Giant Skull Base Atypical Meningioma Presenting With Rapidly Progressive Impaired Consciousness Caused by Severe Venous Congestion

—Case Report—

Shigeru YAMAGUCHI,1 Shunsuke TERASAKA,1 Hiroyuki KOBAYASHI,1
Katsuyuki ASAOKA,2 Junichi MURATA,3 and Kiyohiro HOUKIN1

1Department of Neurosurgery, Graduate School of Medicine,
Hokkaido University, Sapporo, Hokkaido;
2Department of Neurosurgery, Teine-Keijinkai Hospital, Sapporo, Hokkaido;
3Department of Neurosurgery, Sapporo Azabu Neurosurgical Hospital, Sapporo, Hokkaido

Abstract

A 43-year-old female presented with a giant skull base atypical meningioma manifesting as rapid progression of impaired consciousness. The meningioma was located in the ethmoid sinus, sphenoid sinus, nasal cavity, and left middle temporal fossa, and the intracranial portion of the tumor involved the left temporal region with massive surrounding brain edema in the left temporal lobe and basal ganglia. She underwent emergent fronto-temporo-parietal decompressive craniectomy, and the intracranial portion of the tumor was resected to control intracranial pressure. She recovered consciousness and neurological function dramatically, and subsequently underwent radical tumor resection via combined extended transbasal and left lateral transzygomatic infratemporal fossa approaches one month after the initial surgery. The extensive brain edema completely disappeared after tumor resection, and the patient fully recovered without neurological deficits except anosmia and small visual field defect.

Rapid neurological deterioration and disturbance of consciousness caused by extensive peritumoral brain swelling are unusual in meningioma. In this case, the extemporaneous decompressive craniectomy was highly useful in the management of increased intracranial pressure.

Key words: atypical meningioma, decompressive craniectomy, impaired consciousness, skull base meningioma, venous congestion

Introduction

Peritumoral brain edema is frequently associated with meningioma, and elevation of intracranial pressure caused by peritumoral edema is one of the most serious complications of meningioma.4,7,25) The pathophysiology of the edema remains unknown, but tumor-related obstruction of venous drainage may be a causative factor.1,8,9) The extent of this edema is highly variable. This condition has been extensively investigated,1–3,11,14,16,19,21,22,26) but the relationship between peritumoral brain edema and tumor-induced obstructions of adjacent venous systems remains controversial.9,11,20) The cerebral venous system is generally assumed to provide collateral venous drainage around the obstructing lesion because meningiomas usually grow slowly.

Investigation of tumor-related venous obstruction in the development of peritumoral edema in meningioma found that tumor-related obstruction of the sylvian veins was associated with more brain edema.1) In addition, patients with sphenoid ridge and tempo-basal (middle fossa) meningiomas tend to develop more peritumoral brain edema.5,6,10,21) Venous drainage of the occluded sylvian veins leads to increased capillary pressure, which may disturb the balance between formation and resorption of brain edema.1)

We describe a case of a giant skull base atypical meningioma involving the paranasal sinus and the left middle temporal fossa manifesting as rapidly progressive impaired consciousness.

Case Report

A 43-year-old female initially presented with recurrent headaches persisting for 3 months. She was admitted to our hospital after decreased consciousness continued over several hours. Magnetic resonance (MR) imaging revealed a giant homogeneously enhanced extracranial mass occupying the ethmoid sinus, sphenoid sinus, nasal cavity, and left middle temporal fossa (Fig. 1A–C). The intracranial-
Fig. 1 A–C: Axial (A), coronal (B), and sagittal (C) T1-weighted magnetic resonance images with gadolinium on admission demonstrating a homogeneously enhanced mass lesion involving the ethmoid sinus, sphenoid sinus, and nasal cavity, and extending to the left middle temporal fossa. D, E: Axial T2-weighted magnetic resonance images on admission clearly revealing peritumoral brain edema involving the left temporal lobe and basal ganglia with severe mass effect. F: Axial computed tomography scan after resection of the intracranial portion of the tumor and external decompression revealing marked brain swelling of the left temporal lobe and basal ganglia.

