Presentation and management of parasellar and suprasellar metastatic mass lesions

JORGE C KATTAH, ROBERT M SILGALS, HERBERT MANZ, JAIME G TORO, ANATOLY DRITSCHILO, FREDERICK P SMITH

From the Departments of Neurology, Medicine, Vincent T Lombardi Cancer Research Center, Radiation Medicine, and Pathology, Georgetown University School of Medicine, Washington, DC, USA

SUMMARY Ten patients with parasellar metastatic lesions presented with insidious painful or painless ophthalmoplegia. Visual loss secondary to a chiasmal syndrome was identified in three of them. A mass lesion was demonstrated by CT scan with precise outlining of the location and size of the lesion. Symptomatic relief was achieved with local radiotherapy in those patients who had early diagnosis.

The clinical manifestations of parasellar and suprasellar lesions are so characteristic that their constellation of symptoms and signs has a localising value. Although the great majority of mass lesions in these areas represent expanding pituitary tumours, other intracranial neoplasms and a variety of non-neoplastic conditions can also be found. Metastatic tumours involving the parasellar and/or suprasellar regions are relatively infrequent. We report our experience with such lesions accumulated over the past four years. The anatomical and oncological aspects of these lesions have been discussed previously. The main purpose of this paper is to provide greater information about the CT findings and to emphasise the favorable effects of radiotherapy observed in our patients.

Materials and methods

In a period of four years ten patients presented between 1979 and 1982 with neuroophthalmologic complaints (table 1) including painful or painless diplopia and/or insidious visual loss. The mean age was 59 years. Five were men and five were women. Eight of these patients were known to have systemic malignancy and had evidence of disseminated metastatic disease at the time that parasellar or suprasellar lesions were identified (table 2). In two patients the intracranial lesion was the first manifestation of the neoplasm and the diagnosis was made after the biopsy of the mass.

In the majority of the patients clinical examination revealed oculomotor signs (table 1). The simultaneous ipsilateral paralysis of IIIrd and VIth nerves suggested cavernous sinus involvement in three cases; likewise, painful ophthalmoplegia in two additional cases was also consistent with a cavernous sinus lesion. A chiasmal syndrome and ophthalmoplegia were seen in one patient as an early manifestation of the tumour. Nonspecific painless diplopia occurred in three patients. Finally, one patient with diabetes insipidus presented with a painful Horner’s syndrome.

Visual fields were tested in eight patients, as part of their initial examination. In seven no abnormalities were found; the other patient had bitemporal superior field depression. At a later date, two patients developed bitemporal visual field defects identified by confrontation. Formal visual field testing was not possible due to their systemic debilitation.

Radiological studies included computed tomography (CT scan) obtained with a Phillips TomoScan 310 in nine patients. All CT scans were obtained after the infusion of 300 ml of meglumine diatrizoate (Hypaque 60%) administered over a period of five minutes. The scan sequence included four to six 1-5 mm thick transverse and coronal tissue sections with visualisation of the sella turcica and the suprasellar cistern. The CT scan showed a uniformly enhancing mass lesion in eight patients (table 1). The area of enhancement was round or triangular in shape and occupied the entire cavernous sinus in the majority of cases (fig 1). In one patient the lesion involved both the cavernous sinus and the suprasellar cistern. The size of the lesion was best demonstrated by coronal cuts in all patients.

The CSF was examined by lumbar puncture in eight patients in whom the diagnosis was initially uncertain; the results are summarised in table 3. A twofold increase in the level of protein content of the cerebrospinal fluid (CSF) was common, the glucose levels were normal, and the cytology was negative except in one patient with associated carcinomatous meningitis. A cisternal tap was performed in one patient in an effort to establish a diagnosis. The cisternal fluid protein content was increased over the protein level measured in the lumbar spinal fluid. Other para-
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Table 1 Neuroophthalmological and CT scan results

