

Postoperative Contralateral Acute Subdural Hematoma after Removal of Parasagittal Meningioma

Kun-Chieh Yeh, Wei-Chieh Chang, Meng-Yin Yang, and Chiung-Chyi Shen*

Department of Neurosurgery, Taichung Veterans General Hospital, Taichung, Taiwan, Republic of China

A well-enhanced extra-axial meningioma over right frontal parasagittal region was incidentally found in this 53-year-old male. There was no neurological deficit. Right frontal craniotomy with removal of tumor was smoothly performed under general anesthesia. Unfortunately, contralateral anisocoric pupil size was found right after the operation. CT scan of Brain showed contralateral acute SDH with mass effect and uncal herniation. Subsequent craniotomy on the other side was carried out for decompression. Literatures about postoperative intracranial hemorrhage have been reviewed and discussed^{1,2,3}, but remote site acute SDH is rare. We report this rare case and discuss about possible pathophysiology.

Key words: meningioma, postoperative hemorrhage, remote site hemorrhage

Abbreviation: CT, MRI, SDH, POH, SAH, ICH, IICP.

INTRODUCTION

Postoperative intracranial hemorrhage (POH) is a serious and sometimes a fatal neurosurgical complication. And it usually occurs at the site of operation but surprisingly it can be presented in sites remote from the original surgical access as epidural, subdural, or intracerebral hemorrhage.

A number of studies^{1,3} have demonstrated the significant morbidity and mortality associated with intracranial bleeding after neurosurgery. Risk factors include pre-existing medical comorbidities such as hypertension, coagulopathies, hematological abnormalities, intraoperative hypertension and blood loss, certain tumor pathologies, and chronic subdural hematomas.^{2,4,5,6}

We reported a rare case of left fronto-temporo-parietal acute SDH right after the surgical removal of right frontal parasagittal meningioma.

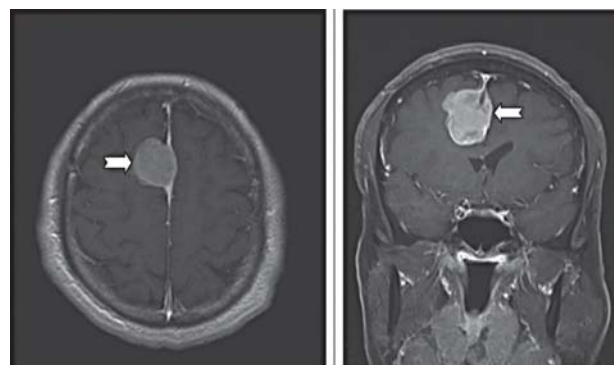


Fig. 1 T1-weighted MR imaging with gadolinium revealed well-enhanced extra-axial mass lesion over right parasagittal region of high frontal area with increased adjacent pachymeningeal enhancement.

CASE REPORT

This 53-year-old male had no hematopoietic disease in the past. Right frontal parasagittal meningioma was diagnosed by physical check-up. Surgical removal was suggested to prevent from possibly ongoing cerebral venous thrombosis because of the tumor invaded into the sagittal sinus. There was no neurological deficit. Routine blood sampling was normal including coagulating factors. T1- and T2- weighted magnetic resonance (MR) imaging demonstrated a gadolinium-enhanced extra-axial tumor lesion ($2.9 \times 3.1 \times 3.5$ cm) over right frontal and parasagittal region with increasing adjacent pachymeninges (Fig. 1). In addition, focal subcortical lesion with high T2WI

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*Corresponding author: Chiung-Chyi Shen, Department of Neurosurgery, Taichung Veterans General Hospital, No. 160, Sec. 3, Taichung-Kang Road, Taichung, Taiwan, Republic of China. Tel & Fax: +886-4-23741218; E-mail: shengeorge@yahoo.com

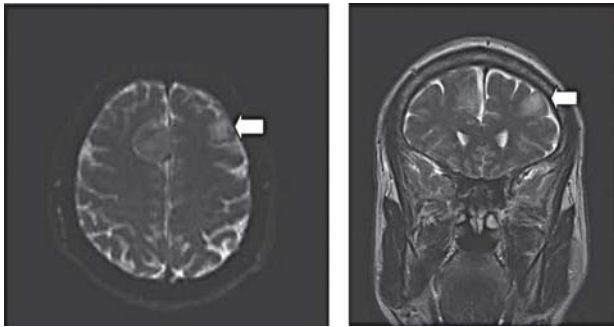


Fig. 2 Focal subcortical lesion with high T2WI signal intensity and without contrast-enhanced T1WI signal intensity over left frontal lobe.

