奈医誌. (J. Nara Med. Ass.) **45**, 132～137, 1994

POSTOPERATIVE VASOSPASM IN PITUITARY ADENOMA WITH PITUITARY APOPLEXY

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Abstract: A patient who developed complications relating to intracranial arterial vasospasm following transcranial removal of a pituitary adenoma with pituitary apoplexy is reported.

A 23-year-old female was admitted because of headache. Computed tomography (CT) and magnetic resonance (MR) imaging revealed a pituitary adenoma with suprasellar extension and pituitary apoplexy and sinusitis in the right maxillary and ethmoid sinuses. A transcranial approach was selected because of active sinusitis. A large, pink intra-and suprasellar neoplasm and intratumoral clot were removed smoothly. Postoperatively, the patient remained stuporous and showed right hemiplegia. Angiography demonstrated stenosis of the left internal carotid artery and severe spasm of perforating arteries from the left middle cerebral artery. Large pituitary adenoma with pituitary apoplexy requires careful perioperative management, with particular attention paid to the surgical approach and procedures.

Index Terms

pituitary adenoma, apoplexy, vasospasm

INTRODUCTION

Vasospasm remains one of the most difficult conditions confronted by neurosurgeons. Vasospasm after subarachnoid hemorrhage is a common and well-known entity, but vasospasm in association with pituitary adenoma has been rarely reported[1-10)6]. We describe a patient who developed complications relating to intracranial arterial vasospasm following transcranial removal of pituitary adenoma with apoplexy.

CASE REPORT

A 23-year-old female was admitted to Osaka Neurological Institute because of headache that had persisted for one month prior to admission. Neurological abnormalities on admission included left temporal hemianopsia. Her serum growth hormone level was 15.0 ng/ml(normal, <5 mg/ml)and prolactin level 29.4 ng/ml (normal, <15 ng/ml). Other tests of endocrine function were normal on baseline and provocative testing. General physical examination was

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normal. Computed tomography (CT) showed a slight high density with partial low density mass in the intra- and suprasellar lesion with no contrast medium enhancement, and showed sinusitis in the right maxillary and ethmoid sinuses. Magnetic resonance (MR) imaging revealed an intrasellar and suprasellar inhomogenous hyperintensity mass on T1 weighted images(WI)and T2WI which was compatible with pituitary apoplexy (Fig. 1), and no gadoliniumdiethylenetriaminepenta-acetic acid (Gd-DTPA) enhancement was seen. Angiography via femoral catheterization demonstrated stretching of the horizontal portion of the bilateral anterior cerebral arteries, suggesting suprasellar extension. The right sinusitis was active, and culture was positive, demonstrating Staphylococcus aureus, so a transcranial approach was selected.

A left frontotemporal craniotomy was carried out. A large, pink suprasellar neoplasm was encountered beneath the chiasm and both optic nerves. The tumor and intratumoral clot were removed easily with minimal bleeding. A normal pituitary gland was compressed superiorly by the tumor, and the pituitary stalk was seen and could be preserved. Autologous fat was placed into the tumor bed to prevent empty sella syndrome. No vascular spasm was evident at the time of closure. Pathological examination of the specimen demonstrated a chromophobe adenoma.

Postoperatively, the patient remained stuporous and showed right hemiplegia. Emergent CT on the day of operation revealed no abnormality (Fig. 2), but emergent angiography demon-

