertiroidizme bağlı tirotoksik periyodik paralizi (TPP) nadir görülür. Tirotoksikozun herhangi bir formu TPP'ye sebep olabilir. Tipik olarak 20-40 yaş arası Asyalı erkeklerde görülür. İnsidansı Japonyada %1,9, ABD'de %0,01-0,02 oranındadır. TPP, atak ve remisyonlar ile seyreder. Ataklar genelde gece veya sabahın erken saatlerinde meydana gelir. Kaslarda ağrı, kramp ve sertlik genelde ilk semptomlardır. Eğer respiratuvar, okuler ve bulber sistem tutulursa letal sonuçlar meydana gelebilir. Biz bu vaka sunumunda bir yıl önce periyodik paralizi tanısı almış tirotoksik periyodik paralizili bir hastayı sunuyoruz. Hastanın semptomları çikolata, gazlı içecek ve uzun süre ayakta kalması ile tekrar ediyordu. Tirotoksikoz tanısı geciktiği için birkaç kez atağı tekrar etmişti. Antitiroid tedavi sonrası hastanın takiplerinde bir daha atağı olmadı.

Anahtar Kelimeler: Paralizi; tirotoksikoz

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Thyrotoxic periodic paralysis (TPP) is a rare manifestation of hyperthyroidism. It can lead to hypokalemia and lethal complications through muscle paralysis. It has also normokalemic and hyperkalemic forms which are rare.[1] Typically TPP presents in Asian males of 20-40 ages.[2,3] It has a incidence of 1.9% in Japan and 0.01-0.02% in USA.[1]

CASE REPORT

A 35 year-old male patient admitted to neurology department with complaints of motor weakness in all extremities, especially the limbs. After ini-

tial tests he was diagnosed with thyrotoxicosis and taken into follow-up in our clinic. One year ago, he has found himself to be unable to move his arms and legs after woken up in the morning. His symptoms had spontaneously resolved towards evening. The symptoms were recurring following consumption of chocolate and soft drinks and long-lasting standing. With those symptoms, he was diagnosed with periodic paralysis by neurology department at that time. He lacked thyroid function tests for that time. Physical examination revealed twitching in the eyes and physiological tremor in the hands; proximal muscle strength of lower limbs was 4/5 bilaterally. Deep tendon reflexes (DTR) was absent. His sensory examination was normal. No other pathological finding was present. He had no positive family history.

Laboratuary findings at the time of attack were as follows: Total calcium and ALP levels were elevated. Phosphate (P) level was in normal range, potassium (K) level was decreased. Thyroid related tests were as follows during his follow-up: thyroid stimulating hormone (TSH) level was decreased. Free thyroxine (fT4) and free triiodotyronine (fT3) levels were elevated. Anti-thyroglobulin and anti-thyroid peroxidase (anti-TPO) were positive (Table 1).

Hertel exophthalmometry examination were +18 for both eyes. Thyroid scintigraphy revealed diffuse hyperactive. Ultrasound examination of thyroid revealed bilateral glandular enlargement and heterogenity. In electromyography (EMG) there was diffuse myopathy. Electrocardiogram was normal.

Oral metimazol (20 mg/day) and propranolol (40 mg/day) therapy was given. There was no attack during patient's follow up.

TABLE 1: Biochemical profile of the patient.

Component	Before treatment	Reference range
Total calcium (mg/dL)	10,8	8,1-10,7
ALP (U/L)	336	95-280
P (mg/dL)	4.52	2.3-4.7
K (mEq/L)	3.1	3.5-5.5
TSH (mIU/mL)	0,005	0,27-4,2
fT4 (ng/dL)	7,77	0,93-1,7
fT3 (pg/mL)	29,3	2-4,4
Anti- thyroglobulin (IU/mL)	622	0-115
anti-TPO (IU/mL)	376	0-34

ALP: Alkaline phosphatase; P: Phosphate; K: Potassium; TSH: Thyroid stimulating hormone; fT4: Free thyroxine; fT3: Free triiodotyronine; anti-TPO: Anti-thyroid peroxidase

DISCUSSION

TPP is a rare manifestation of hyperthyroidism.[1] Thyrotoxicosis is frequent in women but TPP is seen 22-76 times higher in men.[1] There are no family history.[4] Thyroid autoantibodies are positive in 66% of TPP.[4] Attacks usually occur at night or early morning.[5] Early semptoms are usually pain, cramp and stiffness of the muscles. Though proximal myopathy in lower extremities is predominant, all four extremities are affected in 80% of the patients.[2] Respiratory, ocular and bulbar systems are rarely affected and may lead to lethal consequences. Sensory nerves and mental state is not affected, and deep tendon reflexes (DTR) are absent.[6]

Thyroid hormones activate Na^+/K^+-ATPase pump on the cell membrane. Thus extracellular potassium enters intracellular space.[7] Increased urinary calcium excretion, decreased urinary potassium, hypophosphatemia and hypomagnesemia can ben seen in TPP.[6,8] Serum calcium levels remain normal. 75% of patients have mildly elevated serum ALP levels and 70% of patients have increased serum CPK levels.[5] In our patient, calcium levels regressed to normal levels as thyrotoxicosis decreased. Thus, hypercalcemia considered to be due to thyrotoxicosis.

Severity of attacks is not always correlated with the severity of hyperthyroidism.[1] There is no diagnosed thyrotoxicosis in 76% of patients prior to attacks.[9] Spontaneous resolution is seen between 3-48 hours.[5] Any form of thyrotoxicosis, such as toxic nodular goiter, excessive thyroid replacement, amiodarone induced thyrotoxicosis, thyroiditis, TSHoma and especially Graves disease, can cause TPP.[1,10-14] Diagnostic test results were concordant with Graves disease.

Attacks may be aggrevated with high carbohydrate diet, alcohol consumption, exposure

to cold, physostigmine, pilocarpine, diuretic usage, emotional stress, trauma, menstruation, infection, glucocorticoid administration, catecholamine, insulin, amiodarone, high-sodium diet and during resting period following excessive exercise.[1,6,15]

Treatment of acute attacks in TPP:

1. K^+ infusion: KCl 10mEq/hr iv and/or KCl 2g every 2 hours orally is administered.[16] Instead of saline, glucose solution is preferred for potassium infusion.[17]

2. Nonselective betablockers such as propranolol: Beta blockers decrease frequency of attacks and paralysis following carbohydrate intake by affecting adrenergic activity and inhibiting NA/K-ATPase pump acitivity.[16,18] Iv propranolol can be administered if KCl replacement remains ineffective.[19,20] When given at a dose of 40mg four times a day, propranolol can also prevent attack recurrence.[18]

3. Antithyroid treatment

Definitive therapy;[1]

1. Radyoactive iodine (RAI): There's a risk of recurrence for the first 7-10 days following RAI treatment, so propranolol treatment should be continued during this period.[21]

2. Thyroidectomy

3. Avoiding precipitating factors

Periodic paralysis characterized by muscle weakness may be the first symptom of thyrotoxicosis. So, thyroid function tests should be evaluated in all patients with periodic paralysis. Delay in diagnosis and recurrences can be prevented by this way. Also periodic paralysis history should be investigated in thyrotoxic patients. Otherwise, neglecting precipitating factors can lead to occurence of TPP attacks. Also, TPP attacks can recur and lead to severe morbidity during follow-up of Graves patients who are thought to be in remission, subclinical thyrotoxicosis patients who do not need medical treatment and recurrence of thyrotoxicosis after RAI treatment.

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