Unusual site of Carotid Aneurysm

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Case history

Mrs LG a 60-year-old woman was referred to the medical outpatient department by her general practitioner with a 2-month history of increased tiredness, lethargy and weight loss having lost approximately 2 stones in the preceding 5 months. She had been diagnosed with hypothyroidism a few years previously and had been on thyroxine intermittently. She was not on thyroxine when seen in clinic. Blood tests carried out by her general practitioner indicated a free T4 6.7 pmol/l (12–22), thyroid stimulating hormone (TSH) 6.3 mU/l (0.5–4.5) and prolactin 1965 u/l (<650), sodium 139 mmol/l (135–145), potassium 5.1 mmol/l (3.5–5.1) and eGFR 57 ml/min. In the clinic she also complained of generalized myalgia, arthralgia and constipation. There was no history of cold intolerance or hair loss. She also did not give a history of headaches, visual disturbance or galactorrhoea. She was post menopausal with her periods having stopped 10 years earlier and had incidentally been diagnosed with primary biliary cirrhosis many years previously which had been stable and her medications included ursodeoxycholic acid and atenolol. She had a family history of thyrotoxicosis and her sister had died of a ruptured cerebral artery aneurysm 10 years earlier.

On examination she had a pulse of 64 bpm and BP 120/70 mm of Hg. There was reduced visual acuity in her left eye, which had been longstanding with no abnormality on visual field testing on confrontation. Cardiovascular, respiratory and abdominal systems were normal. Her repeat blood tests from the clinic were as show in Tables 1–3.

She underwent a repeat short synacthen test which indicated basal cortisol 83 nmol/l and a 30 min post-adreno cortico tropic hormone (ACTH) level 313 nmol/l. Hydrocortisone 10 mg twice a day was started and an magnetic resonance imaging (MRI) of pituitary gland and formal visual field testing were requested.

Formal visual field testing were within normal limits and the MRI was reported to show a large well defined lesion in the sella extending to the suprasellar compartment but not extending to the optic chiasm with the presence of a flow artefact. A subsequent MR angiogram demonstrated a very substantial aneurysm arising from the internal carotid artery at the level of the sella containing thrombus.

She was referred to neurosurgery and underwent further CT angiogram to delineate the margins of the aneurysm. She was then assessed by interventional radiology and had endovascular coil embolization without complications. At her last clinic appointment she appeared well and had put on weight. As her FT4 levels were still low she was commenced on thyroxine. She is due to be followed-up by the neurosurgical department for repeat MRA 6 months post-procedure.

Discussion

Prevalence of intracranial aneurysms varies widely ranging from 0.4% to 3.6% in autopsy studies and...
up to 6% in studies of patients undergoing cerebral angiography. While most occur in the anterior circulation, those projecting into the sellar region account for ~1% to ~2% of all intracranial aneurysms. It is even rarer for intrasellar aneurysms to present with hypopituitarism, with only a few cases being described in the literature. The mechanisms for these aneurysms developing have not been fully explained but flow-related mechanical stress, atherosclerosis and post-radiation have all been proposed.

Patients with intracranial tumours can present with a host of symptoms depending on the site and size of the aneurysm including in one case report of a patient diagnosed with ‘migraine’. They can present with non-specific symptoms such as fatigue, weight loss or headaches, or with nasal or even subarachnoid bleeds and cavernous sinus syndrome. When these tumours extend into the sellar region, visual field defects and symptoms of pituitary hormonal deficiency may well be the first discernable symptoms. This variation of presenting symptoms may also be influenced by the size and rate of progression of aneurysmal dilatation. This risk being increased once the size exceeds 2.5cm.

Endocrine dysfunction when present with intrasellar aneurysms is usually associated with hyperprolactinaemia. The gonadal axis, adrenal axis and the thyroid axis can be affected in that order. Our patient had a high prolactin level with both adrenal and thyroid axis being affected. It would be difficult to comment on the gonadal axis as she was post-menopausal. When present these endocrine abnormalities are usually irreversible and will require permanent hormone replacement. Pituitary hormone deficiency is most probably due to compression of the hypothalamus or pituitary stalk accounting for the hyperprolactinaemia.