Fig. 2 Left internal carotid angiograms (lateral view) after initial surgery revealing stasis of the left sylvian vein (arrowheads). A, B: Arterial phase; C, D: venous phase.

e al portion of the mass involved the left temporal region and was accompanied by extensive ipsilateral hemispheric brain swelling and significant mass effect (Fig. 1D, E). Corticosteroid and hyperosmotic solution were administered intravenously immediately after admission, but her disturbed consciousness did not improve and anisocoria was subsequently observed. Therefore, emergent left fronto-temporal craniotomy for resection of the intracranial portion of the tumor was performed on the day of admission. The tumor was fragile, reddish, and hypervascular. Severe venous congestion was anticipated because “to-and-fro” sign of the ipsilateral sylvian veins was observed intraoperatively. Intracranial pressure remained elevated despite total removal of the intracranial tumor, so left fronto-temporo-parietal decompressive craniectomy was performed (Fig. 1F). Postoperatively, corticosteroid and hyperosmotic solution administration was required for about 2 weeks.

One week after the initial operation, she underwent angiography. The tumor was supplied by the bilateral external carotid arteries. Left internal carotid angiography showed that the left sylvian vein was cut off abruptly, and stasis was present in the late venous phase (Fig. 2). The left cavernous sinus and sphenoparietal sinus were completely occluded. In addition, the ipsilateral vein of Labbe and basal vein of Rosenthal did not appear in the venous phase of left carotid angiography.

She remained comatose for a week, but fully recovered consciousness 3 weeks after the initial operation. Her neurological deficit also improved, although she had severe left visual impairment. One month after surgery, she underwent radical resection of the remaining tumor in the sphenoid and ethmoid sinuses extending to the pterygopalatine fossa. Following endovascular embolization of the accessory meningeal artery, tumor resection was performed via the anterior and lateral routes through the extended transbasal approach and lateral transzygomatic infratemporal fossa approach, respectively, as described in detail elsewhere.23,24 Large bifrontal craniotomy combined with left temporal craniotomy was performed. Subfrontal dura mater was dissected from the orbital roof and the bilateral olfactory nerves involved in the tumor were interrupted. The bony architecture of the frontal base was already destroyed, and the grayish tumor occupying the ethmoid and sphenoid sinuses was exposed. The tumor did not bleed, and internal decompression of the mass was performed using an ultrasonic aspirator. The bilateral optic canals were preserved intact. Although the tumor did not invade the clival dura or the medial wall of the cavernous sinus, vast bleeding was encountered during dissection of the tumor. Subsequently, the head was rotated to the right, and the left infratemporal fossa was dissected. The second and third branches of the trigeminal nerve were freed from the foramen, and the tumor in the pterygopalatine fossa was removed. Finally, the sphenoid sinus was opened via the lateral route, keeping the vidian canal intact. Gross total resection of the mass was achieved.

Histological examination of the extracranial tumor revealed low grade meningothelial meningioma. However, the specimen from the intracranial part of the tumor was highly cellular with scattered mitotic figures, and the proliferation rate determined by MIB-1 labeling index was 18% (Fig. 3). The histological diagnosis was atypical...
Fig. 3 Photomicrographs of the intracranial part of the tumor revealing a highly cellular tumor with scattered mitotic figures (arrow), and proliferation index of 18%. Hematoxylin and eosin stain (A, B) and Ki-67/MIB-1 immunostain (C), original magnification \( \times 100 \) (A) and \( \times 400 \) (B, C).

