Metastatic breast carcinoma mimicking basal skull meningioma

Gregory W Schmidt
Neil R Miller

From the Neuro-Ophthalmology Unit, The Wilmer Ophthalmological Institute, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Abstract: Two women with breast carcinoma developed clinical evidence of a sphenocavernous syndrome with magnetic resonance (MRI) that suggested a basal skull meningioma but in whom biopsy of the lesion revealed metastatic breast carcinoma. The difficulties in distinguishing between the two lesions on the basis of standard MRI are discussed, as are newer imaging techniques that may reduce or eliminate these problems.

Keywords: meningioma, metastatic carcinoma, MRI, sphenocavernous syndrome, skull base tumor

Although meningiomas have well-known clinical and imaging features, metastases from a variety of different neoplasms such as prostate (Fink 1979; Lippman et al 1986; Franco et al 1999), breast (Shapiro and Janzen 1959), lung (Khalfallah et al 1999), kidney (Killebrew et al 1983), gall bladder (Kawamata et al 1999), colon (Neroni et al 1999), and adenoid cystic carcinoma (Morioka et al 1993), as well as leiomyosarcoma (Buff et al 1991), can simulate them. In particular, although it often involves bone, metastatic breast adenocarcinoma can present as an isolated dural lesion without apparent involvement of adjacent bone (Johnson et al 2002). Indeed, Tsukada and colleagues (1983) found solitary metastases to the dura in breast cancer in approximately 8% of autopsied patients. We describe two patients with pathologically proven adenocarcinoma of the breast, one of whom developed a cavernous sinus syndrome and the other, an orbital apex syndrome, in whom standard magnetic resonance imaging (MRI) showed a lesion of the skull base consistent with a basal skull meningioma. In each case, the correct diagnosis of a soft-tissue, skull-base metastasis was not made until the lesion was biopsied.

Cases

Case 1

A 48-year-old woman developed adenocarcinoma of the breast treated with lumpectomy and radiation therapy. Three years later, she developed painless vertical diplopia and was found to have a right trochlear nerve paresis. MRI revealed an enhancing, right paraclinoid lesion and a smaller, similar lesion on the left (Figure 1). An evaluation revealed no evidence of metastases, and it was thought that the patient had a basal skull meningioma; however, she then developed increasing oblique diplopia and right ptosis and was found to have a right oculomotor nerve paresis. Repeat MRI showed an increase in the size and extent of the right-sided lesion with no change in the appearance of the lesion on the left. The patient underwent biopsy of the lesion that revealed findings consistent with metastatic carcinoma of the breast (Figure 2). She subsequently underwent whole-brain fractionated radiation therapy combined with stereotactic radiosurgery to

Correspondence: Neil R Miller
Maumenee 127, Wilmer Eye Institute, Johns Hopkins University School of Medicine, 600 North Wolfe Street, Baltimore, MD 21287, USA
Tel +1 443 407 4307
Email nrmiller@jhmi.edu

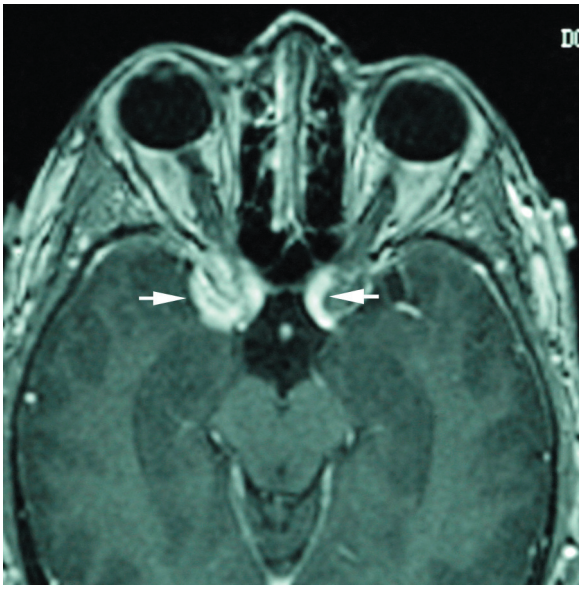

Figure 1 Case 1. Initial T1-weighted, axial MRI after intravenous injection of paramagnetic contrast material shows enhancing lesions surrounding the anterior clinoid processes (arrowheads). The right lesion is larger than the right and may be extending through the superior orbital fissure.

the anterior skull base in the region of the lesion, followed by chemotherapy. Over the next several months, the trochlear nerve paresis resolved, and the oculomotor nerve paresis improved but did not resolve. The patient died 22-months after diagnosis from the effects of metastatic disease.