<table>
<thead>
<tr>
<th>Patient</th>
<th>Symptoms</th>
<th>Examination</th>
<th>Initial CT scan</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Painful ophthalmoplegia</td>
<td>Left lateral rectus palsy</td>
<td>Parasellar mass</td>
</tr>
<tr>
<td>II</td>
<td>Painful ophthalmoplegia</td>
<td>Right lateral rectus palsy and hemifacial spasm</td>
<td>Parasellar mass</td>
</tr>
<tr>
<td>III</td>
<td>Painless ophthalmoplegia</td>
<td>Left incomplete IIIrd and VIth nerve palsies</td>
<td>Parasellar mass</td>
</tr>
<tr>
<td>IV</td>
<td>Painless ophthalmoplegia and binocular visual loss</td>
<td>Bitemporal superior quadrantanopia and left IIIrd nerve palsy</td>
<td>Large suprasellar and parasellar mass</td>
</tr>
<tr>
<td>V</td>
<td>Painless diplopia</td>
<td>Right lateral rectus palsy</td>
<td>Parasellar mass</td>
</tr>
<tr>
<td>VI</td>
<td>Painless diplopia</td>
<td>Right lateral rectus palsy</td>
<td>Parasellar mass</td>
</tr>
<tr>
<td>VII</td>
<td>Painless diplopia</td>
<td>Right IIIrd and VIth nerve palsies</td>
<td>Parasellar mass</td>
</tr>
<tr>
<td>VIII</td>
<td>Painless diplopia</td>
<td>Horner’s syndrome</td>
<td>Not done</td>
</tr>
<tr>
<td>IX</td>
<td>Di and ptosis of left eye</td>
<td>Bilateral VIth nerve and right Vth nerve palsy</td>
<td>Normal</td>
</tr>
<tr>
<td>X</td>
<td>Painless diplopia</td>
<td>Left IIIrd and VIth nerve paresis</td>
<td>Parasellar mass</td>
</tr>
</tbody>
</table>

metors were normal.

Surgery, was performed in two patients without known primary malignancies. The diagnosis was established by the biopsy results. In seven patients with known primary extracranial malignancies, the diagnosis was made on the basis of the clinical and neuroimaging findings. In the remaining two patients with terminal cancer, the diagnosis was confirmed following necropsy examination.

Eight patients received radiotherapy to the brain or parasellar region for palliation of symptoms (table 2). Five were treated with whole brain radiation by opposed lateral fields. Total doses ranged from 2900–4000 rad in 200–300 rad fractions administered five times per week. Two patients received additional 1000 rad boosts to the sellar, suprasellar, and parasellar regions. Two patients were treated with more limited radiation fields. One had previously received radiotherapy for nasopharyngeal carcinoma. When disease recurred in the parasellar region, radiation therapy was administered by 180° arc to the cavernous sinus to a total dose of 3960 rad, followed by a 1000 rad boost. An additional patient with a recurrence of a salivary gland tumour received 4400 rads to the base of the brain by opposed lateral fields. Radiation therapy records were not available for review in one patient.

Among eight patients treated, seven demonstrated significant clinical improvement (table 2). The pain resolved almost immediately and the ophthalmoplegia subsided within two to three weeks after the initiation of treatment. CT scans after radiation were obtained in three of the improved patients, in one of whom the CT showed disappearance of the tumour mass and in the other two the size of the lesion, the local mass effect, and degree of enhancement were decreased. Although nine of the patients in this series died in an average of nine months after the initial ocular signs, it is our opinion that the quality of their lives was substantially improved with treatment.

Discussion

While intrasellar pituitary metastases are not uncommon findings in necropsy or hypophysectomy specimens in patients with systemic malignancy,2,13 symptomatic metastatic lesions involving parasellar or suprasellar structures are infrequent.2,8 Metastatic lesions in this area probably start as a mass placed lateral to the sella turcica; however, rapidly invasive tumours in any location near the sella in left untreated can involve the suprasellar and parasellar spaces in addition to the pituitary gland and possibly the hypothalamus, as illustrated in three of our patients (fig 2A, B).

Ophthalmoplegia was the cardinal manifestation of intracranial involvement in our patients and was associated with signs and symptoms strongly suggestive of cavernous sinus involvement.1 Among the different causes of ophthalmoplegia to be consi-

Table 2 General clinical data in 10 patients with suprasellar and parasellar metastatic lesions