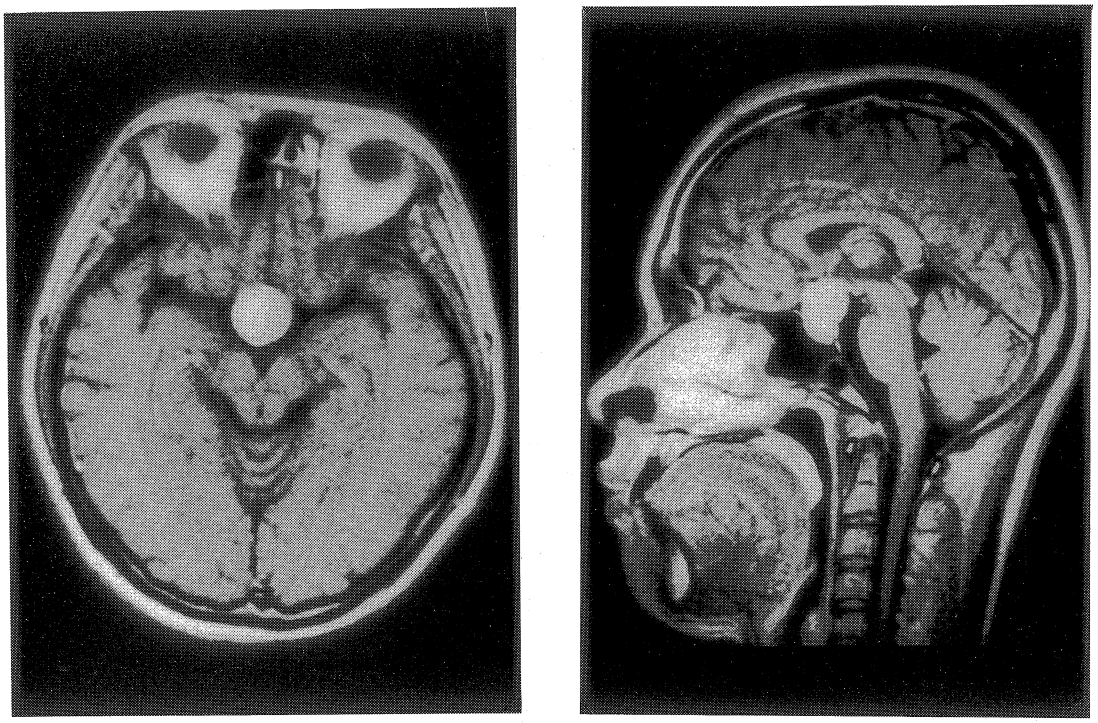

Fig. 1. T1-weighted (TR 600, TE 25 msec) axial (left) and sagittal (right) MR images, demonstrating an intrasellar and suprasellar inhomogenously hyperintense lesion which was compatible with pituitary apoplexy.

strated stenosis of the left internal carotid artery and severe spasm of perforating arteries from the left middle cerebral artery (Fig. 3). No explanation for the clinical deterioration other than the spasm was ever found. She was treated with low molecular dextran, hyperbaric oxygen therapy, glycerin and steroid. One day after operation, the consciousness level was clear, but motor aphasia and the right hemiparesis (2/5) remained. CT revealed a low density area in the right caudate head and frontoparietal lesion (Fig. 4). The symptoms improved gradually, and she was discharged with right mild hemiparesis (4/5) after a three-month admission.

DISCUSSION

Vasospasm after removal has been previously reported in pituitary adenoma, meningioma, medulloblastoma and acoustic neurinoma[1]. Pituitary adenoma is the most common, and to our knowledge 8 cases[2-6] have been reported as a complication following transcranial surgery for pituitary adenoma. The relationship between this sort of vasospasm and the vasospasm observed after subarachnoid hemorrhage has not yet been elucidated[5]. Several hypotheses can be advanced to explain the occurrence of vasospasm after pituitary surgery: intraoperative mechanical manipulation, intraoperative bleeding, vasoactive substances released from the tumor bed, and hypothalamic injury[47].

Mawk[4] reported three cases of cerebral vascular spasm following transfrontal removal of

