Thyrotoxic periodic paralysis: Report of two cases and a short review of the literature

Tirotoksik Periyodik Paralizi: İki Olgu Sunumu ve Literatürün Kısa Derlemesi

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Abstract
Thyrotoxic periodic paralysis (TPP) is a fatal complication of hyperthyroidism which is characterized by muscle weakness and hypokalemia. In the literature, Asian populations are reported to have a vulnerability to TPP. Considering the life-threatening aspect of the disorder, we decided to report two cases with TPP who have been successfully diagnosed and treated. The first case was a 26-year-old man with the diagnosis of Graves' disease. He had the complaint of sudden leg muscle weakness. At laboratory examination, hypokalemia was detected. The diagnosis of thyrotoxic periodic paralysis was made and intravenous potassium replacement was applied. The second case was a 29-year-old man, complaining of generalized muscle weakness. He was paraparesic on admission. Laboratory examination revealed hypokalemia. Following intravenous potassium replacement, his neurologic status completely improved. His further investigation was compatible with Graves' hyperthyroidism. Thyrotoxic periodic paralysis is a rare, but life-threatening clinical entity. It should be kept in mind in the management of hypokalemic cases with muscular and neurologic manifestations. Turk Jem 2007; 11: 62-3

Key words: Thyrotoxic periodic paralysis, thyrotoxicosis, antithyroid agents, hypokalemia

Özet

Anahtar kelimeler: Tirotoksik periyodik paralizi, tirotoksikozis, antihüyoid ajยาntlar, hipokalemia

Introduction
Thyrotoxic periodic paralysis (TPP) is a fatal complication of hyperthyroidism which is characterized by muscle weakness and hypokalemia. In literature, Asian populations are reported to have a vulnerability to TPP and its overall incidence among Chinese and Japanese thyrotoxic patients is about 1.8% and 1.9 %, respectively (1,2). Although thyrotoxicosis commonly affects females, TPP has a strong male predominance. Male to female ratio ranges from 17:1 to 70:1(1-4). Patients are usually young adult males at their 30s or 40s (5-7). Male dominance of the disorder is attributed to the ATPase activity which is increased by androgens and inhibited by estrogens (8). Considering the life-threatening aspect of the disorder, we decided to report two thyrotoxic cases with TPP who had been successfully diagnosed and treated. We aimed to contribute to ongoing medical education of the physicians and focus them on this rare clinical entity.

Case 1
A 26-year-old man, with the diagnosis of Graves’ disease, was admitted to the emergency unit with the complaint of sudden muscle weakness at his legs. He had been treated with methimazol for 6 months. His thyroid function tests kept being above the normal limits, despite of maximal methimazol dosage, so a surgical procedure was chosen as the final therapeutic option. Glucocorticoids and colestro-binding resins were added to therapy prior to surgery.
On admission, he was unable to walk without assistance. His vital signs were normal and the pathological findings at physical examination were bilateral exophthalmus, diffuse palpable thyroid gland and depressed deep tendon reflexes. Laboratory analyses revealed hypokalemia with normal renal and hepatic functions. His electrocardiography (ECG) exhibited sinus tachycardia with T wave depression. Further analyses detected depressed serum thyroid stimulating hormone (TSH) with high free-tri-iodothyronine (fT3) and free-thyroxine (fT4) levels (Details are shown in Table 1). The diagnosis was TPP and treatment with intravenous potassium chloride was immediately applied. Serum potassium levels normalized with simultaneous resolution of weakness at his legs.

Case 2

A 29-year-old man presented to the emergency unit, complaining of sudden generalized weakness. On the day of admission, he had the story of working hard physically at his farm. His body temperature was 37.0°C, blood pressure was 160/120 mmHg, and pulse rate was 120/min. At physical examination, he was found to be paraesthesic. His neurologic status deteriorated during the following 30 minutes and he was quadriparesic then. Lower limbs were more affected. Deep tendon reflexes were lost with normal sensation. His ECG showed right bundle branch block. Laboratory investigation demonstrated low serum potassium with high creatine phosphokinase (CPK); 579 IU/L (reference range 22-200). His hepatic and renal functions were found to be normal. Serum potassium levels improved with intravenous potassium replacement and reached up to physiological limits. Further laboratory data showed supressed TSH and elevated fT3 and fT4 levels (Table 1). Electroneuromyography (ENMG) detected no abnormality. A diagnosis of TPP was made. Endocrinological evaluation including; thyroid ultrasonography and scintigraphy were compatible with Graves’ disease. The radioiodine treatment can strongly be recommended. However, our first case was consuming a β-blocker besides the drugs mentioned above when he was admitted with full blown symptoms. Glucocorticoids can reduce serum potassium levels with their mineralocorticoid activity and via this effect, patients may have a tendency toward hypokalemia (15). The hypokalemic episode of this patient may be explained by glucocorticoid consumption.

Hypokalemic cases who present following a heavy exercise should remind us of the possibility of thyrotoxicosis, as detected in our second case. In literature, the presence of only one case with TPP from Turkey makes these subjects worth reporting (16). Physicians, especially working at emergency units, should be aware of the fact that thyrotoxicosis may present with hypokalemia. Following immediate intravenous potassium replacement, it is mandatory to maintain euthyroidism in order to prevent recurrent attacks of paralysis. Considering the underlying thyroid pathology, options of long-term antithyroid therapy, radioiodine treatment or surgery may be chosen.

**References**


**Table 1. Biochemical analyses of the cases on admission**

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<thead>
<tr>
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<th>Case 1</th>
<th>Case 2</th>
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<tbody>
<tr>
<td>Potassium (mEq/L) (3.5-5.30)</td>
<td>2.56</td>
<td>2.21</td>
</tr>
<tr>
<td>TSH (µIU/mL) (0.3-4.94)</td>
<td>0.0</td>
<td>0.0</td>
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<tr>
<td>fT3 (pmol/L) (2.2-5.34)</td>
<td>10.7</td>
<td>24.87</td>
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<tr>
<td>fT4 (pmol/L) (9-25)</td>
<td>42.11</td>
<td>51.63</td>
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