Case 2

A 56-year-old woman was found to have adenocarcinoma of the breast at which time she was also found to have changes on computed tomographic scanning consistent with bony metastases to the vertebrae. She underwent a radical mastectomy and then began chemotherapy, but shortly thereafter, she developed headache and blurred vision in the inferior field of the right eye. Neuro-ophthalmologic examination showed evidence of an isolated, right retrobulbar optic neuropathy characterized by visual acuity of 20/40 in the eye associated with color vision of 3/10 using Hardy-Rand-Rittler pseudoisochromatic plates, an inferior field defect, and a relative afferent pupillary defect. Ophthalmoscopic examination of the ocular fundi revealed no abnormalities.

The patient underwent MRI that showed an enhancing soft-tissue lesion at the apex of the right orbit extending through the superior orbital fissure onto the medial sphenoid wing and around the temporal lobe tip (Figure 3). There was no involvement of adjacent bone. The differential diagnosis was metastatic carcinoma versus sphenoid wing meningioma. The patient refused biopsy of the lesion but agreed to undergo radiation therapy. The lesion was subsequently treated with conventional fractionated radiation totaling 30 Gy.

Shortly after completing radiation therapy, the patient experienced improvement in visual sensory function, and this was taken as evidence that the lesion was a meningioma, particularly as it did not decrease in size or disappear on MRI as might be expected if it were a metastasis. Three months later, however, she developed recurrent, progressive loss of vision in the right eye and a right oculomotor nerve paresis. After repeat MRI showed an increase in the size of the mass, the patient underwent biopsy and subtotal resection of the mass, histopathologic examination of which was found to be consistent with metastatic adenocarcinoma (Figure 4). She was treated with further radiation therapy as well as chemotherapy but died 6-months later.

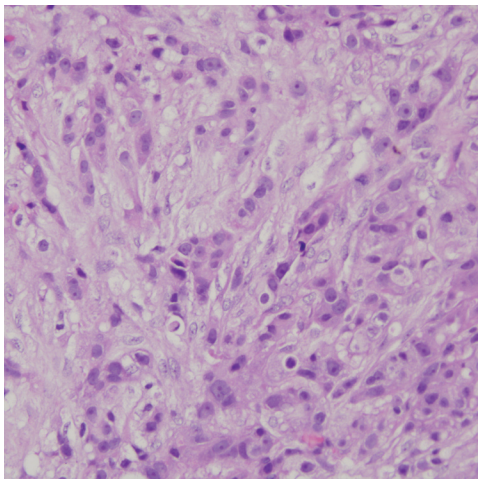

Figure 2 Case 1. Pathologic appearance of biopsy specimen. Note nests and cords of cancer cells (Hematoxylin and Eosin, X200).

Discussion

Many patients with orbital or central nervous system (CNS) metastases from adenocarcinoma of the breast either have or soon develop other manifestations of disseminated disease. The diagnosis of CNS metastasis usually is made by a variety of tests, including serum assay for carcinoembryonic antigen (CEA); bone scan; positron emission tomographic (PET) scanning; MRI of the brain, spine, or both; lumbar puncture; and computed tomographic (CT) scanning or MRI of the chest, abdomen, and pelvis. When evidence of disseminated disease is absent, as in our first patient, the likelihood of a solitary, enhancing, soft-tissue lesion of the skull base being a meningioma is much greater than the likelihood that it is an isolated metastasis; however, even when evidence of disseminated disease is present, as

