Delayed Hepatic Metastasis From a Benign Fibroblastic Meningioma Thirty-One Years After Surgical Resection of the Intracranial Tumor

Introduction
Meningiomas are the most common benign intracranial neoplasms. Extracranial metastasis from benign meningiomas is exceedingly rare and is seen in less than 0.1% to 0.2% of cases.1 Lungs (60%) and liver (34%) are the most common sites of extracranial metastasis.2 We describe a patient with delayed hepatic metastasis of a benign fibroblastic meningioma that was detected 31 years after surgical resection of the intracranial meningioma.

Case Report
An 81-year-old female was diagnosed with meningioma in 1974 at the age of 46 years when she presented with headaches. She underwent total surgical resection at that time. She has a history of a chronic seizure disorder and is treated with anticonvulsant medications. In 1988, the patient was found to have a non-Hodgkin’s lymphoma involving the sacrum. She received chemotherapy with cyclophosphamide, doxorubicin, vincristine, and prednisone, along with radiation. Other medical comorbidities include hypertension and hypothyroidism. The patient is postmenopausal and lives a sedentary life with her spouse. She has never smoked and does not consume alcohol. She has no family history of malignancies.

In 2005, the patient was found to have multiple (12) low attenuation lesions that ranged in size from 1 to 2 cm throughout her liver parenchyma on a computed tomography scan (Figs 1A and 1B). A needle biopsy revealed spindle cells in a whorled pattern with a collagen-rich matrix; the tumor was low grade and had a low proliferation index that was consistent with fibroblastic metastatic meningioma (Figs 2A and 2B). There was no evidence of recurrence of the patient’s lymphoma. She was asymptomatic from her metastasis, and the decision was made to observe her expectantly with yearly imaging. Her liver lesions have remained stable throughout the last 5 years.

Discussion
Meningiomas are common intracranial tumors that account for 14% to 19% of all primary intracranial neoplasms.3 They arise from the meningothelial cells of the arachnoid membrane4 and are more common in women and in the sixth decade of life.4 Local invasion and distant metastasis of intracranial meningiomas have been described in the medical literature, but are rare.3

Meningiomas are usually benign, slow-growing tumors that histologically correspond to WHO grade 1 status. Although less common, atypical (WHO grade 2) and anaplastic (WHO grade 3) meningiomas are more aggressive, with a high risk of local recurrence and a less favorable prognosis.3 The recurrence rate for benign tumors is 3% at 5 years and 21% at 25 years.6

Extracranial metastasis occurs in less than 0.1% to 0.2% of intracranial meningiomas.1 According to WHO criteria, the histologic grade of the tumor is the most important predictive factor for recurrence and metastasis.7 Other factors include high cellularity, mitotic rate, nuclear pleomorphism, presence of foci of necrosis, and invasion of adjacent structures.3 However, these features are not essential for the occurrence of extracranial metastasis, and any histologically benign meningioma has the potential to metastasize.2 This is evident in our case of hepatic metastasis from a benign fibroblastic meningioma. Despite bland nuclear features, lack of mitotic activity, and a low proliferation index, our patient presented with delayed hepatic metastasis. The majority of reports of metastatic meningioma have involved local recurrence of tumors at least once prior to presentation with distant metastasis, as well as multiple craniotomies to treat the recurrent tumor.2-5 However, metastatic meningioma may present in isolation without coexistent or previous local tumor recurrence, as seen in our patient.
The most common sites of metastasis of meningioma are the lungs (60%), abdomen and liver (34%), cervical lymph nodes (18%), long bones, pelvis and skull (11%), pleura (9%), vertebrae (7%), CNS (7%), and mediastinum (5%). Meningiomas may disseminate through hematogenous, lymphatic, or CSF routes. Metastasis through the CNS via CSF leads to disseminated meningiomas. The passage of tumor cells into venous channels allows spread through the right blood circulation into the lungs, liver, and other organs. Isolated hepatic or renal metastasis may also occur through the vertebral (meningorachidian) venous system, which connects the veins of the skull, spinal canal, and vertebral column to the thoracoabdominal wall.

Treatment modalities for intracranial meningiomas include surgery, radiosurgery, and radiation therapy. Surgery seems to be the treatment of choice for resectable lesions. The results after a subtotal resection and postoperative radiotherapy are similar to those seen after a complete surgical excision. Radiation therapy, radiosurgery, and newer options that include stereotactic radiotherapy and intensity-modulated radiation therapy have been used with variable results that depend on the tumor location as well as staging. Long-term local control and survival seem to be similar with all of the treatment modalities.

No definite therapeutic regimen has been established for metastatic meningiomas. Liver metastasis can be surgically resected with a good prognosis for disease-free survival. Postoperatively, recurrence in the residual liver can be treated with transarterial chemoembolization. The role of chemotherapy is limited in meningioma; there is no or limited proven benefit from any systemic therapy, and no clear drug or combination regimen has given consistent responses. If asymptomatic, the hepatic metastasis can be watched expectantly and observed using imaging studies, as for our patient.

Thus, extracranial metastasis of meningiomas should always be included in the differential diagnosis of patients with a history of meningioma who develop lesions that are suggestive of distant metastasis. This holds true for benign meningiomas as well. Extracranial metastasis of meningiomas may be seen several decades after treatment of the intracranial meningioma and in the absence of local intracranial recurrence.

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AUTHORS’ DISCLOSURES OF POTENTIAL CONFLICTS OF INTEREST
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