The patient refused surgery because deemed her symptoms to be not serious. Ten months following this initial discovery, she came to the hospital with left hemiparesis. MRI showed increased size of the mass (Fig. 3).

Cerebral angiography showed that the hypervascular mass was supplied by bilateral middle meningeal arteries. In addition, an incidental unruptured aneurysm at the anterior communicating artery was found (Fig. 4). The clipping of anterior communicating aneurysm was performed first through the left pterional approach. After a month, a right frontal rectangular craniotomy crossing midline was performed for tumor removal.

**INTRODUCTION**

Metastases of systemic cancer to intracranial tumors are rare, but they have been reported in literature since 1930. Breast and lung are the most common sources of tumor-to-tumor metastasis, while kidney, thyroid, prostate, and colon are less likely places of tumor origination. While processes responsible for metastases into meningioma have been proposed, the exact mechanism by which this occurs yet remains elusive. We present a case report of a metastasis of non-small cell lung cancer into an intracranial meningioma.

**Key Words:** Metastasis · Meningioma · Lung cancer · Non-small cell · Tumor-to-tumor · Harbor cancer.
The tumor presented as an encapsulated mass firmly attached to the dura, a typical characteristic of a meningioma. The central part of the tumor was firm, and the peripheral part was friable. The mass was completely removed. The post-surgical course was uneventful.

Microscopic examination revealed fibrous meningioma, and irregularly branching gland-like adenocarcinoma cell clusters intermixed with this fibrous meningioma in some areas (Fig. 5). The metastatic cancer cell clusters were enclosed by a rim of benign histologically distinct meningioma tissue. Histologic features of this adenocarcinoma were identical to those of the patient’s pulmonary adenocarcinoma resected 2 years ago. Immunohistochemical (IHC) staining for TTF-1 and CK7 were positive in this adenocarcinoma, and IHC staining for EMA and vimentin were positive in the background fibrous meningioma. These findings were compatible with a metastasis of pulmonary adenocarcinoma into the meningioma, so-called “tumor-to-tumor metastasis”. Post-operative chemotherapy was administered.

DISCUSSION

Tumor-to-tumor metastases are rare. In 1930, Fried reports first carcinoma metastasis into a meningioma. Two criteria that are used to assess a true tumor-to-tumor metastasis are as follows: 1) metastatic focus enclosed by a rim of benign histologically distinct host tumor tissue and 2) proven primary carcinoma. This case satisfied both of the above criteria. It is unusual for a tumor to metastasize into another tumor. When this phenomenon occurs, meningiomas are the most common intracranial lesion to harbor metastases. Thus, meningiomas seem to be a favorable environment for metastatic seeding and growing. Mechanisms responsible for tumor metastasis into meningioma have been proposed. First, meningiomas are the highly vascularized and exhibit slow growth. Second, their high collagen and lipid content has been postulated to provide a “fertile soil” for the seeding of malignant cells, immunologic factors and expression of certain surface adhesion molecules, E-cadherin in particular, may play a role. However, the precise mechanism by which this unique phenomenon occurs still remains undefined.

Meningiomas are generally indolent tumors that are treated by surgery alone. However, meningiomas that resulted from metastatic carcinoma may exhibit an unusual aggressive character, requiring postoperative radiotherapy for optimal prognosis.

For preoperative diagnosis of a metastasis inside a meningioma, MRI may be helpful, but not reliable enough to use it exclusively. Supplementing the diagnostic procedure by the use of
magnetic resonance spectroscopy as well as the diffusion-weighted magnetic resonance might increase the sensitivity of the diagnosis. There was one reported case that spectroscopy showed characteristic peaks suggestive of malignancy in the meningioma. However, the reliability of these investigations for this specific issue has not been sufficiently defined and ascertained. In the present case, the rapid clinical progression of motor deficits in combination with the rapid increase of the tumor size, and the inhomogeneous tumor appearance in MRI suggested a malignant degeneration of the meningioma or a metastasis within it. As the neuropathologic examination revealed, the rapid growth of the tumor mass and the corresponding progressive clinical deterioration were mainly due to the enlargement of the metastatic mass.

**CONCLUSION**

The possibility of tumor-to-meningioma metastasis should be considered when a sudden clinical deterioration or a rapid tumor growth occurs in patients with a cancer history.

**References**

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**Fig. 5.** Microscopic images. A: The fibrous meningioma with overlying dura of no carcinoma-bearing area (H-E, ×40). B: Higher magnification shows a streaming arrangement of elongated spindle cells resembling fibroblasts and psammomatous calcifications (H-E, ×200). C: In carcinoma-bearing areas, cancer glands are irregularly scattered in fibrous meningioma (H-E, ×200). D: Immunohistochemical staining for TTF-1 shows strong nuclear positivity in glandular structures (×100).