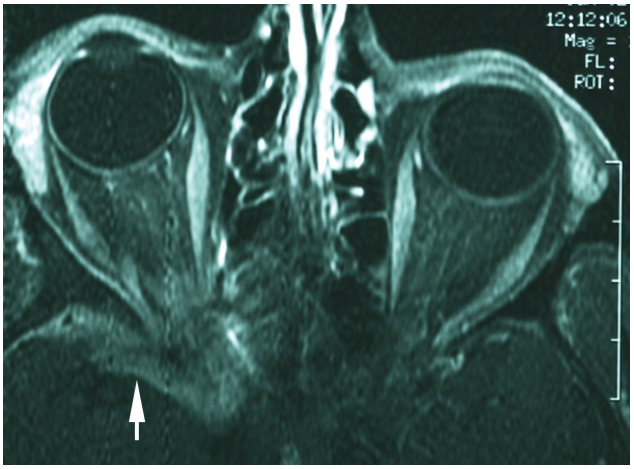

Figure 3 Case 2. Initial T1-weighted, axial MRI after intravenous injection of paramagnetic contrast material shows an enhancing soft-tissue lesion at the apex of the right orbit extending through the superior orbital fissure onto the medial sphenoid wing and around the temporal lobe tip (arrowhead).

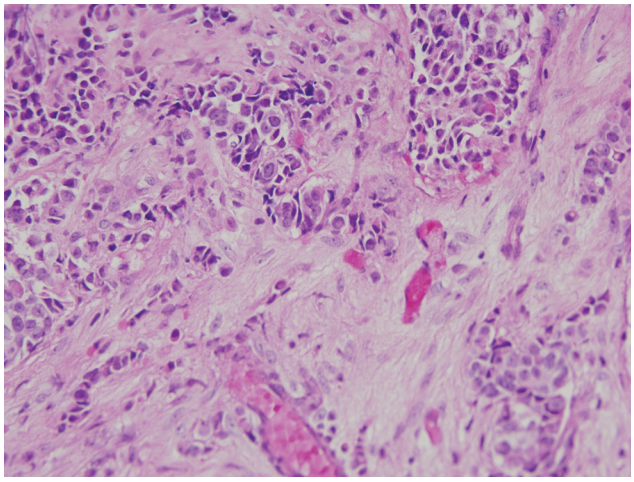

Figure 4 Case 2. Pathologic appearance of biopsy specimen. Note nests and cords of cancer cells (Hematoxylin and eosin, X200).

in our second patient, it may be impossible to differentiate a metastasis from a meningioma using conventional MRI (Johnson et al 2002).

Meningiomas have typical imaging features. They generally appear as an extra-axial mass with a broad attachment to the dura that is hyperdense on CT scanning and that enhances after intravenous injection of iodinated contrast material. On MRI, the lesion usually is isointense with gray matter on T1-weighted images, more variable on T2-weighted images, and homogeneously enhancing on T1-weighted images following intravenous administration of gadolinium-DTPA (Buetow et al 1991; Sheporaitis et al 1992) (Figure 5). Dural metastases generally present in one of two ways: as a subdural hematoma or as a tumor mass. It is the latter presentation that can be difficult to differentiate from a meningioma (Rumana et al 1998; Khalfallah et al 1999). Twenty-nine cases of dural metastases interpreted as meningioma have previously been reported (Tagle et al 2002). Typically, these tumors produce MR images characterized by an increased signal on T2-weighted images and an enhancing dural tail on T1-weighted images after intravenous injection of gadolinium (Quint and McGillicuddy 1994). The enhancement of the meninges adjacent to a meningioma on MRI, also seen with CT scanning, was once considered specific for meningioma (Goldsher et al 1990; Nakau et al 1997); however, it is clear that

