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Case report

Extracranial metastatic meningioma

[1]B E FIGUEROA, [1]D J QUINT, [2]P E McKEEVER and [3]W F CHANDLER

Departments of [1]Radiology, [2]Pathology and [3]Neurosurgery, University of Michigan Medical Center, Ann Arbor, MI, USA

Abstract. Meningiomas rarely metastasize outside the intracranial compartment. We report a case of disseminated metastases from a recurrent intracranial meningioma and review the imaging and pathological literature on metastatic meningioma.

Meningioma is classically regarded as a benign neoplasm which commonly arises along the course of intracranial and spinal meninges, and dural extensions [1]. The majority of meningiomas become symptomatic, owing to local mass effect. The potential of these tumours to become more aggressive and invade the brain and/or calvaria, or to metastasize outside the central nervous system (CNS), is well known [2–4]. The present report illustrates a recurrent meningioma that invaded the skull base and eventually metastasized to the chest and abdomen. We also review the literature on intracranial meningiomas that metastasize outside the CNS.

Case report

A 50-year-old woman presented with a 1-year history of headache and facial paraesthesia in the distribution of the mandibular branch of the left trigeminal nerve. Imaging demonstrated a dural-based mass in the left middle cranial fossa, which extended through the skull base into the infratemporal fossa. Despite the extensive nature of this lesion, total resection of the tumour was achieved via a left temporal craniectomy. A diagnosis of transitional meningioma was established by pathological examination. Post-operatively, the patient was noted to have decreased sensation to pin-prick in the distribution of the second and third divisions of the left trigeminal nerve, in addition to a slight left facial droop. The patient remained clinically and radiographically stable for 5 years, at which time she developed left facial pain, decreased hearing in the left ear, and diplopia on left lateral gaze. CT and MRI (Figures 1a, b) demonstrated local tumour recurrence at the left skull base. New lung masses were also discovered at this time (Figure 1c). Pre-auricular needle biopsy of the recurrent skull base mass and lung mass biopsy confirmed local recurrence of the transitional meningioma, with pulmonary metastases of identical histological appearance. The patient received a radiation dose of 55 Gy to the cranium and declined any treatment of the thoracic metastatic disease. The skull base tumour and pulmonary metastases remained radiographically unchanged until 28 months later, when interval progression in the size of the multiple pulmonary masses was noted on chest radiography. 4 months later, MRI demonstrated that progression of the skull base mass had resulted in occlusion of the left external auditory canal, with associated cholesteatoma formation. Abdominal discomfort prompted additional imaging which demonstrated a hepatic mass which is presumed to represent metastatic disease (Figure 1d). The patient is currently receiving home hospice care.

Discussion

Originating from arachnoid cap cells, meningiomas account for 14–19% of all primary intracranial neoplasms [5]. Although they are typically slow-growing, extra-axial, benign tumours, meningiomas have a potential to metastasize with a reported rate of 0.1% [6–8]. Our case is one of only 76 cases of extracranial metastatic meningioma that have been reported, of which 88% were described before the advent of CT and MRI [2, 4, 6, 8, 13, 15–27].

A variety of factors have been associated with metastatic spread of meningiomas. Most commonly, the histopathological features of such meningiomas have been examined. However, the assessment of the extracranial metastatic potential of meningiomas is complicated by two factors. First, extracranial metastases in these patients

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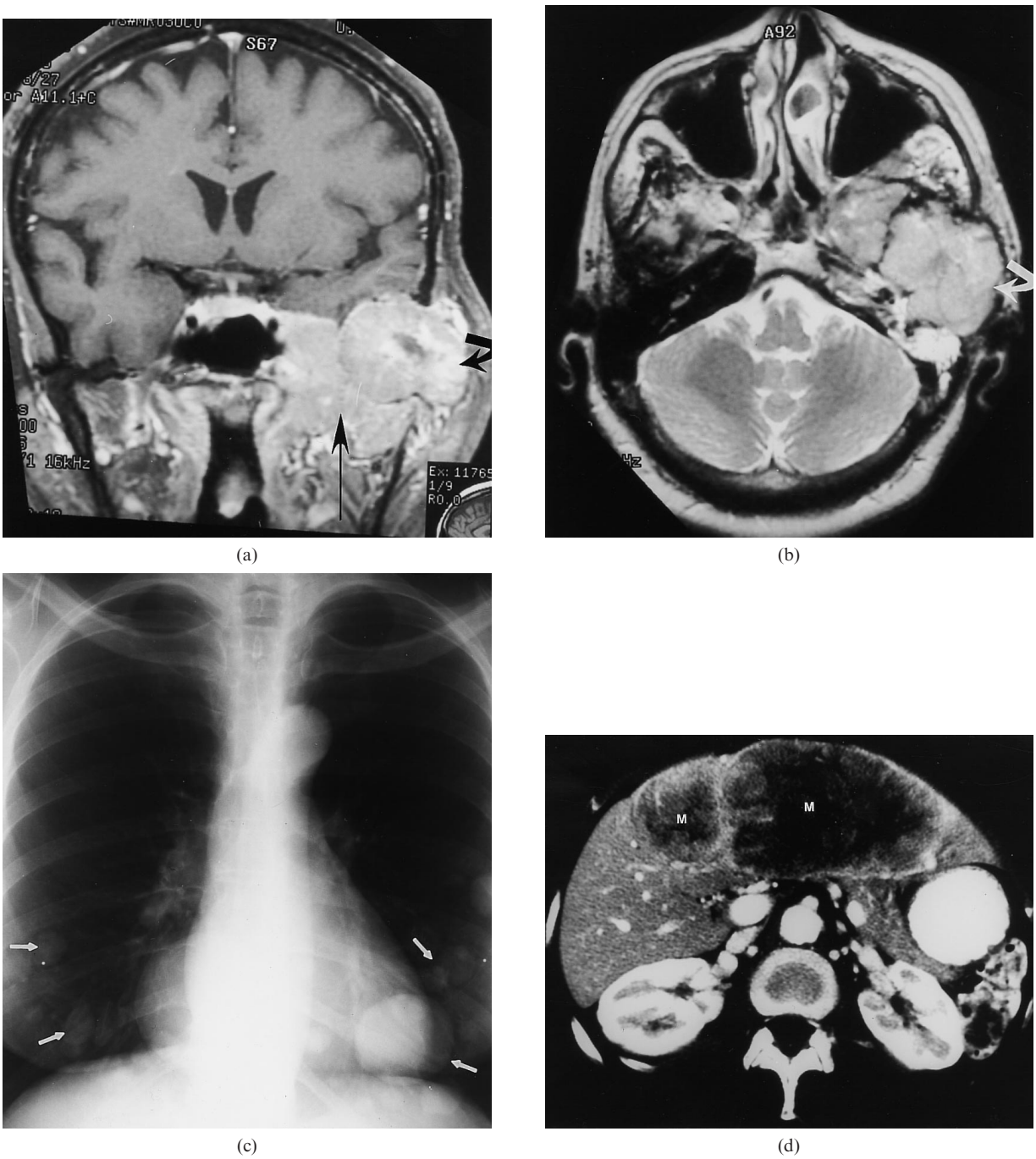
Address correspondence to Douglas J Quint, MD, B1D520, Neuroradiology, Box 30, Department of Radiology, University of Michigan Medical Center, 1500 East Medical Center Drive, Ann Arbor, MI 48109-0030, USA.



