

# Cerebral Infarction Following Pituitary Apoplexy

## —Case Report—

Hiroyoshi AKUTSU, Shozo NOGUCHI, Takashi TSUNODA, Mamoru SASAKI,  
and Akira MATSUMURA\*

*Department of Neurosurgery, Ibaraki Prefectural Central Hospital, Ibaraki;*

*\*Department of Neurosurgery, Institute of Clinical Medicine, University of Tsukuba,  
Tsukuba, Ibaraki*

### Abstract

A 29-year-old man presented with lethargy, headache, high fever, and visual disturbance. Neurological examination showed mydriatic pupil, ptosis, diminished light reflex, and ophthalmoplegia on the left. Magnetic resonance (MR) imaging showed the typical findings of pituitary apoplexy, and cerebral angiography disclosed mild narrowing of the A<sub>1</sub> segment of the left anterior cerebral artery (ACA). Transsphenoidal tumor resection was performed. Transient severe right hemiparesis occurred directly after the operation. Computed tomography demonstrated cerebral infarction in the territory of the left Heubner's and medial lenticulostriate arteries. Pituitary apoplexy followed by cerebral infarction is very rare. Vasospasm of the perforating arteries of the ACA probably caused the cerebral infarction. Subarachnoid blood or vasoactive agents released from the tumor were the most likely cause of the vasospasm. MR imaging findings of contrast enhancement around the vessels may indicate reactive processes around the vessels.

Key words: pituitary apoplexy, cerebral infarction, vasospasm

### Introduction

Pituitary apoplexy is a well-known clinical syndrome manifesting as headache, meningeal irritation, visual loss, ophthalmoplegia, and changes in consciousness. Cerebral infarction associated with pituitary apoplexy is rare. Here we describe a case of pituitary apoplexy complicated by cerebral infarction.

### Case Report

A 29-year-old man developed high fever and diarrhea, and several hours later suffered sudden onset of headache and visual disturbance. He was lethargic and unable to walk the next day. On admission 2 days later, the patient was drowsy but well-oriented, and could obey verbal commands. Neurological examination revealed hypalgesia in the left forehead, anisocoria (left > right), ptosis,

diminished light reflex, and ophthalmoplegia on the left. His left eye could discern only light. His body temperature was 39°C and his neck was stiff. Blood tests showed the white cell count was 9400 mm<sup>3</sup>, C-reactive protein level was 24.66 mg/dl, and sodium level was 126 mmol/l. The thyroid hormone and prolactin levels were slightly decreased but other hormone levels were normal. Cerebrospinal fluid was not examined.

Computed tomography (CT) showed a large pituitary tumor with suprasellar and lateral extension into the left cavernous sinus. No subarachnoid hemorrhage (SAH) was detected (Fig. 1). Magnetic resonance (MR) imaging indicated that the tumor was partially hyperintense on T<sub>1</sub>-weighted images, suggesting hematoma, with enhancement of the peritumoral brain surface (including the hypothalamus), and around the walls of the A<sub>1</sub> segment of the left anterior cerebral artery (ACA) and the M<sub>1</sub> segment of the left middle cerebral artery (MCA)

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Author's present address: H. Akutsu, M.D., Department of Neurosurgery, Tsukuba Medical Center, Tsukuba, Ibaraki, Japan.

(Fig. 2A, B). T<sub>2</sub>-weighted MR imaging revealed hyperintense areas in the bilateral frontal lobes and hypothalamus suggesting edema (Fig. 2C). Cerebral angiography on the day of admission disclosed mild narrowing of the A<sub>1</sub> segment of the left ACA.

The patient was treated with antibiotics and steroids. His temperature decreased to 37°C, but the consciousness disturbance progressed slightly, his left eye became blind and fixed in the mid-position, and light reflex was lost on the left.