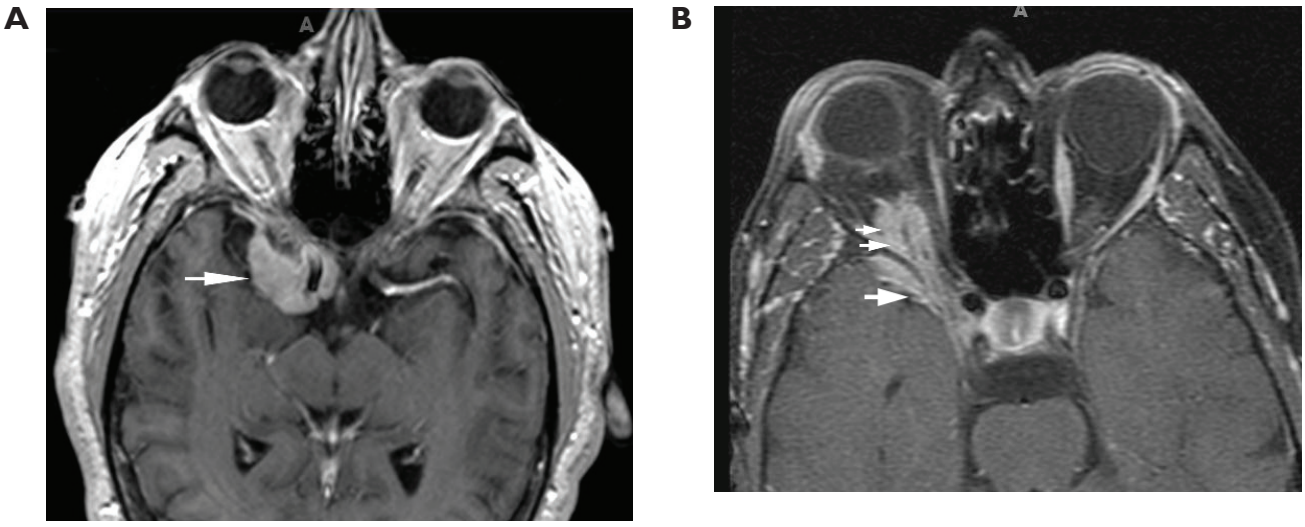

Figure 5 T1-weighted, axial MRIs after intravenous injection of paramagnetic contrast material in two cases of biopsy-proven sphenoid wing meningiomas. (**A**) Note similarity of enhancing lesion (arrowhead) that surrounds the right anterior clinoid process to the appearance of the lesion in Case 1. (**B**) Note similarity of enhancing lesion that involves the right orbit (small double arrowheads) as well as the medial sphenoid wing around the temporal lobe tip (large single arrowhead) to our Case 2.

this sign also can be present in patients with meningeal metastases (Wilms et al 1991; Quint and McGillicuddy 1994).

Although standard MRI may be unable to differentiate between intracranial meningiomas and metastases, it appears that dural metastases and meningiomas have different perfusion-sensitive characteristics when assessed using dynamic perfusion MRI (DPMRI) (Kremer et al 2004), a technique that provides information about a tumor's vascularity that is not obtained using conventional MRI. DPMRI has been used not only to differentiate dural metastases from meningiomas (Kremer et al 2004) but also to differentiate high-grade from low-grade gliomas (Provenzale et al 2002), lymphomas from high-grade gliomas (Kremer et al 2002; Hartmann et al 2003), and meningiomas from neuromas (Maeda et al 1994). The technique is based on the measurement of the MR signal intensity using a T2-weighted sequence during the first pass of a bolus of a paramagnetic contrast agent. This signal intensity is transiently reduced by susceptibility effects due to the first pass of paramagnetic agent through the vasculature (Barbier et al 2001). It provides a map of cerebral blood volume (CBV) and allows the calculation of the relative cerebral blood volume (rCBV), defined as the ratio between the CBV in the tumor and the CBV in the white matter. In contrast to the high rCBV typical of meningiomas, dural metastases show a low rCBV (Kremer et al 2004). Thus, this technique appears to be able to distinguish between these two lesions. Had this technique been available for our patients, we might have been able to spare them a craniotomy.

Conclusion

Although metastatic breast carcinoma was considered in the differential diagnosis of these two cases, it was not until exploratory surgery and histopathological study that the correct diagnosis could be established. It can be difficult to distinguish dural metastases from meningiomas using standard neuroimaging techniques, but new tools such as dynamic susceptibility contrast MR imaging may help make this a less difficult problem in the future.

Disclosure

Presented as a poster at the 2006 Meeting of the North American Neuro-Ophthalmology Society.

References

Barbier E, Lamalle L, Decorps M. 2001. Methodology of brain perfusion imaging. *J Magn Reson Imaging*, 13:496–520.

Buetow MP, Buetow PC, Smirniotopoulos JG. 1991. Typical, atypical, and misleading features in meningioma. *Radiographics*, 11:1087–106.

Buff BL Jr, Schick RM, Norregaard T. 1991. Meningeal metastasis of leiomyosarcoma mimicking meningioma: CT and MRI findings. *J Comput Assist Tomogr*, 15:166–7.

Fink LH. 1979. Metastasis of prostatic adenocarcinoma simulating a falx meningioma. *Surg Neurol*, 12:253–7.

