Clinical Reports

Pituitary function with a solitary intrasellar plasmacytoma

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Summary: A solitary intrasellar plasmacytoma with marked pituitary fossa destruction and yet near normal pituitary gland function is described. We suggest that a minimal disturbance of endocrine function together with a radiologically abnormal pituitary fossa indicates that the primary lesion may lie outside the pituitary fossa.

Introduction

Extramedullary plasmacytomas may affect a variety of tissues but most commonly occur in the upper respiratory tract, particularly the maxillary sinuses, rhinopharynx, nasal and tonsillar fossae (Ennuyer et al., 1963). They account, however, for less than 1% of malignant tumours of the nasopharynx (Fletcher & Million, 1965).

A solitary plasmacytoma may be difficult to diagnose if it arises in the sella turcica since clinically it can mimic a non-functioning pituitary adenoma. Before January 1982 only 12 cases of intracranial solitary extramedullary plasmacytomas had been described in the English literature and in none had the diagnosis been considered before tumour removal (Atweh & Jabbour, 1982).

There are very few reports of computerized axial tomography (CT) scan findings in intracranial plasmacytoma, although it is well known that in the parasellar region tumours other than those arising out of the pituitary fossa can occur showing similar CT scan enhancement patterns to those of pituitary tumours. Radiology is therefore not particularly helpful in distinguishing between a pituitary adenoma and a parasellar tumour except that at an early stage a lateral skull X-ray may show the parasellar tumour arising from within the surrounding bone with a normal pituitary fossa. A case of gross pituitary fossa destruction with minimal disturbance of endocrine function is described where surgery revealed the cause to be a parasellar, not an intrasellar lesion.

Case report

A 66 year old male presented with a 3 week history of diplopia on left lateral gaze and frontal headache. Examination revealed a left sixth cranial nerve palsy. Skull X-ray showed marked destruction of the pituitary fossa, while computerized tomography confirmed pituitary fossa destruction and following contrast medium, a high density lesion was shown high in the fossa extending laterally into the cavernous sinus on either side. There was no evidence of suprasellar extension. Biochemical investigations revealed normal basal values; thyroxine (T4) 106 nmol/l (normal 55–150 nmol/l), prolactin 93 mU/l (normal

Table I Results of pre-operative endocrine assessment

<table>
<thead>
<tr>
<th>Insulin (0.1 U/kg)</th>
<th>Time (min)</th>
<th>0</th>
<th>+30</th>
<th>+60</th>
<th>+90</th>
<th>+120</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose (mmol/l)</td>
<td>4.2</td>
<td>1.2</td>
<td>2.1</td>
<td>2.9</td>
<td>2.9</td>
<td></td>
</tr>
<tr>
<td>Growth hormone (mU/l)</td>
<td>1.7</td>
<td>1.5</td>
<td>44.5</td>
<td>10.2</td>
<td>10.2</td>
<td></td>
</tr>
<tr>
<td>Cortisol (nmol/l)</td>
<td>133</td>
<td>84</td>
<td>323</td>
<td>276</td>
<td>276</td>
<td></td>
</tr>
<tr>
<td>Luteinizing hormone (U/l)</td>
<td>93</td>
<td>479</td>
<td>451</td>
<td>274</td>
<td>274</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>LHRH (100 µg)</th>
<th>Time (min)</th>
<th>0</th>
<th>+20</th>
<th>+60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Luteinizing hormone (U/l)</td>
<td>7.4</td>
<td>24.3</td>
<td>23.2</td>
<td></td>
</tr>
<tr>
<td>Follicle stimulating hormone (U/l)</td>
<td>3.3</td>
<td>6.3</td>
<td>7.0</td>
<td></td>
</tr>
<tr>
<td>TRH (200 µg)</td>
<td>Time (min)</td>
<td>0</td>
<td>+20</td>
<td>+60</td>
</tr>
<tr>
<td>TSH (mU/l)</td>
<td>&lt;0.5</td>
<td>2.6</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td>Metyrapone (250 mg/2 h)</td>
<td>Basal</td>
<td>Day 1</td>
<td>Day 2</td>
<td>Day 3</td>
</tr>
<tr>
<td>Urinary 170HCS (mg/24 h)</td>
<td>9.4</td>
<td>7.7</td>
<td>32.0</td>
<td>19.4</td>
</tr>
</tbody>
</table>

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<420 mU/l) and testosterone 16 nmol/l (normal range 10–30 nmol/l). Dynamic endocrine function tests including the response to adequate hypoglycaemia, luteinizing hormone releasing hormone (LHRH), thyrotropin releasing hormone (TRH) and metyrapone are shown in Table I. A slightly impaired cortisol response to insulin induced hypoglycaemia (normal rise > 500 nmol/l), a low/normal thyroid stimulating hormone (TSH) response to TRH (normal increment of > 2.5 mU/l), normal gonadotrophin response to LHRH and a normal rise of urinary 17 hydroxy-corticosteroids (> 15 mg 17OHCS/24 h) are shown.

In July 1982 a sellotomy was performed and a large friable tumour was found infiltrating the base of the skull and posterior nasal cavity and completely occupying the sphenoidal air sinus. Histologically the tumour was composed of plasma cells of varying degrees of maturity, the appearance being compatible with a solitary extramedullary plasmacytoma or a deposit of multiple myeloma. Subsequent laboratory investigation disclosed an ESR of 15 mm/h, haemoglobin of 13 g/dl, normal urea, sodium, potassium, calcium and alkaline phosphatase levels. Bone marrow examination, plasma and urine electrophoresis and serum immunoglobulins were within the normal range. Bone scan and skeletal survey were normal. In view of the findings the diagnosis of solitary extramedullary plasmacytoma was made and post-operatively the patient received 4 500 rads over 25 fractions. The patient remains well, pituitary function 18 months after surgery is normal apart from a minimally impaired cortisol response to insulin hypoglycaemia.

Discussion

Where there is marked radiological pituitary fossa destruction due to an intrasellar pituitary tumour it is unusual to find well preserved pituitary function on dynamic testing. Minimally disturbed pituitary function together with grossly abnormal radiological findings would therefore suggest that the primary lesion may well lie outside the pituitary fossa. This is in accord with a previously reported case of an intrasellar plasmacytoma where there was no clinical evidence of pituitary function disturbance (Urbanski et al., 1980).

References

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