Thyrotoxic Periodic Paralysis: Case Report

Tirotoksik Periyodik Paralizi

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 an 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 symptoms 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


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. Typically TPP presents in Asian males of 20-40 ages. It has an incidence of 1.9% in Japan and 0.01-0.02% in USA.

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.

Laboratory 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 triiodothyronine (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.

**DISCUSSION**

TPP is a rare manifestation of hyperthyroidism. Thyrotoxicosis is frequent in women but TPP is seen 22-76 times higher in men. There are no family history. Thyroid autoantibodies are positive in 66% of TPP. Attacks usually occur at night or early morning. Early symptoms 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. 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.

Thyroid hormones activate Na+/K+-ATPase pump on the cell membrane. Thus extracellular potassium enters intracellular space. Increased urinary calcium excretion, decreased urinary potassium, hypophosphatemia and hypomagnesemia can ben seen in TPP. Serum calcium levels remain normal. 75% of patients have mildly elevated serum ALP levels and 70% of patients have increased serum CPK levels. 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. There is no diagnosed thyrotoxicosis in 76% of patients prior to attacks. Spontaneous resolution is seen between 3-48 hours. Any form of thyrotoxicosis, such as toxic nodular goiter, excessive thyroid replacement, amiodarone induced thyrotoxicosis, thyroiditis, TSHoma and especially Graves disease, can cause TPP. Diagnostic test results were concordant with Graves disease.

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

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**TABLE 1:** Biochemical profile of the patient.

<table>
<thead>
<tr>
<th>Component</th>
<th>Before treatment</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total calcium (mg/dL)</td>
<td>10.8</td>
<td>8.1-10.7</td>
</tr>
<tr>
<td>ALP (U/L)</td>
<td>336</td>
<td>95-280</td>
</tr>
<tr>
<td>P (mg/dL)</td>
<td>4.52</td>
<td>2.3-4.7</td>
</tr>
<tr>
<td>K (mEq/L)</td>
<td>3.1</td>
<td>3.5-5.5</td>
</tr>
<tr>
<td>TSH (mIU/mL)</td>
<td>0.005</td>
<td>0.27-4.2</td>
</tr>
<tr>
<td>fT4 (ng/dL)</td>
<td>7.77</td>
<td>0.93-1.7</td>
</tr>
<tr>
<td>fT3 (pg/mL)</td>
<td>29.3</td>
<td>2-4.4</td>
</tr>
<tr>
<td>Anti-Thyroglobulin (IU/mL)</td>
<td>622</td>
<td>0-115</td>
</tr>
<tr>
<td>anti-TPO (IU/mL)</td>
<td>376</td>
<td>0-34</td>
</tr>
</tbody>
</table>

ALP: Alkaline phosphatase; P: Phosphate; K: Potassium; TSH: Thyroid stimulating hormone; fT4: Free thyroxine; fT3: Free triiodothyronine; anti-TPO: Anti-thyroid peroxidase
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 activity.16,18 Iv propranolol can be administered if KCl replacement remains ineffective.19 When given at a dose of 40mg four times a day, propranolol can also prevent attack recurrence.18

3. Antithyroid treatment

Definitive therapy;4

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 occurrence 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.

REFERENCES

17. Chen DY, Schneider PF, Zhang XS, He ZM, Chen TH. Fatality after cardiac arrest in thyrotoxic periodic paralysis due to profound hypokalemia resulting from intravenous glucose administration and inadequate potassium replacement. Thyroid 2012;22(9):969-72.