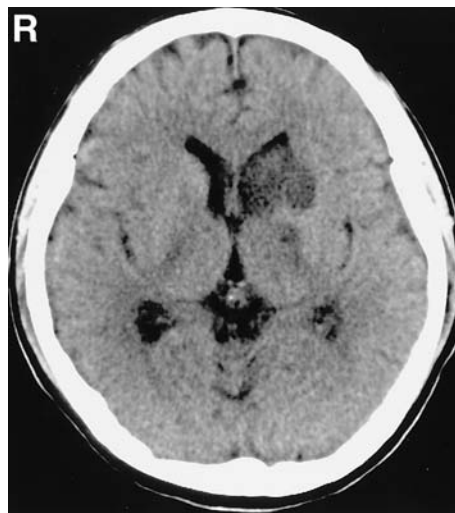
Transsphenoidal excision of the tumor was per-

formed on the day after admission. The sphenoid sinus was filled with hematoma. The tumor was hemorrhagic and necrotic, and was subtotally removed except for a small portion invading the left cavernous sinus. Histological examination of the specimen revealed diffuse necrosis and hemorrhage in the pituitary adenoma, compatible with pituitary apoplexy.

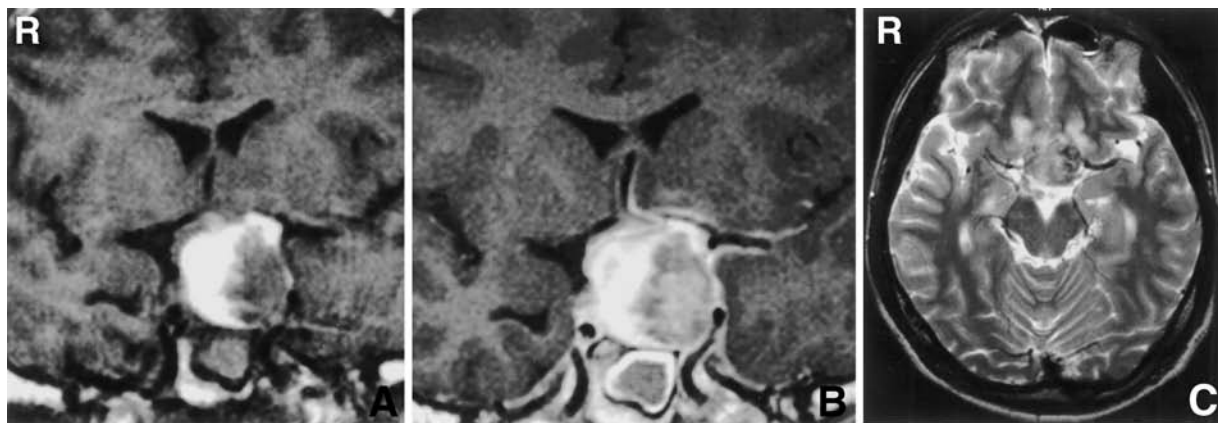
The left pupil became reactive to light but he remained drowsy with severe right hemiparesis directly after the operation. CT immediately after