Pituitary apoplexy may also be a rare presentation of an intrasellar aneurysm. It would manifest as severe headache, visual loss, cranial nerve palsies (especially third, fourth and sixth cranial nerves) and depressed conscious level. It results most commonly from haemorrhage into a pituitary tumour but may also occur with aneurysms in the presence or absence of tumours.

In patients presenting with intracranial symptoms a computed tomography (CT) is usually requested. A CT scan of an intrasellar aneurysm would demonstrate a homogenous pituitary mass possibly with rim-like calcification but it would be difficult to differentiate from a pituitary tumour. As an intrasellar aneurysm can mimic a pituitary adenoma, distinguishing the two diagnoses reliably with imaging techniques is paramount. Hence in the presence of endocrine abnormalities an MRI is preferred as it produces greater anatomical detail and allows multiplanar imaging. Aneurysms are better identified on MRI as areas of black (signal void) on both T1 and T2 weighted images. In addition, in the presence of a thrombus an area of white (high intensity signal) will be seen. Aneurysms of the internal carotid will be seen to be contiguous with the parent vessel supplying it on MRI, to have flow effects within and also clear defined margins. There may also be a dark rim seen on T2 weighted images likely to represent haemosiderin deposition within the aneurysm. However, even an MRI may not be able to distinguish an aneurysm from an abscess. Hence angiography still remains the gold standard for the diagnosis of carotid aneurysms and to determine potential treatment options such as endovascular repair or surgery.

In our patient who presented with non-specific symptoms, the inadequately raised TSH in the presence of low free thyroxine levels first alerted us to a pituitary origin for her endocrine abnormalities. Hyperprolactinaemia can commonly be found in

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**Table 1 Metoclopramide Test**

<table>
<thead>
<tr>
<th>Metoclopramide test</th>
<th>0 minutes</th>
<th>30 minutes</th>
<th>60 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prolactin mU/l</td>
<td>2168</td>
<td>2678</td>
<td>2476</td>
</tr>
</tbody>
</table>

**Table 2 Short Synacthen Test**

<table>
<thead>
<tr>
<th>Short Synacthen test</th>
<th>0 minutes</th>
<th>30 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortisol level nmol/l</td>
<td>164</td>
<td>582</td>
</tr>
</tbody>
</table>

**Table 3 Hormonal Profile**

<table>
<thead>
<tr>
<th>Blood test</th>
<th>Results</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>T3 pmol/l</td>
<td>3.3</td>
<td>3.1–6.8</td>
</tr>
<tr>
<td>Free T4 pmol/l</td>
<td>5.8</td>
<td>12–22</td>
</tr>
<tr>
<td>Anti TPO</td>
<td>173</td>
<td>&lt;34</td>
</tr>
<tr>
<td>TSH mU/l</td>
<td>6.2</td>
<td>0.5–4.5</td>
</tr>
<tr>
<td>Prolactin mU/l</td>
<td>2167</td>
<td>102–496</td>
</tr>
<tr>
<td>IGF 1</td>
<td>&lt;3</td>
<td>9.8–27.6</td>
</tr>
<tr>
<td>LH U/l</td>
<td>0.7</td>
<td>Depending on stage of cycle</td>
</tr>
<tr>
<td>FSH U/l</td>
<td>4.2</td>
<td>Post-menopausal level &gt;26</td>
</tr>
</tbody>
</table>

FSH follicle stimulating hormone; IGF1 insulin growth factor 1; LH, luteinizing hormone.
primary hypothyroidism. It was also difficult to access her old thyroid function tests and we cannot be sure if her diagnosis of primary hypothyroidism a few years previously was appropriate. In view of her symptoms it was important that her steroid axis was investigated urgently. As her problems were long standing a short synacthen test was deemed adequate and the test conducted the same day indicated a sub normal response to ACTH. She was started on steroids and later on thyroxine as the thyroid axis was also found to be abnormal. An MRI confirmed the sellar lesion which was confirmed by angiography. The patient subsequently underwent endovascular coil embolization and is doing well.

**Funding**

None.

**Conflict of interest:** None declared.

**References**