Fig. 4 A, B: Axial (A) and sagittal (B) T1-weighted magnetic resonance images with gadolinium showing a small residual tumor located in the nasal cavity (arrow) 4 months after radical resection of the tumor. C, D: Axial T2-weighted magnetic resonance images showing resolution of the brain edema in the left temporal lobe and basal ganglia.

meningioma.

The patient had an uneventful recovery without additional neurological deficit except anosmia. Left visual impairment was also improved gradually, and only a small visual field defect of her left eye remained. One month after radical tumor resection, she underwent cranioplasty using custom-made titanium mesh plates (Muranaka Medical Instruments Co. Ltd., Osaka). MR imaging performed 4 months postoperatively revealed a small residual tumor in the nasal cavity and complete resolution of cerebral edema (Fig. 4). Since the residual tumor size had slightly increased one year later, she underwent fractionated stereotactic radiotherapy limited to the residual tumor.15)

She remained well 2 years after surgery.

Discussion

In the present case, uncal herniation caused by the rapidly growing tumor located in the middle temporal fossa was an important factor for her impaired consciousness. In addition, severe brain edema was observed in the left temporal lobe and basal ganglia. This peritumoral edema increased unpredictably, leading to unexpected rapid progression of impaired consciousness and neurological deterioration. Since the ipsilateral sylvian veins, sphenoparietal sinus, and cavernous sinus were occluded by the tumor, this unexpected peritumoral brain edema may have been caused by insufficient development of the venous collateral system in the ipsilateral cerebral hemisphere.

In this case, we could not perform angiography preoperatively because of the rapid progression of impaired consciousness. Angiography performed after decompression confirmed venous stasis in the sylvian veins and obstruction of the ipsilateral cavernous sinus. The tumor had blocked the drainage system, leading to extensive edema in the ipsilateral cerebral hemisphere. In addition, the venous collateral circulation of the left temporal lobe and basal ganglia, such as the inferior anastomotic vein of Labbe, inferior temporal vein, and basal vein of Rosenthal, were not detected. The failure to develop these venous channels might have facilitated the massive and wide-ranging venous congestion.

Decompressive craniectomy is efficacious for patients with refractory brain edema after tumor excision.5) In this case, decompressive craniectomy was also suitable for control of intracranial pressure until the collateral pathways around the obstructive lesion developed. Subsequently, she underwent resection of the intracranial part of the tumor via a pterional surgical approach during the initial surgery. We anticipated that the peritumoral brain edema would increase initially because injury to the sylvian veins with the pterional approach frequently causes postoperative damage to the brain parenchyma.12) Therefore, the normalization of intracranial pressure both improved her consciousness and neurological status but also allowed safe resection of the residual tumor.

The tumor extended to the intracranial and extracranial regions around the left cavernous sinus and the majority of the tumor was located in the ethmoid sinus, sphenoid sinus, and nasal cavity. It was difficult to determine whether the origin of this tumor was extracranial or intracranial. Generally, total resection of meningioma in the cavernous sinus is not always feasible.16) However, in high grade meningiomas, the extent of surgical resection is apparently correlated with the clinical outcome of meningioma patients.13,17) In addition, considering the complications of radiotherapy, visual disturbances or hypopituitarism resulting from removal of tumor around the optic nerves and sella turcica had to be avoided. For lesions of the frontal fossa and paranasal sinus that extend into the cavernous sinus, an extended transbasal approach is suitable for detachment of the tumor from the optic canals and...
sella turcica.\textsuperscript{23} We achieved maximum safe tumor resection by this extended transbasal approach combined with a left lateral transzygomatic infratemporal fossa approach. A small residual tumor remained in the nasal cavity, and so stereotactic radiotherapy was performed for this lesion without neurological or endocrinological complications.

References


Address reprint requests to: Shunsuke Terasaka, MD, Department of Neurosurgery, Graduate School of Medicine, Hokkaido University, North-5th, West-7th, Kita-ku, Sapporo, Hokkaido 060–8638, Japan. e-mail: terasas@med.hokudai.ac.jp