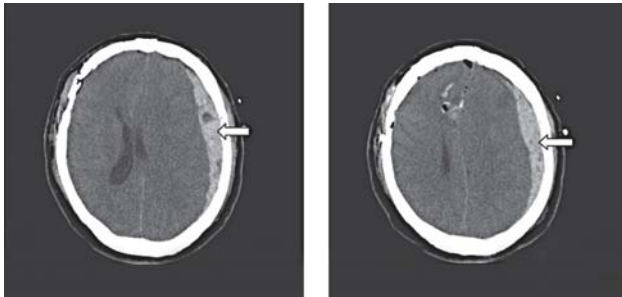


Fig. 3 CT scan of brain revealed subdural hematoma over left fronto-temporo-parietal region.

signal intensity and without contrast-enhanced T1WI signal intensity over left frontal lobe was noted (Fig. 2).

After thoroughly pre-operative surveys, right frontal craniotomy with removal of tumor was smoothly performed. The patient was positioned supine with the head flexed so the scalp over the center of the tumor is the highest point. The skin flap had been large enough to give adequate exposure around the tumor. The bone flap was carried about 2 cm across the midline to the side opposite the tumor. The dura over the convexity is cut at least 1 cm away from the tumor. Because the tumor involved only the edge of the sinus, the dura was initially cut a few millimeters parallel to the sinus, leaving a small plaque of tumor. The tumor was internally decompressed to avoid traction on the surrounding brain. Then, the capsule was carefully reflected into the area of decompression, dividing arachnoid and vascular attachments and protecting the brain with cottonoids. Finally, we removed the residual tumor by curette and bipolar coagulator. There was no intra-operative hypertension or massive intra-operative bleeding during this procedure. However, contralateral anisocoric pupil size was immediately noticed after uncovering surgical drapes. CT scan of brain

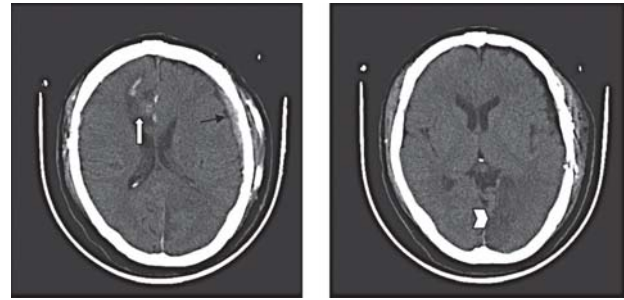


Fig. 4 Non-contrasted CT scan of brain revealed SDH at left fronto-parietal lobe (black arrow), ICH at right frontal lobe (white arrow) and left PCA infarction (obelisk).

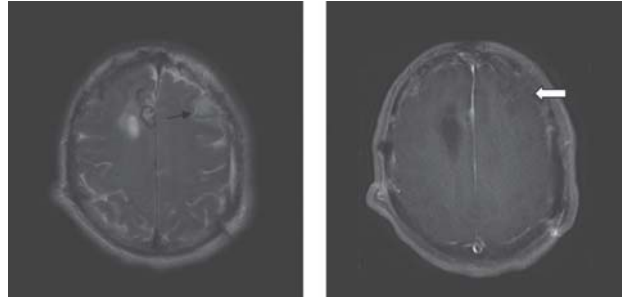


Fig. 5 Focal subcortical lesion with high T2WI signal intensity (black arrow) and without contrast-enhanced T1WI signal intensity over left frontal lobe (white arrow).

revealed left fronto-temporo-parietal acute SDH with mass effect and uncal herniation (Fig. 3). Subsequent left fronto-temporo-parietal craniotomy and evacuation of the subdural hematoma were performed (Fig. 4). No obvious vascular anomaly be found after removed from the hematoma. But a tiny end of drainage vein was identified.

Postoperatively, the patient made a progressive recovery. He was discharged home on the 28th post-operative day with right hemiplegia. Besides, MRI of brain was performed for follow-up 6 months after the surgery (Fig. 5). It still showed high signal lesion over left frontal lobe in T2-flare.

DISCUSSION

To the best of our knowledge, this is the first reported case of contralateral acute SDH after removal of frontal and parasagittal meningioma. In our case, there was no risk factor listed in previous reports of POH like pre-existing medical comorbidities such as hypertension, coagulopathies, hematological abnormalities, intraoperative

hypertension and blood loss⁷, certain lesion pathologies including tumors in situ, chronic subdural hematomas, and deficiencies in hemostasis.