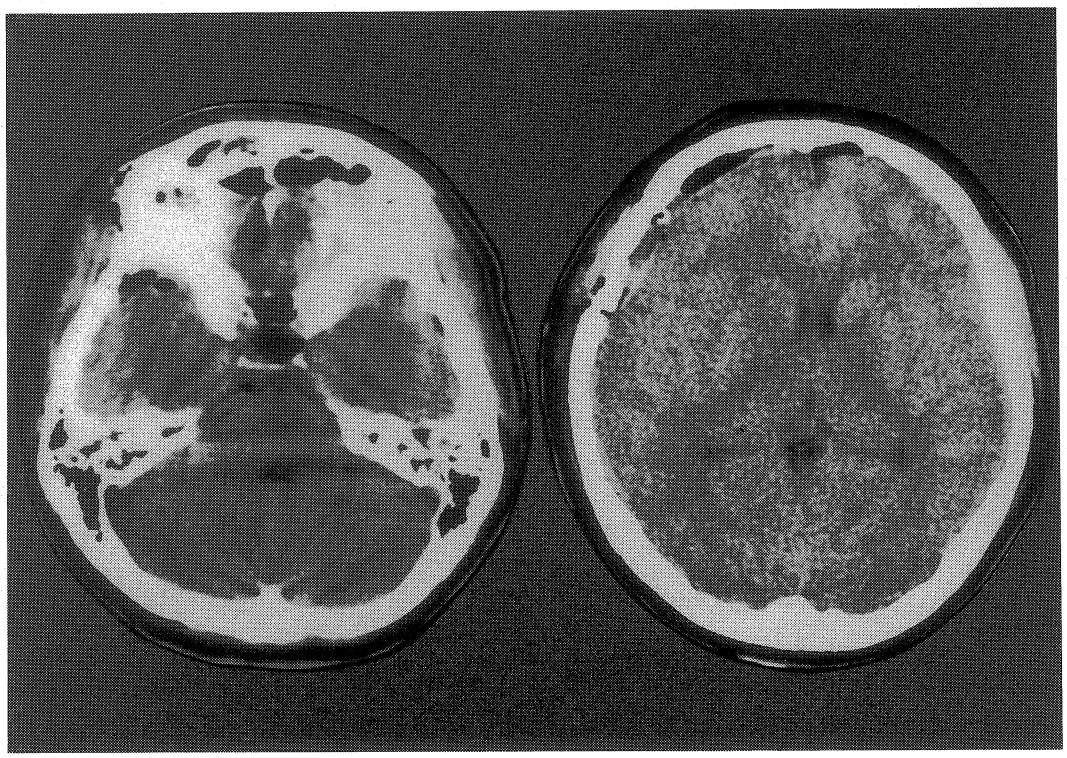

Fig. 2. Emergent CT just postoperatively, showing no abnormality except postoperative changes.

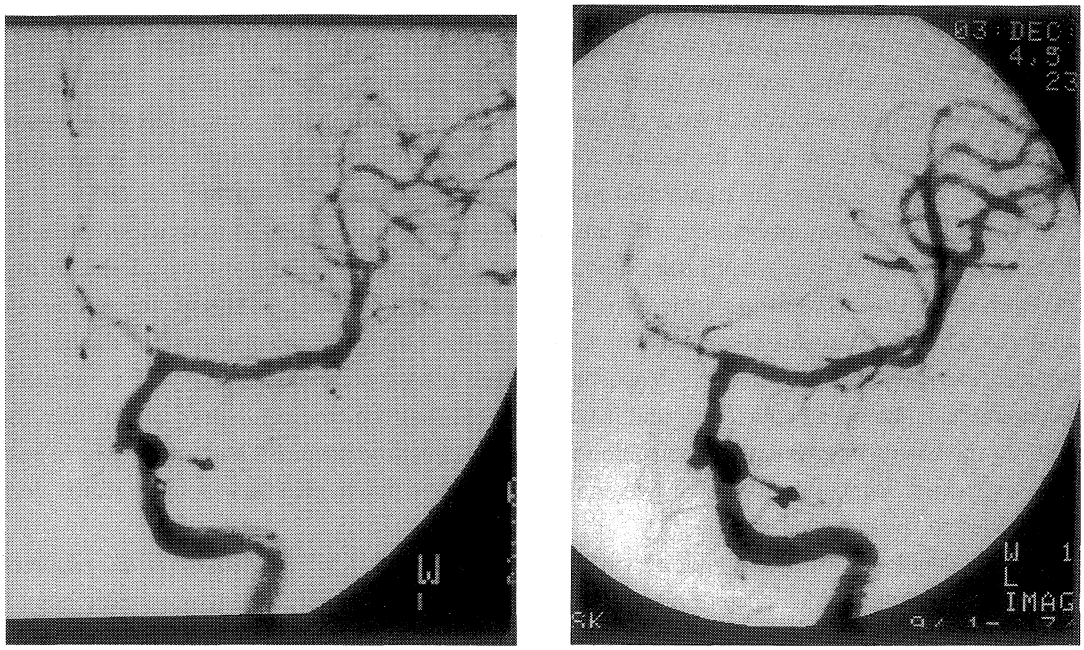

Fig. 3. left : Preoperative angiography, revealing elevation of the left anterior cerebral artery (Al portion). right : Postoperative angiography, demonstrating spasm of the left internal carotid artery and anterior cerebral artery with poor filling. Perforating arteries from the left anterior and middle cerebral artery were opaque.

