Conversion of autoimmune hypothyroidism to hyperthyroidism with thyroid eye disease

M Moriarty, E Mills, H L Yap, A Hamda

Department of Diabetes and Endocrinology, Watford General Hospital, West Hertfordshire Hospitals NHS Trust, UK

**Background**

41 year old lady

Current smoker

**Medical history:**

Hypothyroidism x 15 years

+ve TPO antibodies

On levothyroxine

Psychotic depression

Borderline personality disorder

**Presentation**

Four year history with progressive eye signs

TFTs - suppressed TSH

On examination active thyroid eye disease

- Restriction of extra-ocular movement
  - Proptosis
  - Diplopia
- Reduced visual acuity

**Eye Disease**

- Advised to stop smoking
- Commenced 12 week course of pulsed intravenous methylprednisolone
- Good initial response however disease after 6 months
  - Persistent diplopia
  - Significant proptosis
- Underwent orbital decompression with excellent functional response

**Thyroid function tests**

- Gradual reduction in levothyroxine dose
- Persistent biochemical evidence of hyperthyroidism
- +ve TSH receptor antibodies and Thyroid uptake scan showed increased uptake throughout the gland
- No clinical features of thyrotoxicosis
- Commenced on carbimazole with dose adjustment over 8 months
- Developed hypothyroidism on treatment which continued on withdrawal of carbimazole
- Recommended on thyroid replacement therapy

**Thyroid Function Test Results During Treatment**

<table>
<thead>
<tr>
<th></th>
<th>April 13</th>
<th>Aug 13</th>
<th>Sept 13</th>
<th>April 14</th>
<th>July 14</th>
<th>Sept 14</th>
<th>Nov 14</th>
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</thead>
<tbody>
<tr>
<td>TSH</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>6.87</td>
<td>3.65</td>
<td>5.94</td>
<td>0.46</td>
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<tr>
<td>Free T4</td>
<td>28.7</td>
<td>22.3</td>
<td>24.3</td>
<td>7.8</td>
<td>10.1</td>
<td>8.3</td>
<td>12.5</td>
</tr>
<tr>
<td>Free T3</td>
<td>7.4</td>
<td>7.7</td>
<td>10.6</td>
<td>2.3</td>
<td>Off treatment</td>
<td>4.2</td>
<td></td>
</tr>
<tr>
<td>Levothyroxine dose reduced</td>
<td>Dose further reduced</td>
<td>Carbimazole started</td>
<td>Carbimazole stopped</td>
<td>Off treatment</td>
<td>Recommenced levothyroxine</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Challenges to treatment**

- Given her significant psychiatric history she was reviewed regularly by the local mental health team. Her mental state deteriorated during the course of steroids but responded well to psychiatric intervention.
- She had poor engagement with the endocrine team leading to a delay in optimising her thyroid function. A multidisciplinary approach centred on the opthamology clinic was ultimately successful.

**Discussion**

- Though most commonly considered as Graves’ ophthalmopathy, thyroid eye disease has been described in all forms of autoimmune thyroid disease.
- We postulate that this lady had TSH receptor antibodies which were variably stimulating or blocking the TSH receptor as the likely underlying cause for her presentation. Though rare this illustrates the need to have a high index of suspicion for possible conversion of hypothyroidism to hyperthyroidism in such patients.
- Attributing the biochemical abnormality solely to over replacement with thyroid hormone misses an opportunity to commence appropriate therapy.

**References**