This tumor was located in right frontal and parasagittal region and diagnosed as mixed meningothelial and fibrous type (WHO grade I) histologically.⁸ It is generally considered that this tumor induces less difficulty in its operative removal than that in other sites. For example, medial sphenoid wing⁹ or clivus¹⁰, which frequently involves important structures like the arteries and cranial nerves. In this case, bilateral anterior cerebral arteries were clearly found and protected during the operation without evidence of tumor invasion or compression. By the reason of possible sagittal sinus occlusion during the procedure, we protected the sagittal sinus very well and prevented from venous hypertension. There was no evidence of obstacle of sagittal sinus flow in the operation.

The rates of POH following intracranial procedures reported in the literatures vary greatly, ranging from 0.8% to 50.0%.^{1,2,11} Remote intracranial hemorrhages were even rare. It is defined as postoperative bleeding which occurs remotely from the operative site. Mostly, it has been described in posterior fossa surgery^{12,13}, evacuation of chronic subdural fluid collections¹⁴, and intratumoral bleeding after a shunting procedure.^{15,16} The common point was the significant change of ICP. It may sometimes require surgical evacuation. The risk factors for its development include hypertension^{1,12}, female gender^{12,13}, brain atrophy¹³, preoperative shunting procedures^{13,15,16}, upright positioning¹⁴, and preoperative ventricular dilatation.¹⁴

Subdural hematoma is associated with intracranial hypotension were reported in some articles.^{17,18} Nearly all kinds of intracranial lesion can cause IICP. That is why we perform neurosurgery to remove the intracranial lesions. During the operations, vigorous CSF outflow during surgery lowered the pressure in the syrinx cavity, leading to significant but transient intracranial hypotension and consequently the formation of subdural hematoma.⁴ In this case, right frontal craniotomy may lead to a pressure gradient between hemispheres. It may contribute to a widening subdural space and accumulation of fluid on the other side. Intraoperative tissue retraction and CSF drainage subsequently made it disable from restore its normal shape and created a subdural space. Furthermore, drainage vein over the brain surface might be tear and cause hemorrhage and hematoma formation.

In our case, pre-operative MRI of brain gave us a clue of another possibility of contralateral acute SDH. MRI disclosed a lesion over left frontal in T2-flare (Fig. 2). It

was highly suspected as a low grade glioma. The rate of brain tumors related intracranial hemorrhage is reported at 0.6–14.6%, whereas the incidence of gross bleeding in cases of gliomas of all grades is 3.7–7.2%.^{5,6,19,20} Patients with low-grade astrocytomas are thought to account for less than 1% of tumors presenting with hemorrhage³⁰ and there have been only a few reports of hemorrhages associated with pilocytic astrocytomas.^{21,22,23,24,25,26,27,28,29} The cause of hemorrhage is unclear but may be related to the abnormal vasculature within the tumor. The abnormal vascularization of the tumor also results in vessel wall which proved difficult to seal.³¹ Moreover, tumor enzyme activity destroying the tumor–brain barrier might be responsible for a lack of tumor capsule and a softening of brain tissues³², which upon decompression may subsequently become hyper-perfused and hemorrhagic.³³ But in our case, we didn't see the bleeder over left frontal lobe during the operation of removal of hematoma. Post-operative CT scan of brain also did not reveal hemorrhage over tumor-brain interface, such as SAH or ICH. MRI of brain which was performed for follow-up 6 months after the surgery (Fig. 5) still showed high signal lesion over left frontal lobe in T2-flare. The acute SDH in our case caused by spontaneous tumor bleeding is still less like by the reason of very low incidence.^{5,6,19,20}

Besides, in the study conducted by Boleslaw *et al.*³⁴, statistical analysis revealed a significant association between hemorrhages and retiform capillaries in three types of tumors (glioblastomas multiforme, oligodendrogliomas, and astrocytomas). Thus, the microcapillary circulation may still result in this rare complication. However, no obvious vascular anomaly was found during the decompression surgery. In our opinion, the cause of acute SDH in this case was first considered as transient intracranial hypotension and leading to drainage vein tear and hemorrhage. It was less likely the vascular lesion or tumor bleeding.

CONCLUSION

The most important keys to minimize the hazardous sequelae are to be aware of this potential complication, attempt to a precocious diagnosis and to provide prompt treatment in all cases. In our case, the most possibility of post-op remote acute SDH may be caused by transient intracranial hypotension during the procedure. Thus, brain retraction and CSF drainage should be kindly, not be excessive to avoid more severe intracranial hypotension.

Despite its rare occurrence, this unusual situation deserves to be highlighted as it can cause significant

morbidity if ignored or missed. Thus, routine imaging within 24–48 h of surgery^{35,36,37}, and up to 7 days post-procedure^{11,14,38} as a screening tool are suggested.

DISCLOSURE

The author declares that this study has no conflict of interest.

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