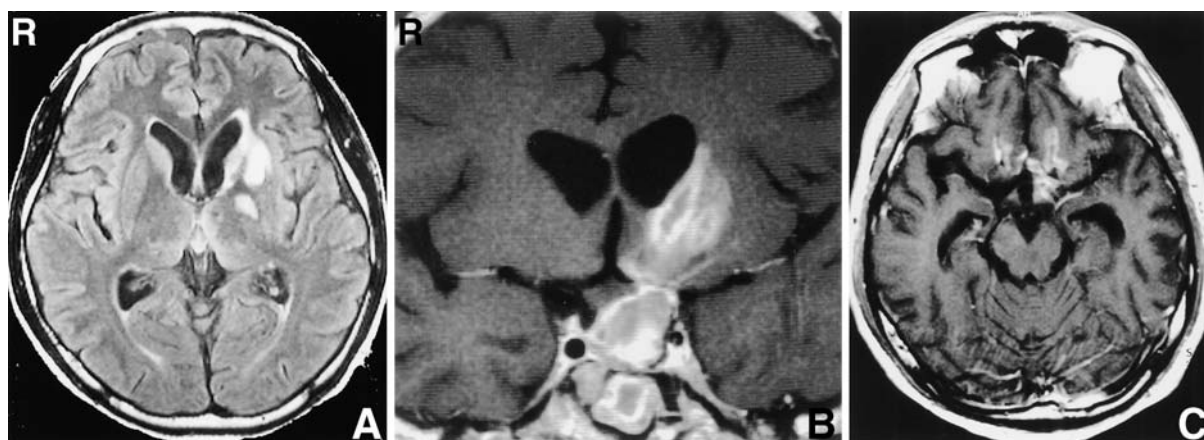
**Fig. 1** Computed tomography scan showing a heterogeneous mass of slightly high density in the suprasellar cistern.



**Fig. 3** Computed tomography scan taken immediately after the operation showing hypodense areas in the territory of the left Heubner's and medial lenticulostriate arteries.



**Fig. 2** Coronal T<sub>1</sub>-weighted magnetic resonance (MR) images (A) and with gadolinium (B) showing the intra- and suprasellar mass and enhancement of the peritumoral brain surface, and the walls of the A<sub>1</sub> segment of the left anterior cerebral artery and the M<sub>1</sub> segment of the left middle cerebral artery. T<sub>2</sub>-weighted MR image showing edema on the surface of the bilateral frontal lobes and hypothalamus (C).



**Fig. 4** Magnetic resonance images taken 1 month after the operation. Fluid-attenuated inversion recovery image showing high intensity areas in the territory of the left Heubner's and medial lenticulostriate arteries (A), and T<sub>1</sub>-weighted images showing contrast enhancement in the territory of the left Heubner's and medial lenticulostriate arteries, and small residual tumor (B), and contrast enhancement on the surface of the bilateral frontal lobes and hypothalamus (C).

the operation demonstrated hypodense areas in the territory of the left Heubner's and medial lenticulostriate arteries (Fig. 3). The right hemiparesis disappeared 3 hours after onset and his level of consciousness gradually improved. He became alert 2 weeks after the operation. His visual acuity recovered completely, but right homonymous hemianopsia persisted. The third, fourth, and sixth cranial nerve pareses resolved. Endocrinologic testing revealed hypoadrenalism, and cortisone replacement therapy was started. MR imaging 1 month after the operation revealed cerebral infarction in the territory of the left Heubner's and medial lenticulostriate arteries, and contrast enhancement in the bilateral frontal lobes, hypothalamus, and areas of cerebral infarction, and a small residual tumor (Fig. 4).

## Discussion

Cerebral ischemia associated with pituitary apoplexy is very rare, with only nine reported cases as summarized in Table 1.<sup>3-5,7,8,11,12</sup> The ischemic events were attributed to cerebral vasospasm in six cases and to mechanical compression by the tumor in three cases. The cerebral infarction was located at the ACA territory in four cases, the MCA territory in two cases, and diffusely in one case. Angiographical stenosis was detected in the internal carotid artery in six cases, in the MCA in two cases, in the ACA in three cases, and in the basilar artery in one case. SAH appeared in only one case. The tumor size was large in all eight described cases and suprasellar

extension was present in all cases. The ischemic event occurred at the day of onset in five cases, 1 day after onset in one case, 5 days after onset in one case, 14 days after onset in one case, and 21 days (postoperatively) after onset in one case.