Franco E, Gil-Neciga E, Cano G, et al. 1999. Sphenoid metastasis mimicking a meningioma as the initial feature of adenocarcinoma of the prostate. *Rev Neurol*, 29:929–32.

Goldsher D, Litt AW, Pinto RS, et al. 1990. Dural "tail" associated with meningiomas on Gd-DTPA-enhanced MR images: characteristics, differential diagnostic value, and possible implications for treatment. *Radiology*, 176:447–50.

Hartmann M, Heiland S, Harting I, et al. 2003. Distinguishing of primary cerebral lymphoma from high grade glioma with perfusion weighted magnetic resonance imaging. *Neuroscience Lett*, 338:119–22.

Johnson MD, Powell SZ, Boyer PJ, et al. 2002. Dural lesions mimicking meningiomas. *Human Pathol*, 33:1211–26.

Kawamata T, Kawamura H, Kubo O, et al. 1999. Central nervous system metastasis from gallbladder carcinoma mimicking a meningioma. Case illustration. *J Neurosurg*, 91:1059.

Khalfallah M, Roche PH, Figarrela-Branger D, et al. 1999. Isolated dural metastasis mimicking meningioma. A case report. *Neurochirurgie*, 45:250–4.

Killebrew K, Krigman M, Mahaley MS Jr, et al. 1983. Metastatic renal carcinoma mimicking a meningioma. *Neurosurgery*, 13:430–4.

Kremer S, Grand S, Remy C, et al. 2002. Cerebral blood volume mapping by MR imaging in the initial evaluation of brain tumors. *J Neuroradiol*, 29:105–13.

Kremer S, Grand S, Remy C, et al. 2004. Contribution of dynamic contrast MR imaging to the differentiation between dural metastasis and meningioma. *Neuroradiology*, 46:642–8.

Lippman SM, Buzaid AC, Iacono RP, et al. 1986. Cranial metastase from prostate cancer simulating meningioma: report of two cases and review of the literature. *Neurosurgery*, 19:820–3.

Maeda M, Itoh S, Kimura H, et al. 1994. Vascularity of meningiomas and neurinomas: assessment with dynamic susceptibility contrast MR imaging. *AJR Am J Roentgenol*, 163:181–6.

Morioka T, Matsushima T, Ikezaki K, et al. 1993. Intracranial adenoid cystic carcinoma mimicking meningioma: report of two cases. *Neuroradiology*, 35:462–5.

Nakau H, Miyazawa T, Tamai S, et al. 1997. Pathologic significance of meningeal enhancement ("flare sign") of meningiomas on MRI. *Surg Neurol*, 48:584–91.

Neroni M, Artico M, Pastore FS, et al. 1999. Diaphragma sellae metastasis from colon carcinoma mimicking a meningioma. A case report. *Neurochirurgie*, 45:160–3.

Provenzale JM, Wang GR, Brenner T, et al. 2002. Comparison of permeability in high-grade and low-grade brain tumors using dynamic susceptibility contrast MR imaging. *AJR Am J Roentgenol*, 178:711–6.

Quint D, McGillicuddy J. 1994. Meningeal metastasis of the cerebellopontine angle demonstrating "dural tail" sign. *Can Assoc Radiol J*, 45:40–3.

Rumana CS, Hess KR, Shi WM, et al. 1998. Metastatic brain tumors with dural extension. *J Neurosurg*, 89:552–8.

Shapiro R, Janzen AH. 1959. Osteoblastic metastases to floor of skull simulating meningioma en plaque. *Am J Roentgenol Radium Ther Nucl Med*, 81:964–6.

Sheporaitis L, Osborn A, Smirniotopoulos J, et al. 1992. Radiologic-pathologic correlation: intracranial meningioma. *AJNR Am J Neuroradiol*, 13:29–37.

Tagle P, Villanueva P, Torrealba G. 2002. Intracranial metastasis or meningioma? An uncommon clinical diagnostic dilemma. *Surg Neurol*, 58:241–5.

Tsukada Y, Fouad A, Pickren JW, et al. 1983. Central nervous system metastasis from breast carcinoma. Autopsy study. *Cancer*, 52:2349–54.

Wilms G, Lammens M, Marchal G, et al. 1991. Prominent dural enhancement adjacent to nonmeningiomatous malignant lesions on contrast-enhanced MR images. *AJNR Am J Neuroradiol*, 12:761–4.