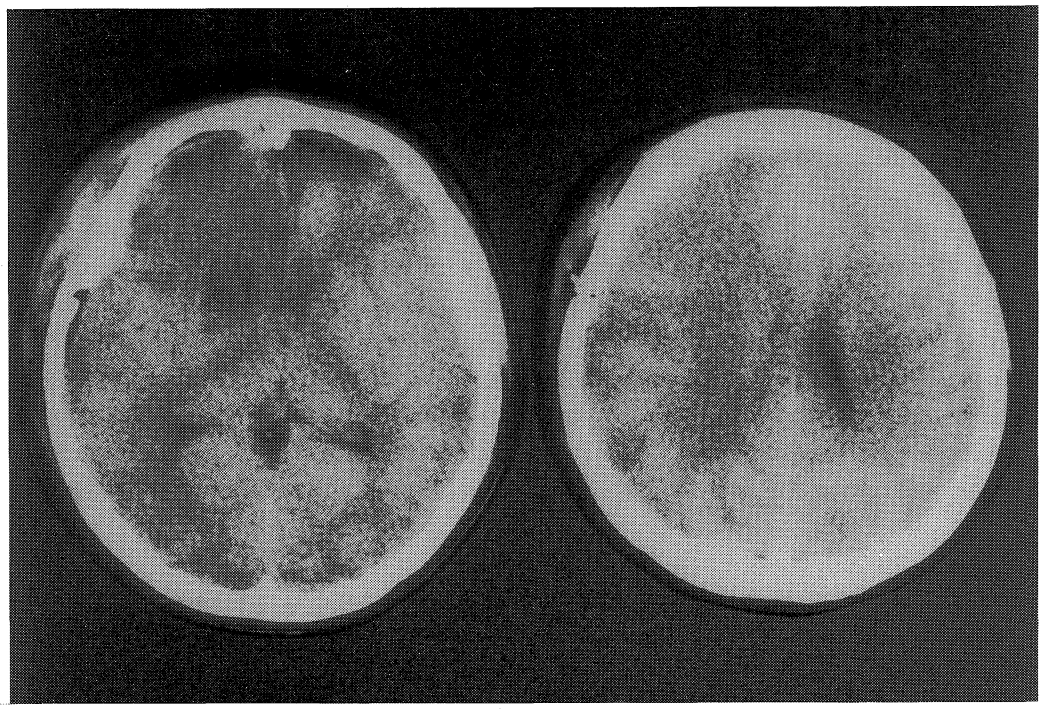

Fig. 4. One day after operation, CT revealed a low density area in the right caudate head and frontoparietal lesion.

large pituitary neoplasms, and experienced his fourth such case proved fetal in spite of extreme care[5]. He operated taking care not to open the arachnoid membrane surrounding the lesional internal carotid artery, and the tumor capsule was manipulated minimally. The patient developed progressive cerebral infarction in spite of volume-loading, low molecular weight dextran administration, and heparinization, and died. Mawk speculated that vasoactive substances released from the tumor bed may induce cerebral arterial spasm[5].

A case of vasospasm associated with pituitary appolexy ofter head trauma was reported[3]. Pozzati[8] reported a case of pituitary apoplexy complicated by bilateral carotid spasm and cerebral infarction without extrinsic carotid compression in a 15-year-old boy, and mentioned that the early occurrence of vasospasm suggested the participation of powerful vasoactive agents released from the tumor. Vasospasm after transsphenoidal removal of a pituitary adenoma has also been reported[2,9], and it was possible that the free passage of blood from the sella to the basal cisterns was responsible for the subsequent vasospasm and infarction. These cases strongly suggest that vasoactive materials derived from pituitary apoplexy are very hazardous to the cerebral arteries.

