

Intraparenchymal Sylvian Fissure Meningioma

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Meningiomas arise from meningothelial cells that line the arachnoid membrane. So most meningiomas are dural-based lesion. But meningiomas without dural-attachment do occur and are less common. We report our experience of intraparenchymal sylvian fissure menigioma. A 21-year-old female presented with a one-month history of headache that was associated with long-term intermittent partial seizure. CT revealed about $4.5 \times 4.3 \times 5.5$ cm sized calcified mass with enhancement in right temporal lobe. On MR imaging, the lesion was observed in the right temporal lobe that was low-signal intensity on T2WI and iso-signal intensity on T1WI with well enhancement. Operation was performed via right orbitocranial approach. The internal surface of dura was intact. Tumor was totally removed except the capsule of tumor adhered to main trunk of middle cerebral artery. The histopathology showed meningioma, psammomatous type. Intraparenchymal meningioma should be considered in the differential diagnosis of intraaxial lesions in patients of any age group.

KEY WORDS: Intraparenchymal · Meningioma · Sylvian fissue.

Introduction

eningiomas represent approximately 15-20% of primary intracranial tumor in adults and have common location¹⁾. Meningiomas arise from meningothelial cells that line the arachnoid membrane. Therefore, most meningiomas are dural-based lesions and commonly located along the falx, along sphenoid bone, or over the convexity. However, meningiomas rarely arise from the stromal cells of the pia-arachnoid that invest the perforating blood vessels. These meningiomas will appear either within the sylvian cistern or within the ventricle²⁾.

An entirely intraparenchymal meningioma is rare. We report our experience of intraparenchymal sylvian fissure menigioma.

Case Report

The patient was a 21-year-old female who suffered from headache for a one-month. She presented with long-term episodes of partial seizure. On admission, she was distressed with headache, but neurologically intact.

The computerized tomography (CT) scan revealed about $4.5 \times 4.3 \times 5.5$ cm sized calcified mass with enhancement and mass effect in right temporal lobe (Fig. 1A). On Magnetic resonance imaging (MRI), a lesion was observed in the right tem-

poral lobe and iso-signal intensity on T1WI with well enhancement (Fig. 1B, C, D). The mass was associated with minimal perilesional edema. MR Angiography reveals displacement of right MCA and its branches to the medial and superior portion. The mass was stained from Rt.MCA. The provisional diagnosis was cavernous angioma, oligodendroglioma or Ganglioglioma.

Operation was performed via right orbitocranial approach. The internal surface of dura was intact (Fig. 2A). The morphology and color of arachnoid and pia mater were also normal. A solid tumor could be palpated within the temporal lobe. Tumor was totally removed except the capsule of tumor adhered to main trunk of middle cerebral artery (Fig. 2B). Postoperative CT showed remnant mass around right middle cerebral artery (Fig. 1E, F) Pathologic examination of the surgical specimen disclosed highly cellular tumor showing syncytial and fibroblastic cells (Fig. 2C). Microscopically the lesion included classical meningeal whorl as well as perivascular fibroblastic proliferation and multiple cyst. Dystrophic calcification was seen and the cells were moderately differentiation, however, mitosis or necrosis were not seen. It was immunopositive for EMA and Ki-67 labeling index was less than 1% (Fig. 2D).

The final diagnosis was psammomatous intraparenchymal meningioma. One year later, the patient have right hemiparesis with motor grade IV.

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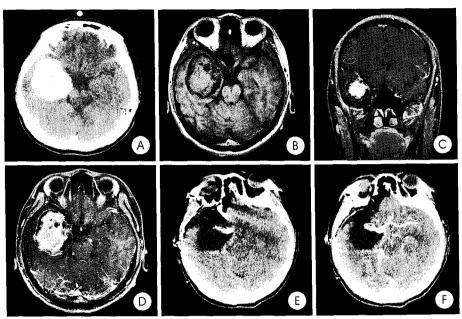


Fig. 1. Computed tomography (CT) scan reveals about 4.5X4.3X5.5cm calcified mass with enhancement and mass effect in right temporal lobe (A). On magnetic resonance image (MRI), a lesion is observed in the right temporal lobe and iso—signal intensity on T1WI with enhancement (B, C, D). Postoperative CT shows remnant mass calcified (E, F)

