Meningioma Associated with Intratumoral Abscess Formation
—Case Report—

Tsuyoshi SHIMOMURA, Shigeaki Hori, Naoto KASAI, Kenichi TSURUTA*, and Hitoshi OKADA**

Department of Neurosurgery, Oita Medical University, Oita;
*Department of Neurosurgery, Kenwakai Otemachi Hospital, Kitakyushu, Fukuoka;
**Department of Neurosurgery, Omiya Red Cross Hospital, Yono, Saitama

Abstract
A rare case of meningioma associated with intratumoral abscess formation occurred in a 64-year-old female presenting with septic meningitis and a right frontal mass lesion after a gynecological operation under spinal anesthesia. The mass lesion was totally removed and revealed as an incidental meningioma with an intratumoral abscess. Hematogenous infection of Bacteroides oralis was thought to be the cause of the intratumoral abscess formation.

Key words: meningioma, abscess, septic meningitis, intratumoral abscess formation

Introduction
Intratumoral abscess formation is very rare, especially in intracranial tumors where the major location is pituitary lesions directly extending from infected sinuses. Previous cases of the coexistence of meningioma and abscess have not been intratumoral abscesses but postoperative foreign body granuloma. Here we report a case of meningioma associated with intratumoral abscess.

Case Report
A 64-year-old female was admitted to the Department of Gynecology in Kenwakai Otemachi Hospital on February 16, 1988, with lumbago. One year previously, she had been admitted to another hospital for 3 months because of sudden onset of left hemiparesis due to right cerebral infarction. On February 23, 1988, she underwent an operation for a prolapsed uterus under spinal anesthesia. On the 10th postoperative day, she became slightly drowsy and her temperature rose to 39°C. Precontrast computed tomography (CT) demonstrated an isodense mass with perifocal edema in the right frontal pole, and a low-density area in the right corona radiata due to the cerebral infarction in the previous year (Fig. 1 left).

She was transferred to the neurosurgical department on March 10. Neurological examination reveal—

Fig. 1 left: Precontrast CT scan on March 4, showing an isodense round mass with faint perifocal edema in the right frontal lobe and an old infarction in the right corona radiata. right: Precontrast CT scan on March 16, showing higher density of the right frontal mass and enlarged perifocal edema.
ed meningeal irritation signs and slight left hemiparesis. Laboratory blood tests showed signs of severe inflammation: white blood cell count 18,600/mm³, erythrocyte sedimentation rate 120 mm/hr, and C-reactive protein 14.1 mg/dl. Cerebrospinal fluid (CSF) obtained by lumbar puncture was cloudy and contained 1579/mm³ of white blood cells, 1100 mg/dl of protein, and 38 mg/dl of glucose. Culture of the CSF was negative but the blood contained *Bacteroides oralis*. She was also suffering from heart failure and myxoedema caused by hypothyroidism due to chronic thyroiditis. Further precontrast CT on March 16 demonstrated the right frontal mass as a denser area than before and the perifocal edema had enlarged (Fig. 1 right). Right carotid angiography revealed a hypovascular mass lesion with a slight marginal brush in the right frontal pole.

The diagnosis was a combination of sepsis, septic meningitis, and brain abscess. She was treated heavily with antibiotics: intrathecal piperacillin (0.5 g) and gentamicin (10 mg) once a week for 4 weeks, intravenous latamoxef (3 g) every day for 2 weeks, and intravenous piperacillin 6 g a day for 1 week and 4 g a day for 3 weeks. Thyroxine replacement therapy for her hypothyroidism and intravenous injection of diuretics for heart failure were also given. The signs of inflammation gradually subsided and she became almost alert, although the low-grade fever was continued. CSF examination on April 16 revealed white blood cell count 28/mm³, protein 450 mg/dl, and glucose 43 mg/dl. She became euthyroid and recovered from the myxoedema and heart failure. CT on April 26 demonstrated decreased perifocal edema.

On May 6, she underwent a right frontal craniotomy to treat the cerebral abscess. An elastic hard tumor, 2.5 cm in diameter, originating from the anterior falx and covered by thick, cloudy arachnoid membrane was totally removed with little hemorrhage. The falx at the tumor origin was electrocoagulated.

Fig. 2 Photograph of the cut surface of the surgical specimen, showing intratumoral abscesses (arrows).

Fig. 3 upper: Photomicrograph of the tumor at the maximum diameter, showing meningioma cells more abundant where the tumor was attached to the falx (bottom). HE stain, ×8. middle: Photomicrograph of the tumor specimen, showing transitional type meningioma containing psammoma bodies. HE stain, ×100. lower: Photomicrograph of the wall of an abscess cavity containing infiltration of numerous inflammatory cells such as neutrophils and plasmacytes and proliferated capillaries. HE stain, ×100.
The cut surface of the surgical specimen was yellowish-white, and revealed several small cavities, 1–4 mm in diameter, containing yellowish pulpy pus (Fig. 2). Histological examination revealed transitional type meningioma cells and psammoma bodies mainly in the portion adhered to the falx (Fig. 3 upper, middle), intratumoral abscess cavities containing numerous inflammatory cells such as neutrophils and plasmacytes, and proliferated capillary vessels (Fig. 3 lower). Gram-negative rods were identified in the abscess cavities.

The postoperative course was uneventful. She received chemotherapy with cefoxitin (2 g) every day for 10 days. She was discharged without inflammatory findings or neurological deficits other than slight left hemiparesis due to the previous cerebral infarction.

Discussion

Excluding direct infections, only three cases of intratumoral abscesses have been reported: one within a metastatic brain tumor caused by Salmonella enteritidis, and two within glioblastomas caused by Salmonella enteritidis or Staphylococcus aureus. All three cases resulted from hematogenous infection. Noguerado et al. suggested that long-term steroid therapy and the immunosuppressive effect of glioblastoma had probably been factors in the etiology of the abscess. Ichikawa et al. reported intratumoral abscess formation within multiple glioblastomas associated with intratumoral hematoma, and considered that steroid therapy, lack of blood-brain barrier, and nutritious hematoma within the tumor may have induced the abscess formation by sepsis following phlebitis. Brain abscess developing after cerebral infarction or intracerebral hematoma due to destruction of the blood-brain barrier and the reduction of the local immunological system against bacteria have also been reported.

In our case, the association of meningioma and abscess occurred in a patient who developed septic meningitis and sepsis following an operation for prolapsed uterus under spinal anesthesia. No bacterial culture of the intracranial tumor was obtained, but the intratumoral pus contained Gram-negative rods characteristic of Bacteroides oralis, which also present in the blood culture before the intracranial operation. Therefore, this organism was probably the cause of the abscess. The origin of the intratumoral abscess may have been direct extension of meningitis or hematogenous spread of sepsis. Histological study of the operative specimen revealed the abscess inside the tumor, not around the tumor. Ichikawa et al. speculated that absence of a blood-brain barrier contributed to hematogenous infection in glioblastoma and metastatic brain tumor. Meningioma may also cause fenestration of vessels and destruction of the blood-brain barrier like metastatic brain tumor and glioblastoma. Therefore, in our case, hematogenous infection is more likely as a cause of intratumoral infection than infection secondary to meningitis. Her poor general condition and reduced local immunity probably facilitated the infection resulting in the abscess within the meningioma.

References


Neurol Med Chir (Tokyo) 34, July, 1994


*Address reprint requests to:* T. Shimomura, M.D., Department of Neurosurgery, Oita Medical University, 1-1 Idaigaoka, Hasama-machi, Oita-gun, Oita 879-55, Japan.