(a) (b)

(c) (d)

Figure 1. Recurrent skull base meningioma with pulmonary and hepatic metastases. Coronal contrast-enhanced T_1 weighted MR (a), and axial T_2 weighted MR (b) scans through the middle cranial fossa demonstrate homogeneously enhancing recurrent meningioma along the floor of the left middle cranial fossa, with inferior extension through the skull base (straight arrow) and lateral extension into the pre-auricular subcutaneous soft tissue (curved arrows). Chest radiograph (c) demonstrates multiple lung metastases (arrows). Abdominal CT (d) demonstrates spread of neoplasm to the liver [M].

[28–31] occur in only 0.1% of cases [32], and consequently, most descriptions are reports of individual cases. Second, the meningioma subtypes have been recently redefined. While there are several reports of extracranial metastases from angioblastic meningiomas [9, 30, 33, 34], angioblastic meningioma is not listed in the latest World Health Organization (WHO) classification of tumours. This raises the question as to how many of these reported meningiomas with extracranial metastases actually represent meningeal haemangiopericytomas, angiomatous meningiomas or other tumours [35]. It seems that at least some of these lesions were haemangiopericytomas [36–38]. Despite these uncertainties, the following tendencies emerge: (a) meningiomas of WHO grade higher than grade I (papillary, malignant and haemangiopericytic) have the greatest tendency to

metastasize [6, 8, 31, 32, 39–43]; (b) haemangiopericytomas of the meninges metastasize frequently [28, 36, 38]; (c) a high rate of cellular proliferation is not essential for extracranial metastases [35]; and (d) an individual meningioma of virtually any histological type, including WHO grade I histologically benign meningiomas, may metastasize [2, 29, 44–48].

A given meningioma frequently displays more than one histopathological variant. There is often a transition between a classic and papillary form, either at the primary site or in the metastasis [3, 6, 8].

More recently, new histological parameters for defining malignancy have been suggested, to identify meningiomas with higher growth potential and therefore presumed higher metastatic potential [4]. Such a grading system is based on assessment of hypercellularity, nuclear pleomorphism, mitotic figures, areas of necrosis, and other indices of malignancy, and may prove helpful in predicting the prognosis of a given meningioma from its histological features. Immunohistochemical analysis of a nuclear protein related to cell proliferation, Ki67, is also useful in evaluating the potential of meningiomas to recur and/or metastasize [10].

Neuroimaging signs suggestive of aggressive behaviour include marked peritumoral oedema from contiguous brain, heterogeneous contrast enhancement, minimal or no calcification, and indistinct or irregular margins [1]. The location of the intracranial tumour does not appear to be a factor in determining the probability of distal spread, as supratentorial, infratentorial, and spinal meningiomas have all been associated with metastases [2, 11]. Meningioma can spread by direct extension or metastasize within the CNS by cerebrospinal fluid pathways and thereby produce multiple primary meningiomas, a pattern of spread which has not been associated with extracranial spread of meningiomas [12]. Blood-borne passage of tumour cells through venous channels is the most likely mechanism for distal spread, as metastases are associated with prior surgery or the invasion of a venous sinus in 75% of cases [11, 13]. Xenotransplantation of human meningioma tumour cells into immune-compromised mice has produced brain tumours that frequently metastasize to the lung following a debulking procedure [14]. Clinically, meningioma metastases are seen in the lungs over 60% of the time. Other sites of metastases, in order of decreasing frequency, are: liver, long bones, vertebrae, ribs, pleura, mediastinum, and lymph nodes [2, 11].

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