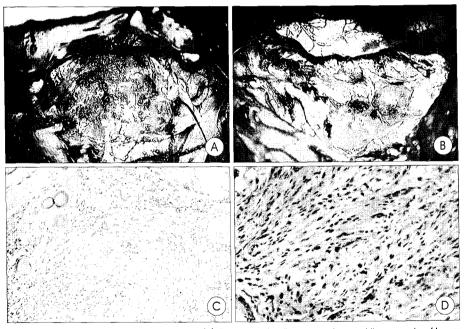


Fig. 2. The internal surface of dura was intact (A). Tumor was totally removed except the capsule of tumor adhered to main trunk of middle cerebral artery (B). Pathologic finding of the surgical specimen discloses highly cellular tumor showing syncytial and fibroblastic cells, and dystrophic calcification is seen (C, H&E X100). It was immunopositive for epithelial membrane antigen (D X200).

Discussion

M ost meningiomas are dural-based lesions but meningiomas without dural attachment occur. Intraparenchymal meningioma is used to describe tumors that arise within the brain tissue and characterize meningiomas that are not

dural based^{4,8)}. The key feature in the neuroimaging diagnosis of intraparenchymal compared with dural meningioma is the absence of dural attachment. These lesions are believed to arise from ectopic meningothelial cells within the stroma of the choroids plexus, tela choroidea or the pia mater. Typical locations where such a meningioma may develop without dural attachment include the intraventricular region, pineal region, and within the sylvian fissure^{6,9,10)}.

It is a rare entity, especially in adults. However, 12.5% of childhood meningiomas are intraparenchymal, and are mostly ventriclerelated⁶⁻⁸⁾. There are meningiomas in the deep sylvian cleft anchored to the internal carotid artery and its branches⁶. Almost all cases of sylvian fissure meningiomas are divided into primary sylvian fissure meningiomas and meningiomas secondary to underlying meningoangiomatosis2). Pirmary sylvian fissure meningiomas was located in the sylvian fissure attached to the internal carotid artery and its branches. The suspected cells of origin are the stromal cells of the pia-arachnoid that wrap the perforating blood vessels as they enter the surface of the brain⁸⁾. Mut et al reported that the intraparenchymal meningioma was associated with an underlying meningoangiomatosis, rare meningovascular lesion characterized by proliferation of meningothelial and fibroblast-like cells encircling small cortical vessels5). A clonal proliferation of perivascular meningothelial

cells encircling the vessels of the meningoangiomatosis, possibly secondary to aberrant production of growth factors or loss of inhibitory factors, such as NF2 gene inactivation, may have led to meningioma formation. Biopsy sampling of the surrounding brain pare-nchyma in cases of intraparenchymal meningioma may help to identify cortical meningoangiomatosis.

Our case can be classified into the sylvian fissure meningioma. The tumor capsule was anchored to the main trunk of middle cerebral artery and this finding suggest that the origin might be the stromal cells of the pia-arachnoid around the perforating vessels.

Differential diagnosis of intraparenchymal meningiomas is very difficult preoperatively. Our case did not reveal any of classic findings of extraaxial meningioma on radiologic studies, such as broad-based dural attachment, signal changes in skull due to tumor infiltration and perilesional edema. However, other findings of meningiomas were present, such as a sharp demarcation between the tumor and the brain, homogenous enhancement after intravenous administration of a contrast agent and calcification. In these intraparenchymal meningiomas, radiologic characteristics such as cystic formation, absence of dural attachment, presence of peritumoral edema and sarcomatous lesions have been relatively common and thus it is often difficult in the preoperative diagnosis to distinguish meningioma from high-grade glioma, cavernous angioma, or metastatic brain tumors^{3,8)}. Therefore, these neoplasms should be considered in the differential diagnosis of intraaxial lesions.

Conclusion

 ${f I}$ ntraparenchymal meningiomas are rare. Preoperative diagnosis is difficult considering the rarity and the similarity

of imaging finding to other more common intra-axial lesions. However, these neoplasms should be considered in the differential diagnosis of intraaxial lesions in patients of any age group.

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