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SHORT REPORT

Solitary metastasis of breast carcinoma in the optic chiasm

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Abstract
Optic chiasmatic compression from suprasellar metastatic lesions is well known, but metastasis intrinsic to the optic chiasm has not been reported. A 45-year-old woman, with treated breast carcinoma, presented with headache and chiasmatic syndrome from a large suprasellar tumour. At surgery, an exophytic chiasmatic tumour was encountered, with an appearance similar to a glioma. The pathological appearance was consistent with the primary neoplasm in the breast.

Key words: *Breast carcinoma, metastasis, optic chiasm, suprasellar tumours.*

Introduction

Visual symptomatology related to intracranial metastases is uncommon, occurring usually in patients with advanced systemic cancer,[1,2] and rarely as the first presentation of the metastasis itself.[3,4] Extrinsic compression of the optic chiasm by metastatic tumours is known to occur rarely, with the pituitary gland, hypothalamus and the cavernous sinus regions most commonly involved.[1–8] Theoretically, metastases can occur anywhere in the craniospinal axis, but isolated metastasis intrinsic to the optic chiasm has not been described in the literature.

We report a patient with a chiasmatic syndrome caused by breast cancer metastatic to the optic chiasm.

Case report

A 45-year-old woman presented with retro-orbital headache and progressive deterioration of her vision for 2 months. She was known to have adenocarcinoma of the breast treated 2 years previously with surgical excision, local radiotherapy and chemotherapy. General physical examination was unremarkable; there was no evidence of metastatic disease. Neurological examination revealed a fully alert and cooperative patient, without evidence of neurological deficits outside the visual system. Ophthalmological examination revealed decreased visual acuity in both eyes (OS 20/80, OD 20/100), decreased colour vision and bitemporal hemianopsia. The optic fundi, ocular and pupillary motility, gross orbital anatomy and intraocular pressure were normal.

Computed tomography (CT) revealed a 2.5 × 2.7 × 3.0 cm well circumscribed hyperdense suprasellar mass, which enhanced homogeneously (Fig. 1). The sella turcica was normal, and there was no hyperostosis or dural enhancement. Magnetic resonance scans could not be obtained owing to extreme claustrophobia. Pituitary gland function was normal, except for a mildly elevated serum prolactin level.

At operation, a right subfrontal approach was undertaken to explore the lesion and decompress the chiasm. The tumour appeared to be a large exophytic mass arising from the chiasm and infiltrating the hypothalamic region, somewhat similar to an optic chiasm glioma (Fig. 2). The optic chiasm appeared pale and markedly enlarged, and both optic nerves and the right optic tract appeared normal. The pituitary stalk appeared normal and was displaced posteriorly by the tumour. A small incision was made on the superior surface of the chiasm and biopsy specimens were taken.

Histopathological examination demonstrated nests of neoplastic epithelial cells surrounded by a narrow collar of brain tissue necrosis and reactive astrocytosis (Fig. 3a). Immunohistochemical studies showed that the tumour cells were positive for cytokeratins with AE3 and AE1 monoclonal antibodies (Fig. 3b), and negative for vimentin, glial fibrillary acid protein and oestrogen receptors.

In the postoperative period there was no change in the patient's neurological status. She received 30 Gy of whole-brain irradiation over 2 weeks, resulting in

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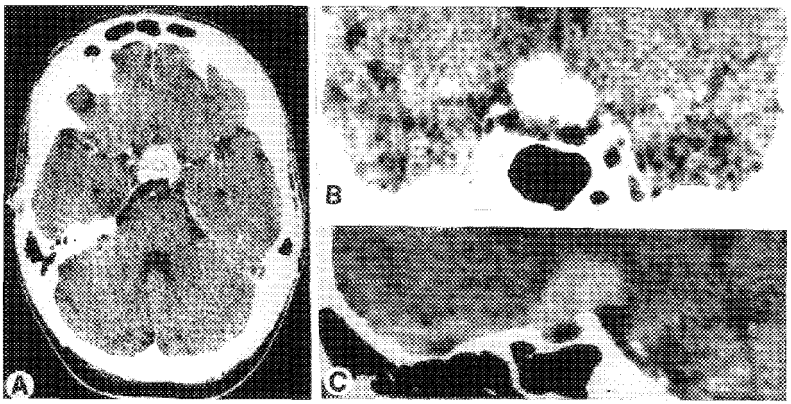

FIG. 1. Preoperative axial (A) contrast-enhanced CT scan with coronal (B) and sagittal (C) reconstruction demonstrating a large homogeneously enhancing suprasellar tumour.

