Common neurological complications of untreated Graves’ disease include cognitive dysfunction, tremor, ophthalmopathy, myopathy and polyneuropathy. Myasthenia gravis and seizures are uncommon associations, while thyrotoxic periodic paralysis, stroke and chorea occur only rarely.\(^1\)

We present a patient with Graves’ disease and the acute motor axonal neuropathy (AMAN) variant of Guillain-Barré syndrome, which masqueraded as so-called Basedow paraplegia.\(^1-5\) Had the diagnosis of Basedow paraplegia been adhered to, the patient would have been denied the opportunity of receiving gammaglobulin therapy.

**Case report**

An 18-year-old female was admitted in November 2014 with acute onset of severe global leg and arm weakness that had started 4 days before her admission. There was a background history (over the previous 11 months) of proptosis, dyspnoea on exertion, palpitations, irritability and forgetfulness. General examination revealed tachycardia and a symmetrical diffusely enlarged goitre. Proptosis and lid lag were also present. Neurological examination revealed motor weakness, with her legs more affected than her arms. There was global hypotonia and deep tendon arreflexia, with sparing of all sensory modalities. Blood tests confirmed the clinical suspicion of hyperthyroidism. Serum potassium levels were repeatedly normal (Table 1), urine porphobilinogen screening was negative and antiganglioside antibodies were absent.

TSH = thyroid-stimulating hormone.

<table>
<thead>
<tr>
<th>Table 1. Laboratory results</th>
<th>On admission</th>
<th>1 week after admission</th>
<th>2 weeks after admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>TSH (mIU/L) (0.48 - 4.26)</td>
<td>0.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free thyroxine (pmol/L) (7.6 - 16.1)</td>
<td>55</td>
<td>30.4</td>
<td>27.5</td>
</tr>
<tr>
<td>Potassium (mmol/L) (3.5 - 5.1)</td>
<td>4.9</td>
<td></td>
<td>4.0</td>
</tr>
<tr>
<td>TSH receptor antibody (U/L) (&lt;1.75)</td>
<td>36.26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebrospinal fluid protein (g/L) (0.15 - 0.45)</td>
<td>0.86</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

R = right; CMAP = compound muscle action potential; SNAP = sensory nerve action potential; ABP = abductor pollicis brevis; ADM = adductor digiti minimi; EDB = extensor digitorum brevis; AH = adductor hallucis. The R median, ulnar, peroneal and tibial nerve latencies were 2.8 ms, 2.4 ms, 3.0 ms and 3.5 ms, respectively (within normal limits), and their conduction velocities 54.4 m/s, 70.3 m/s, 43.4 m/s and 55.3 m/s, respectively, were also normal. The sensory peak latencies of the R median (palmar), R ulnar (palmar) and R sural (point B) nerves were 1.9 ms, 2.0 ms and 3.3 ms, respectively (within normal limits), and their conduction velocities 55.3 m/s, 51.3 m/s and 51.5 m/s, respectively (within normal limits).

![QR Code](QR.png)
The electroneurographic studies of 11 and 25 November 2014 showed significantly decreased compound muscle action potential (CMAP) amplitudes and preserved sensory nerve action potential (SNAP) amplitudes, with normal distal latencies and conduction velocities, favouring a diagnosis of an AMAN variant of the Guillain-Barré syndrome (Tables 2 and 3). Needle examination of the tibialis anterior muscle on 25 November 2014 showed the presence of fibrillation potentials and clear neurogenic polyphasic motor units. A 5-day course of intravenous immunoglobulins, 24 g/day, was administered. Carbimazole was prescribed at 20 mg 8-hourly, and on the development of a skin reaction the dose was decreased to 10 mg 8-hourly. Propranolol was administered at a dose of 20 mg 6-hourly. The thyrotoxicosis gradually improved, but the patient's neurological condition had only marginally improved at the time of discharge.

Discussion


Descriptions of Basedow paraplegia [1-5] appear to conform to the development of an ‘acute flaccid paraplegia with absent reflexes’ against the background of hyperthyroidism. It is, however, possible that this clinical presentation may reflect the occurrence of an acute idiopathic polyneuropathy, [3] possibly associated with an underlying predisposition to autoimmune diseases. [3]

The association between hyperthyroidism and acute flaccid areflexic neuropathy receives little credence in the following well-known clinical textbooks: Dyck and Thomas’ Peripheral Neuropathy [5] comments on its uncertain association and the difficulty to distinguish it from acute idiopathic polyneuropathy; Bradley’s Neurology in Clinical Practice [8] refers to its association as fortuitous; and Williams’ textbook of Endocrinology [9] and Harrison’s Internal Medicine [10] do not even mention the association.

Conclusion

It is important to consider the occurrence of other treatable causes of motor paralysis in patients with Graves’ disease, such as acute idiopathic polyneuropathy presenting with a rapid onset of flaccid paralysis. The entity of Basedow paraplegia as a diagnosis, per se, was found to be misleading.

Acknowledgements.

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References