Fainting matters: a case of an autoimmune polyglandular syndrome (APS) with an atypical involvement of the parathyroid gland

Ieva Ruža1,2,3, Sabine Upmale3, Justīne Māliņa3, Inta Leitāne2

1Department of Internal Medicine, Riga Eastern Clinical University Hospital, Latvia
2Tomakalna Outpatient Clinic, Riga, Latvia
3Riga Stradiners University, Latvia

**Introduction:** The autoimmune polyglandular syndrome (APS) is characterized by the coexistence of at least two glandular autoimmune mediated diseases.

We report a combination of an autoimmune thyroid disease, Addison’s disease and an atypical involvement of the parathyroid gland.

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aldosterone (40 – 310 pg/ml)</td>
<td>3.7</td>
</tr>
<tr>
<td>Renin (4.4-46.1 mU/L)</td>
<td>2.1</td>
</tr>
<tr>
<td>ACTH (7.2 – 63.3 pg/ml)</td>
<td>44.5</td>
</tr>
<tr>
<td>Cortisol 8:00 (3.7-19.4 mg/dl)</td>
<td>3.7</td>
</tr>
<tr>
<td>TSH (0.35 – 4.94 mU/L)</td>
<td>12</td>
</tr>
<tr>
<td>FT4 (0.71 – 1.25 ng/ml)</td>
<td>0.95</td>
</tr>
<tr>
<td>Calcium (2.1 – 2.55 mmol/l)</td>
<td>2.03</td>
</tr>
<tr>
<td>PTH (15 – 68 pg/ml)</td>
<td>62.4</td>
</tr>
<tr>
<td>Creatinine (62 – 106 mmol/L)</td>
<td>95</td>
</tr>
</tbody>
</table>

**Insulin stress test – insufficient rise**

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma cortisol at 8:00 (3.7-19.4 mg/dl)</td>
<td>3.7</td>
</tr>
<tr>
<td>30 min after infusion</td>
<td>4.1</td>
</tr>
<tr>
<td>60 min after infusion</td>
<td>4.4</td>
</tr>
<tr>
<td>15 min after hypoglycemia</td>
<td>10.0</td>
</tr>
<tr>
<td>60 min after hypoglycemia</td>
<td>12.9</td>
</tr>
</tbody>
</table>

**First visit:**

A 73-years old white woman was admitted to the clinic for evaluation due to recurrent syncopes during the previous 3 years that presented along with:
- vertigo
- cold sweats
- general fatigue
and was associated with prolonged standing.

- Loss of 15 kg weight within the past year
- No skin changes observed

**3 months ago she was diagnosed with:**
- Chronic autoimmune thyroiditis and primary hypothyroidism
- Parathyroid gland adenoma (SPECT-CT)
- Orthostatic hypotension

**Investigations:**
- Head CT scan – basal ganglia calcinosis
- Head MRI, EEG, brachiocephalic vessel duplex ultrasonography, Holter monitoring revealed no substantial changes
- DXA scan – lumbar T-score -3.1 SD
- Antibody screen (ANA, thyroid peroxidase, transglutaminase) was negative.
- Low cortisol in 24h urine
- No data on diabetes mellitus or malignancy

**SPECT-CT – left upper parathyroid gland adenoma**

**Diagnosis:**
- Primary adrenal insufficiency with glucocorticoid and mineralocorticoid deficiency
- Chronic autoimmune thyroiditis with primary hypothyroidism
- Hypocalcaemia
- Calcinosis of basal ganglia (possible Fahr’s syndrome)
- Parathyroid gland adenoma
- Secondary osteoporosis due to malabsorption
- Chronic kidney disease

Based on these findings, type 2 autoimmune polyglandular syndrome was diagnosed.

**Conclusions:**
- PTH level was normal, but the coexistence of hypocalcaemia and parathyroid gland adenoma must be taken into consideration
- We would like to stress the importance of investigation for autoimmune glandular diseases and electrolyte level in case of unexplained syncope.

**Treatment:**
- L-Thyroxine
- Oral calcium
- Alphacalcidol
- Hydrocortisone
- Fludrocortisone

A substantial improvement of symptoms was seen in a control visit after 3 months.

Contact e-mail: dr.ieva.ruza@gmail.com