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Primary tumour</th>
<th>Interval* between diagnosis and metastases</th>
<th>Other systemic metastases</th>
<th>Additional CNS lesions</th>
<th>Results of treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>51</td>
<td>F</td>
<td>Lymphoma</td>
<td>28 mo.</td>
<td>+</td>
<td>---</td>
<td>Improved</td>
</tr>
<tr>
<td>II</td>
<td>61</td>
<td>M</td>
<td>Nasopharyngeal cancer</td>
<td>51 mo.</td>
<td>-</td>
<td>Hemifacial spasm</td>
<td>Unknown</td>
</tr>
<tr>
<td>III</td>
<td>74</td>
<td>M</td>
<td>Cancer of salivary gland</td>
<td>12 yr.</td>
<td>+</td>
<td>---</td>
<td>Improved</td>
</tr>
<tr>
<td>IV</td>
<td>69</td>
<td>F</td>
<td>Lung</td>
<td>Initial presentation</td>
<td>+</td>
<td>---</td>
<td>Improved</td>
</tr>
<tr>
<td>V</td>
<td>38</td>
<td>F</td>
<td>Breast</td>
<td>4 yr.</td>
<td>+</td>
<td>Lumbar epidural metastases</td>
<td>Resolved</td>
</tr>
<tr>
<td>VI</td>
<td>70</td>
<td>F</td>
<td>Nasopharyngeal</td>
<td>Initial presentation</td>
<td>+</td>
<td>---</td>
<td>Resolved</td>
</tr>
<tr>
<td>VII</td>
<td>50</td>
<td>M</td>
<td>Thyroid cancer</td>
<td>12 mo.</td>
<td>-</td>
<td>---</td>
<td>Improved</td>
</tr>
<tr>
<td>VIII</td>
<td>47</td>
<td>F</td>
<td>Breast</td>
<td>3 yr.</td>
<td>+</td>
<td>Not treated</td>
<td></td>
</tr>
<tr>
<td>IX</td>
<td>57</td>
<td>M</td>
<td>Colon</td>
<td>2 yr.</td>
<td>+</td>
<td>+ Not treated</td>
<td></td>
</tr>
<tr>
<td>X</td>
<td>71</td>
<td>F</td>
<td>Breast</td>
<td>18 mo.</td>
<td>+</td>
<td>Carcinomatous meningitis</td>
<td></td>
</tr>
</tbody>
</table>

*Interval between initial diagnosis and signs for parasellar and/or suprasellar involvement.
While upper eyelid ptosis is frequent in patients with vincristine neuropathy, ophthalmoplegia is not as frequent. Among twenty subjects treated with vincristine by Albert, Wong, and Henderson, individual extraocular muscle palsies and ptosis developed in thirteen patients; five of whom had lateral rectus paresis; the ophthalmoplegia in their cases was dose-related and reversible after discounting the drug. Superior oblique paresis was reported in 10% of leukemic patients treated with vincristine.

In this series two patients had nasopharyngeal carcinomas; the importance of a thorough ENT examination in patients with progressive or painful ophthalmoplegia cannot be overemphasised.

Third nerve palsy with misdirection of fibres was not seen in any of our patients during the period of observation. Formal endocrine studies were not performed routinely in our cases. With the exception of one subject with diabetes insipidus, no overt clinical endocrine abnormalities were present. Frequently endocrinologically symptomatic metastatic lesions are secondary to simultaneous involvement of the pituitary gland and the hypothalamus. Diabetes insipidus is particularly frequent with metastatic lesions, suggesting a predilection for systemic cancer to invade the capillary network that supplies the hypothalamic nuclei, pituitary stalk, and neurohypophysis. One third of all patients with diabetes insipidus have metastatic and less commonly primary tumours in the suprasellar region.

Recent reports also emphasise a high incidence of panhypopituitarism which is often missed in patients with advanced carcinoma.

The contrast-enhanced CT scan demonstrated a uniformly enhancing mass lesion in 9 of the cases. We found direct coronal cuts to be quite helpful in outlining the actual size and shape of the lesion. In some cases sellar radiography may be necessary to demonstrate sellar destruction. The spinal fluid in eight of our patients showed nonspecific abnormal findings. The protein was increased, the glucose content was normal, and the cytology and cultures were negative in most cases. Only one of the subjects had leptomeningeal carcinomatosis. Based on these observations, in a given patient with cancer who

