Thyrotoxic Periodic Paralysis in a Turkish Male; The Recurrence of the Attack after Radioiodine Treatment

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Abstract. Thyrotoxic hypokalemic periodic paralysis (THPP) is a rare complication of hyperthyroidism and an uncommon form of hypokalemic periodic paralysis. Its differentiation of more common forms of periodic paralysis is important because aggressive treatment can place the patient at risk for rebound hyperkalemia. Treatment of the underlying thyroid dysfunction cures the muscle symptoms. Here we describe a 37-year-old Turkish male with THPP whose paralysis attack recurred soon after administration of radioactive iodine.

Key words: Thyrotoxicosis, Hypokalemic periodic paralysis, Iodine radioisotopes

THYROTOXIC hypokalemic periodic paralysis (THPP) is a rare complication of hyperthyroidism in the Western population. It occurs primarily in males of Asian descent, including patients of Japanese, Chinese, Vietnamese, Korean, Filipino, American Indian, and Hispanic ancestry [1–10]. Although the association of thyrotoxicosis and periodic paralysis has been well-known since 1931 [11] it is not reported from Turkey in English literature, most probably because of unfamiliarity with the disorder. We herein report a 37-year-old Turkish male patient with THPP who also experienced a paralysis attack ten days after the administration of radioiodine despite the treatment with propranolol.

Case report

A 37-year-old Turkish male patient presented with the complaint of approximately ten episodes of muscle weakness for 2 years. These attacks occurred in all but one episode at night, following exertion or consump-
sistent with Graves’ disease and THPP and he was put on carbimazole and propranolol treatment once again. After one month of antithyroid therapy he had 10 mCi radioactive iodine ($^{131}$I).

The patient was admitted to another medical center ten days after radioactive iodine treatment for a several-hour episode of paralysis. Physical examination revealed symmetric flaccid paralysis with areflexia in all four limbs. In that period carbimazole was stopped but propranolol was continued. On this occasion his potassium level was obtained and found to be 2.6 mEq/L. Thyroid function testing revealed elevated FT$_4$ (3.5 ng/dL), FT$_3$ (7.34 pg/mL; normal, 1.8–4.6 pg/mL) and suppressed TSH (0.005 $\mu$U/mL). The patient regained full muscle power without any potassium supplementation. The patient has become hypothyroid after 3 months and put on thyroxine replacement therapy. He has had no recurrence of paralysis for 6 months. The patient’s clinical course is summarized in Table 1.

### Table 1. The patient’s clinical course including thyroid hormones and thyrotropin values

<table>
<thead>
<tr>
<th>Possible triggering event</th>
<th>FT4 (ng/dL) (N: 0.8–1.9)</th>
<th>T4 (µg/dL) (N: 4.5–12)</th>
<th>TSH (µU/mL) (N: 0.4–5.0)</th>
<th>K (mEq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Last attack before RI</td>
<td>5.34</td>
<td>NA</td>
<td>0.04</td>
<td>NA</td>
</tr>
<tr>
<td>Recurrent attack after RI</td>
<td>3.5</td>
<td>NA</td>
<td>0.005</td>
<td>2.6</td>
</tr>
<tr>
<td>3 months follow up after RI</td>
<td>NA</td>
<td>4.9</td>
<td>15.66</td>
<td>NA</td>
</tr>
<tr>
<td>6 months follow up after RI</td>
<td>NA</td>
<td>6.4</td>
<td>4.16</td>
<td>4.49</td>
</tr>
</tbody>
</table>

FT4: free thyroxine; T4: total thyroxine; TSH: thyroid stimulating hormone; K: potassium; RI: radioiodine; NA: not available.

Discussion

THPP predominantly affects males, most often of Asian descent. Indeed, THPP has been reported to occur in 4.3% to 8.2% of hyperthyroid Japanese males [1, 12] and 12.9% of hyperthyroid Chinese males [2]. The association of paralysis with thyrotoxicosis is infrequent in the Caucasian population and to the best of our knowledge this is the first Turkish patient with THPP in English literature.

THPP is characterised by sudden transient recurrent episodes of painless weakness or paralysis without alteration in consciousness or sensation. In patients with THPP typical features of hyperthyroidism are often subtle or absent as in our patient. The diagnosis of thyrotoxicosis usually postdates the onset of paralysis, and biochemical hyperthyroidism is usually confirmed at the time of presentation [13]. It commonly occurs in Graves’ disease but has been reported with all causes of thyroid hormone excess [2, 10, 14–16].

Similar to our patient paralysis attacks in hyperthyroidism tend to occur during the night [17, 18] and the proximal muscles of the lower extremities are more affected than the other muscle groups [9, 17]. Although exercise may attenuate paresis in THPP, there is a period of rest after exercise that precipitates paralysis [17].

The pathogenesis of THPP is uncertain. It is well-known that the attacks of paralysis no longer occur once thyrotoxicosis is cured; therefore, a necessary condition for the disorder is the presence of excessive amount of thyroid hormones in serum. Thyroxine is known to increase catecholamine-mediated shift of potassium into cells via its stimulatory effects on the sodium, potassium-adenosine triphosphatase (Na, K-ATPase). A change in the activity of Na, K-ATPase has been demonstrated in thyrotoxic patients and it is reported that untreated thyrotoxic subjects with THPP have a higher Na, K-ATPase activity than thyrotoxic subjects without THPP [3, 19]. The Na, K-ATPase pump is also activated by insulin and androgen which may explain why the attacks occur after high carbohydrate meals and the male predilection for the disease [20, 21]. Whatever the underlying mechanism the fundamental defect in THPP is increase in intracellular potassium with concomitant reduction of extracellular potassium. This influx of potassium into cells causes a failure in depolarisation of the muscle cell membrane.

The standard therapy for THPP attacks is potassium replacement. Potassium replacement treats paralysis and also decreases the risk of cardiac arrhythmia. However, it should be emphasised that hypokalemia in THPP results from an unbalanced shift of potassium rather than total body depletion of potassium [22]. Therefore if potassium replacement is initiated, it is important to use judicious doses and monitor the potassium levels frequently during the treatment. In a retrospective study, potassium supplementation resulted in
rebound hyperkalemia in more than 40% of the cases [17]. Additionally propranolol has proven to be a valuable adjunct to remission of the acute attack and the prevention of the recurrences [4].

The ultimate objective of therapy for THPP is to establish the euthyroid state because paralysis attacks appear to be thyrotoxicosis dependent. There is high recurrence rate of hyperthyroidism after long-term medical therapy, therefore early radioiodine ablation of thyroid is recommended, as was employed in our patient [15, 18]. However, transient increase in serum thyroid hormone levels within the first seven to 10 days, which is a well-known complication of radioactive iodine therapy, can precipitate paralysis attacks and propranolol alone may not prevent them as in our patient. Patients should be warned about this complication of radioiodine administration and followed up regarding the recurrence of the THPP attacks after ablative therapy. Treatment with anti-thyroid drug for longer periods to empty the thyroid hormone reserves before radioiodine administration would have been better to minimize the risk of recurrence of THPP in the period of radiation thyroiditis.

In summary, we present a case of THPP in an ethnic group which is not previously described in English literature. This case shows that radioiodine can lead to increased thyroid hormone levels after a few days of administration and precipitate the paralysis attacks.

Acknowledgement

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References