Thyrotoxic periodic paralysis (TPP) is a very rare, life-threatening complication of thyrotoxicosis that is characterized by episodes of severe hypokalemia and muscle paralysis. The electrolyte and muscle disorders are associated with Grave’s thyrotoxicosis and are fully reversible through medical treatments. In rare instances, medical treatment is not successful in controlling the disease, and otolaryngologists are called upon to assist in management. TPP is a rare indication for total thyroidectomy, and otolaryngologists should be aware of the condition and its proper management.

INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is a relatively unknown condition in the United States. The condition is common among young adult Asian males and is characterized by severe hypokalemia and flaccid muscle paralysis in the setting of thyrotoxicosis. Standard management of the disease is with medical therapy to correct the electrolyte and metabolic abnormalities. However, surgical therapy with total thyroidectomy is indicated for uncontrolled disease, and our report is the first successful report in the otolaryngology literature of performing a total thyroidectomy to treat TPP.

CASE REPORT, CONTINUED

Given the failure of medical therapy, the patient’s endocrinologist recommended surgical therapy with total thyroidectomy. He was referred to our otolaryngology clinic for further management.

We performed a total thyroidectomy. The operation was uneventful and the patient had stable ionized calcium levels. He was discharged on post-operative day one after an uncomplicated hospital course. He was started on thyroid hormone replacement therapy. After 6 months of follow-up, the patient’s serum electrolyte and thyroid hormone tests were within normal limits, and he did not have any recurrent episodes of TPP. There were no surgical or treatment-related complications.

DISCUSSION, CONTINUED

The primary goal of treatment of a TPP episode is correction of metabolic abnormalities to prevent major cardiopulmonary complications. Initial treatment is aimed at cautiously normalizing serum potassium levels through intravenous administration of potassium chloride. Dilligence is necessary to prevent overcorrection and subsequent hyperkalemia caused by an intravascular potassium shift. Once the acute electrolyte abnormalities have been corrected, medical therapy of thyrotoxicosis and adrenergic state is instituted with methimazole and propanolol. Occasionally, medical therapy is inadequate in treating the Grave’s disease, and patients require surgical therapy. Our case is the first report in the otolaryngology literature of a patient with uncontrolled TPP requiring a total thyroidectomy.

In our case, surgical therapy was effective and in managing the disease and preventing future episodes of periodic paralysis.


table 1

<table>
<thead>
<tr>
<th>Normal values</th>
<th>1st episode</th>
<th>2nd episode</th>
<th>3rd episode</th>
<th>6 months post-op</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum K+ (mEq/L)</td>
<td>3.0-5.0</td>
<td>2.1</td>
<td>1.3</td>
<td>1.8</td>
</tr>
<tr>
<td>TSH (mIU/L)</td>
<td>0.3-3.0</td>
<td>0.07</td>
<td>0.01</td>
<td>0.06</td>
</tr>
<tr>
<td>Free T4 (μg/L)</td>
<td>0.7-2.0</td>
<td>1.88</td>
<td>2.30</td>
<td>2.03</td>
</tr>
<tr>
<td>Total T4 (μg/L)</td>
<td>4.5-12.5</td>
<td>11.2</td>
<td>11.99</td>
<td>9.3</td>
</tr>
<tr>
<td>Total T3 (μg/L)</td>
<td>90-220</td>
<td>229.1</td>
<td>175.4</td>
<td>316.0</td>
</tr>
</tbody>
</table>

Key: K+- Potassium; TSH- thyroid stimulating hormone; T4- thyroxine; T3- tri-iodothyronine

TPP is a rare metabolic complication of thyrotoxicosis that results in episodes of severe hypokalemia and muscle paralysis. The condition is most common among Asian males, with an overall incidence of less than 2% in Asians and an estimated male to female ratio of 20:1.1,2 However, this acquired form of acute paralysis is relatively unknown in the United States, with reported incidences of only 0.1-0.2% of all thyrotoxic patients.1 A PubMed search and literature review yielded zero references to thyrotoxic periodic paralysis in the otolaryngology literature.

The clinical presentation is typically a young adult Asian male between 20-40 years of age who has recurrent episodes of muscle weakness or complete paralysis. Attacks are most common overnight and after heavy meals or strenuous exercise.3 The symptoms typically begin in the proximal legs and progress to complete flaccid paralysis of all extremities and skeletal muscles. Respiratory and smooth muscles are not affected, although respiratory arrest has been reported.1 Most patients do not have a known history of hyperthyroidism until they present with a TPP attack.3 A diagnosis of TPP can be made when a patient has biochemical evidence of hyperthyroidism with a suppressed TSH and elevated thyroid hormone levels in the setting of muscle weakness and hypokalemia.1 Electromyogram evidence of muscle weakness and electrocardiogram changes secondary to hypokalemia are common but unnecessary in making the diagnosis of TPP.

The pathogenesis of TPP is related to increased activity of the Na/K-ATPase pump that results in a massive shift of potassium into the intracellular compartment. Thyroid hormone and β-adrenergic responses increase the activity of the Na/K pump, resulting in hypokalemia and a TPP attack.1 TPP is a sporadic disorder and is unrelated to familial hypokalemic periodic paralysis chanelopathy. The HLA and Kir2.6 genes have been implicated, however the genes responsible for making a patient susceptible to TPP have not yet been elucidated.1,3

REFERENCES