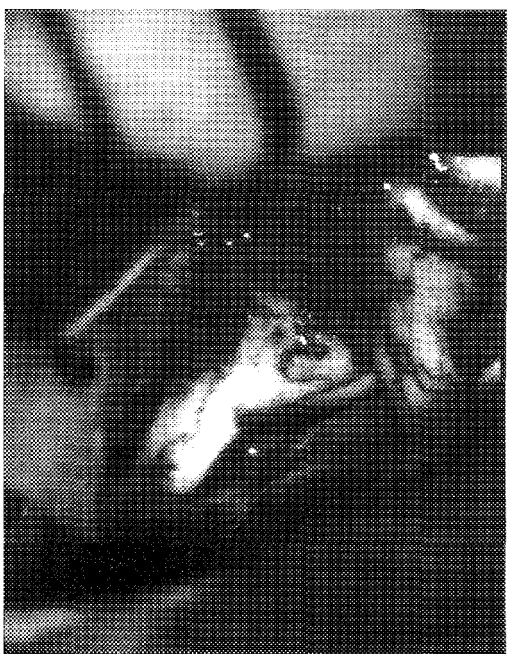

FIG. 2. Operative photographs through a right subfrontal approach showing a tumour intrinsic to the optic chiasm.

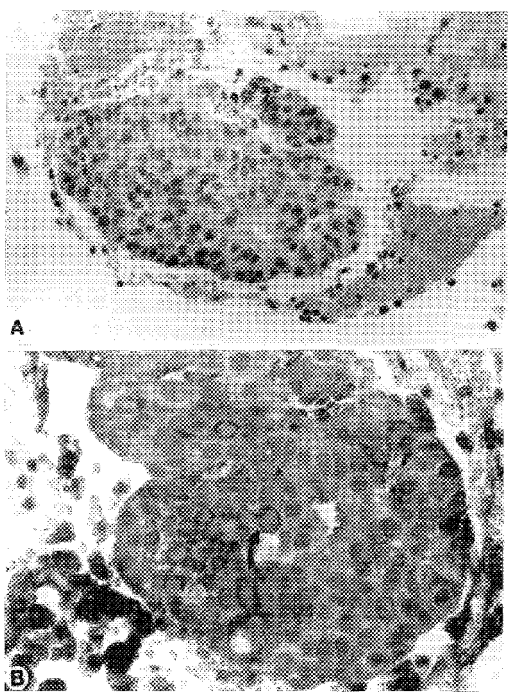

FIG. 3. Photomicrograph of the specimen taken from the optic chiasm tumour. (A) Nests of tumour cells invading the optic fibres (H&E). (Magnification × 100.) (B) Immunohistochemical staining for keratins with AE3 monoclonal antibodies reveals an immunoreactive tumour cell (arrow). (Magnification × 900).

mild subjective improvement of her vision. She tolerated only two cycles of systemic chemotherapy (methotrexate, cyclophosphamide, and 5-flourouracil), because of severe neutropenia. The patient's condition gradually deteriorated, with marked cognitive changes, diabetes insipidus and panhypopituitarism for which she received hormone replacement therapy.

Posttreatment follow-up CT demonstrated an increase in tumour size. The patient expired 6 months after the operation; her family declined a postmortem examination.

Discussion

Intracranial sites of metastatic tumours commonly include brain parenchyma, leptomeninges and dura; metastases in cranial nerves are unusual. In an autopsy review of 3359 cases, Takakura *et al.* found only 24 cases (0.7%) to have had metastasis in the cranial nerves; the sixth and the seventh cranial nerves were

the most frequently involved.[9] Whether the metastases were exclusive to the cranial nerves and the exact mechanism were not explained in the report.

We speculate two mechanisms for optic chiasm metastasis, haematogenous and direct spread. Classically, brain metastases, with few exceptions, are located near the gray-white matter junction presumably because of the acute change in the blood flow from the richly vascularized cortex to the less well supplied white matter.[10] The optic pathways, which are derived from evagination of the forebrain and considered to be a tract rather than a nerve, abuts a mass of gray matter (lateral geniculate body) at its posterior margin.[11] This concept makes haematogenous theory unlikely to be the mechanism of spread to the chiasm.

Alternatively, infiltration of the chiasm by tumour cells from near structures, mainly the pituitary gland is another mechanism. The clinical presentation of our case, the intraoperative findings and the absence of early associated endocrinopathy may favour the former mechanism. Most pituitary metastases are diagnosed at autopsy, but those causing symptoms usually present with pituitary dysfunction such as diabetes insipidus or hypopituitarism, with or without visual symptoms.[1 3,7]

Radiologically, the differential diagnosis of a suprasllar lesion is diverse; and in our case, pituitary adenoma, meningioma and metastasis are the most common tumours in this age group.[12] The long history of visual loss and the CT features of a hyperdense lesion that enhanced homogeneously were suggestive of tuberculum sellae meningioma. The presence of a normal sella made pituitary adenoma unlikely. The history of cancer, even when considered at an early stage or cured, is an important factor to consider in the differential diagnosis of intracranial tumours. MRI may delineate the lesion, narrow the differential diagnosis, but it may not establish the precise location of the tumour in relation to the chiasm and surgery remains the only diagnostic option.

The management of metastasis in such a location remains a challenge for the surgeon and the oncologist. Complete surgical excision and radiosurgery of metastatic tumours within the optic chiasm are associated with significant morbidity and some of these patients may benefit from adjuvant therapy.

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