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# Case Report

# Unusual growth pattern of a meningioma

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### Abstract

**Background:** Rhabdoid meningioma exhibits high mitotic activity, anaplasia, and increased markers of cell proliferation. Here we describe a rhabdoid meningioma with a pattern of growth extending into the subarachnoid space and filled the cortical sulci.

**Case Description:** A 72-year-old woman presented with headache and was admitted to our hospital. Neurologic and physical examinations revealed no abnormalities. Contrast-enhanced axial T1-weighted images showed a well-enhanced, dural-based mass compressing the right temporal and frontal lobes, and extending into the sylvian cistern and filling the cortical sulci. The patient underwent partial resection and the histologic findings demonstrated rhabdoid meningioma.

**Conclusion:** Although this type of tumor is known to be aggressive in its growth, extension into the adjacent cisternal space and the filling of the cortical sulci are rare. The combination of histologic anaplasia with the highest reported proliferation rate, loss of cohesion of neoplastic cells, and the location of the tumor led to the unique growth pattern.

Key Words: Magnetic resonance imaging, meningioma, rhabdoid



## INTRODUCTION

Meningiomas arise from meningothelial cells on the arachnoidal membrane, grow slowly, and then finally compress the adjacent brain. Most meningiomas grow inward toward the brain as discrete well-defined, dural-based masses and are spherical or lobulated. The borders between the tumor and the brain are usually round and clear because they preserve the histologic structures, such as the tumor capsule, cerebrospinal fluid, arachnoid mater, and pia mater.<sup>[5]</sup> However, this growth pattern cannot be universally applied.<sup>[7,10]</sup>

Here we describe a case of rhabdoid meningioma that

showed a unique growth pattern. The tumor extended into the subdural space and filled the cortical sulci.

## **CASE REPORT**

A 72-year-old woman presented with headache and was admitted to our hospital. Neurologic and physical examinations demonstrated no abnormality. Computed tomography scans showed an abnormal mass lesion in the right frontotemporal convexity region. The lesion was surrounded by marked cerebral edema. Mass effect was noted on the midbrain. Magnetic resonance (MR) imaging showed a mass with a similar signal intensity

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on the T1-weighted image and high signal intensity on the T2-weighted image [Figure 1], respectively. Contrast-enhanced axial T1-weighted images showed a well-enhanced, dural-based mass compressing the right temporal and frontal lobes, and extending into the sylvian cistern and filling the cortical sulci. It is specifically these subarachnoid components of the tumor in the sulci that showed relatively little mass effect. Coronal and sagittal enhanced T1-weighted images also showed unusual enhancement patterns [Figure 1]. Cerebral angiography showed a mild tumor blush supplied by the right middle meningeal artery and middle cerebral artery.

The patient underwent a right frontotemporal craniotomy, and the tumor was partially resected. The removed



Figure 1: Axial T2-weighted magnetic resonance image showing significant perilesional edema (a). Axial enhanced T1-weighted magnetic resonance images (b) showing a well-enhanced, duralbased mass compressing the right temporal and frontal lobes, extending into the sylvian cistern (arrows) and filling the cortical sulci (arrow heads). It is specifically these subarachnoid components of the tumor in the sulci that showed relatively little mass effect. Coronal and sagittal enhanced T1-weighted images also showed unusual enhancement patterns (c, d)

specimens consisted of the tumor tissues only, and did not include the surrounding brain tissues. Microscopically, dyscohesiveness of tumor cells was observed. The tumor was globally composed of rhabdoid cells showing large eosinophilic cytoplasm and eccentrically placed, mediumsized round nuclei with prominent nucleoli [Figure 2]. There were many tumor cells with an inclusion-like round structure in their cytoplasm. The patterns seen in conventional meningiomas, such as whorl or psammoma body formations, were not observed. Neither necrosis nor microvascular proliferation was present in the tumor, although scattered mitotic cells (3 mitoses per 10 highpower fields) were seen. Immunohistochemically, the cytoplasm was strongly positive for vimentin [Figure 3]. Epithelial membrans antigen was focally expressed. GFAP, S-100, and AE1/3 were negative. The MIB-1 staining index was approximately 8%. Postoperative course was uneventful and MR images demonstrated resection of the temporal mass lesion [Figure 4]. The patient was discharged home for outpatient radiotherapy treatment. MR imaging performed 24 months later did not revealed regrowth of the residual tumors.

#### DISCUSSION

Rhabdoid meningioma is a relatively rare tumor. It was classified as a WHO grade III tumor in 2000, along with papillary and anaplastic meningioma. It is a pathologic diagnosis with the presence of loosely cohesive cells with eccentric nuclei and hyaline, paranuclear inclusions being characteristic.<sup>[3]</sup> Electron microscopy revealed that characteristic interdigitating cellular processes and intercellular junctions are poorly developed or absent.<sup>[2]</sup> Typically, the tumor exhibits high mitotic activity, anaplasia, and increased markers of cell proliferation. According to the literature, most rhabdoid meningiomas



Figure 2: Tumor cells have eccentric nuclei with abundant eosinophilic "rhabdoid" cytoplasm. (H and E, ×40)



Figure 3: Cytoplasm shows strong immunoreactivity for vimentin. Many cells in this area have round intracytoplasmic inclusion-like structures. (Immunohistochemical stain for vimentin, original magnification ×40)



Figure 4: Axial enhanced TI-weighted magnetic resonance image showing a resection of the temporal mass lesion

grow rapidly and aggressively and have a very poor prognosis.<sup>[1,4,8,9,11,12]</sup> Radiologic presentations are highly variable, and there have been reports of rhabdoid meningioma presenting as a cystic lesion and as intraventricular tumor. There are no sensitive radiologic features to distinguish this variant from other types of meningioma.<sup>[6]</sup> Although this tumor is known for its aggressive growth, extension into the adjacent cisternal space and into the cortical sulci is rare. The combination of the histologic anaplasia with the highest reported proliferation rate, loss of cohesion of neoplastic cells, and the location of the tumor, most likely led to the unusual pattern of growth.

Many radiographic patterns of meningioma have been reported, but the present case is quite rare. Several patients have presented with tumors that showed a unique infiltrating morphology. The borders between the tumor and the brain were unclear but seemed to be fused together, which resulted in a brush-like appearance on MR images. Investigators have speculated that the tumor infiltrate into the Virchow–Robin spaces might serve as a conduit for the spread of the tumor process.<sup>[7,10]</sup> In the present case, MR imaging showed a very large enhanced mass lesion with an octopus-like shape with a clear-cut plane between the adjacent parenchyma.

we considered was that the tumor disrupted the adjacent arachnoid membrane, grew into the subarachnoid space, and extended along the cistern. In this way, the tumor growth filled the cortical sulci.

The management of malignant meningioma has included maximal surgical debulking followed by radiation therapy with or without systemic chemotherapy. We suggest that it would be difficult to completely resect such a tumor without causing additional neurologic disabilities. Thus, we resected the temporal mass lesion, and fractional radiation was targeted to the residual tumor. We continue to monitor the patient closely with surveillance imaging and clinical examination.

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