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**Table 3  Cerebrospinal fluid analysis**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Glucose</th>
<th>Protein</th>
<th>Pressure</th>
<th>Cytology</th>
<th>Cell Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>60 mg%</td>
<td>37 mg/dl</td>
<td>Normal</td>
<td>Negative</td>
<td>0 WBC</td>
</tr>
<tr>
<td>III</td>
<td>84 mg%</td>
<td>31 mg/dl</td>
<td>Normal</td>
<td>Negative</td>
<td>0 WBC</td>
</tr>
<tr>
<td>V</td>
<td>61 mg%</td>
<td>85 mg/dl</td>
<td>Normal</td>
<td>Negative</td>
<td>0 WBC</td>
</tr>
<tr>
<td>VI</td>
<td>83 mg%</td>
<td>41 mg/dl</td>
<td>Normal</td>
<td>Negative</td>
<td>0 WBC</td>
</tr>
<tr>
<td>VII</td>
<td>61 mg%</td>
<td>60 mg/dl</td>
<td>Normal</td>
<td>Negative</td>
<td>1 WBC</td>
</tr>
<tr>
<td>VIII</td>
<td>63 mg%</td>
<td>73 mg/dl</td>
<td>Normal</td>
<td>Negative</td>
<td>0 WBC</td>
</tr>
<tr>
<td>IX</td>
<td>84 mg%</td>
<td>77 mg/dl</td>
<td>Normal</td>
<td>Negative</td>
<td>0 WBC</td>
</tr>
<tr>
<td>X</td>
<td>169 mg%</td>
<td>200 mg/dl</td>
<td>Normal</td>
<td>Positive</td>
<td>Not done</td>
</tr>
</tbody>
</table>

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**Fig 1  Coronal views of contrast-enhanced CT scan of the brain. The arrows point to a large right-sided parasellar mass lesion in a patient with ophthalmoplegia and primary anaplastic carcinoma of the thyroid.**

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Fig 2. (a) View of cranial floor depicting metastases of colonic adenocarcinoma in and above sella turcica and the left anteromedial middle cranial fossa, involving Meckel's cave with the Gasserian ganglion (× 1). The larger tumour deposits are marked by arrows. (b) Coronal section at anterior hypothalamic level through columns of fornix, anterior commissure, and optic chiasm. A metastasis (in continuity with the suprasellar mass illustrated in fig 2(a) is seen in the posterior margin of the chiasm. (Luxol-fast blue hematoxylin-eosin, × 8.)

Table 4 Causes of ophthalmoplegia in patients with systemic cancer

| (1) | Leptomeningeal carcinomatosis |
| (2) | Increased intracranial pressure |
| (3) | Metastases to the base of the skull: (a) Orbital metastases (b) Parasellar metastases (c) Petrous apex metastases (d) Clivus metastases (e) Brain stem metastases |
| (4) | Opportunistic meningeal infections |
| (5) | Side effects of chemotherapy: (a) vincristine (b) 5 fluorouracil |
| (6) | Radiation-induced cranial neuropathy |
develops ophthalmoplegia and has a juxtasellar mass in the CT scan, additional tests do not seem necessary to establish a diagnosis unless other unexplained clinical signs are present.

Haematogenous spread of distant neoplastic tissue probably explains most of the metastatic lesions here considered. The bone surrounding the sella is probably the initial site with secondary involvement of the cavernous sinus, pituitary gland, and suprasellar cistern. In one of our cases with metastatic colon carcinoma to the base of the skull, necropsy revealed infiltration by the adenocarcinoma of cavernous sinus, pituitary gland, chiasm and hypothalamus (fig 2A, B). The majority of our patients had widespread metastatic disease at the time that ocular signs developed. In the patients with nasopharyngeal tumours a likely pathway of extension to the skull base is infiltration of the large cranial nerve foramina which have a direct access to the subarachnoid space and cavernous sinus. Nasopharyngeal tumours account for 20% of all cavernous sinus syndromes. Invasion of the pituitary gland parenchyma and the pituitary stalk by metastatic tumour is an additional frequent mechanism, particularly in patients with carcinoma of the breast. Leptomeningeal carcinomatosis associated with large tumour nests sitting in the perisellar region, as observed in one of our cases is another possible route. Finally, an unusual mechanism of perineural neoplastic infiltration from primary skin carcinomas can result in a cavernous sinus syndrome. Pituitary chromophobe adenomas are rarely an incidental finding in patients with cancer; however, it is important to differentiate metastatic perisellar tumours from invasive pituitary adenomas or adenocarcinomas which are generally associated with markedly increased serum prolactin levels.

Finally, we believe that rapid identification and treatment of metastatic disease in the perisellar region has a potential for measurable clinical improvement.

References

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