The incidence of pituitary apoplexy varies from 1.5 %to 27.7 %[11]. Wakai[11] *et al.* reported an unexpectedly high incidence of pituitary adenoma (16.6 %) involving asymptomatic hemorrhage (7.5 %), and mentioned that any case of major attack with severe symptoms of pituitary apoplexy must be considered as a neurosurgical emergency. Actually, many cases with symptomatic pituitary apoplexy are treated as emergencies, but no case of postoperative vasospasm after emergent operation has been reported in the literature. It may take a definite time for vasoactive materials to be derived from pituitary hemorrhage.

Our patient was treated conservatively for vasculopathy and made a considerable neurological recovery, possibly because the amount of vasoactive material released was small and early treatment was administered. This is the first report of postoperative vasospasm in pituitary adenoma with pituitary apoplexy demonstrated by MRI preoperatively. If pituitary apoplexy had happened with headache one month prior to admission, two months had passed until operation.

We selected a transcranial approach because of active sinusitis, but a transcranial approach may be a contraindication in pituitary adenoma with pituitary apoplexy due to the risky vasoactive materials above mentioned. In our case, it may have been better to perform the operation through a transsphenoidal approach after improvement of the sinusitis.

In conclusion, pituitary adenoma with apoplexy should be treated very carefully, with particular attention paid to the operative approach and procedure. Still more, it is important to consider cerebral vasospasm as a cause of clinical deterioration after pituitary adenoma operation, and to perform early angiographic evaluation and current spasm treatment.

REFERENCES

1) **Shirasaka, A., Ninchoji, T., Uemura, K., Shinohara, Y., Kuwahara, T.** and **Yamazaki, K.** : Delayed vasospasm complicated with the postoperative stage of the meningioma—A case report. Neurol. Surg. **21** : 745-749, 1993.

2) **Goto, T., Sasanuma, J., Koshikaykma, H., Yasuda, T., Goto, H.** and **Watanabe, K.** : Two cases of vasospasm complicated with the postoperative stage of parasellar tumor. Proceeding of Spasm Symposium

3: 188-192, 1988.

3) **Itoyama, Y., Goto, S., Miura, M., Kuratsu, J., Ushio, Y.** and **Matsumoto, T.**: Intracranial arterial vasospasm associated with pituitary apoplexy after head trauma. Neurol. Med. Chir. (Tokyo) **30**: 350-353, 1990.

4) **Mawk, J. R., Ausman, J. I., Erickson, D. L.** and **Maxwell, R. E.**: Vasospasm following transcranial removal of large pituitary adenomas. J. Neurosurg. **50**: 229-232, 1979.

5) **Mawk, J. R.**: Vasospasm after pituitary surgery. J. Neurosurg. (Letter) **58**: 972, 1983.

6) **Ono, S., Misumi, S., Nukui, H.** and **Kawafuchi, J.**: Vasospasm following removal of a large pituitary adenoma by the subfrontal approach. Neurol. Med. Chir. (Tokyo) **21**: 609-614, 1981.

7) **Hyde-Roman, M. D., Roessmann, U.** and **Brodkey, J. S.**: Vasospasm following transsphenoidal tumor removal associated with the arterial changes of oral contraception. Surg. Neurol. **20**: 120-124, 1983.

8) **Pozatti, E., Frank, G., Nasi, M. T.** and **Giuliani, G.**: Pituitary apoplexy, bilateral carotid vasospasm, and cerebral infarction in a 15-year-old boy. Neurosurgery **20**: 56-59, 1987.

9) **Camp, P. E., Paxton, H. D., Buchan, G. C.** and **Gahbauer, H.**: Vasospasm after transsphenoidal hypophysectomy. Neurosurgery **7**: 382-385, 1980.

10) **Krayenbühl, H.**: Beitrag zur Frage des cerebralen angiospastischen insultes. Schweiz. Med. Wochenschr. **90**: 961-965, 1960.

11) **Wakai, S., Fukushima, T., Teramoto, A.** and **Sano, K.**: Pituitary apoplexy: its incidence and clinical significance. J. Neurosurg. **55**: 187-193, 1981.