The etiology of cerebral infarction was difficult to determine in our case because angiography did not show apparent vasospasm, and was performed only at admission, and not when the ischemic event occurred. One possible cause of the cerebral infarction was vasospasm due to subarachnoid blood released from the pituitary apoplexy or intraoperative vessel injury, other chemical substances, or mechanical compression during the operation. However, we thought that mechanical compression during operation was not associated with the ischemia because CT immediately after operation had already detected hypodense areas, which indicated that irreversible ischemic change had occurred between the admission and the beginning of the operation (the operative time was within 3 hours). Other possible causes of cerebral infarction include mechanical compression of the arterial vessels due to the mass effect of the tumor or intraoperative manipulation, and dehydration due to hyperthermia, cerebral salt wasting syndrome, or some other factor. Some cases of pituitary apoplexy were associated with cerebral infarction due to mechanical compression of the arterial vessels caused by the tumor mass effect.<sup>4,8,12</sup> However, in our case, cerebral angiography showed no apparent mechanical compression of intracranial arteries.

The pathophysiology of vasospasm following

**Table 1** Reported cases of cerebral ischemia following pituitary apoplexy

Author (Year)	Age/ Sex	Vascular territory of infarction	Angiographical findings	SAH in CT	Symptom	Tumor size	Days after onset	Mechanism
Rosenbaum et al. (1977) <sup>12)</sup>	77/M	rt MCA	rt ICA occlusion, lt ICA stenosis	n.e.	lt hemiparesis	large	0	compression
Cardoso and Peterson (1983) <sup>3)</sup>	34/F	diffuse	bil ICA, MCA, and ACA stenoses	n.e.	altered consciousness	large	21 (postop.)	vasospasm
	38/M	—	bil ICA and BA stenoses	n.e.	—	n.d.	0	vasospasm
D'Haens et al. (1983) <sup>5)</sup>	43/M	bil ACAs	lt ICA stenosis	—	aphasia, rt hemiparesis	large	0	vasospasm
Pozzati et al. (1987) <sup>11)</sup>	15/M	rt MCA	bil ICA stenoses	—	drowsiness, rt facial palsy	large	0	vasospasm
Clark et al. (1987) <sup>4)</sup>	40/M	lt ACA	rt ICA stenosis, lt ICA occlusion	—	rt hemiplegia, rt facial palsy	large	0	compression
Itoyama et al. (1990) <sup>7)</sup>	45/M	—	lt ACA and MCA stenoses	+	rt hemiparesis	large	14	vasospasm
Lath and Rajshekhar (2001) <sup>8)</sup>	40/M	rt ACA	—	—	lt hemiplegia	large	1	compression
Present case	29/M	lt Heubner's and medial lenticulo- striate arteries	lt ACA mild stenosis	—	drowsiness, lt hemiplegia (transient)	large	5	vasospasm

ACA: anterior cerebral artery, BA: basilar artery, CT: computed tomography, ICA: internal carotid artery, MCA: middle cerebral artery, n.d.: not described, n.e.: not examined, postop.: postoperative, SAH: subarachnoid hemorrhage.

pituitary apoplexy is still unclear, although several hypotheses have been advanced, such as the presence of subarachnoid blood, vasoactive chemical substances released from the tumor or the hypothalamohypophyseal area, hypothalamic damage and dysfunction, and intraoperative manipulation.<sup>3-5,7,8,11-13)</sup> These factors are also possible causes of vasospasm following surgery for pituitary adenoma and other parasellar tumors.<sup>1,2,6,10)</sup> Subarachnoid blood is well known as a possible primary cause of vasospasm after aneurysmal SAH. In our case, SAH may have been involved in the vasospasm although CT did not show SAH, possibly because 3 days had passed since the onset. The other likely cause of vasospasm is vasoactive agent released from the tumor or the hypothalamohypophyseal area. Extracts obtained from the hypothalamus and pituitary stalk induce vasospasm,<sup>13)</sup> so vasospasm following pituitary apoplexy may be caused by the release of vasoactive agents from the tumor or the hypothalamohypophyseal area.<sup>9,11)</sup>

In the present case, MR imaging before and after the operation demonstrated contrast enhancement of the parasellar brain surface and the walls of the left ACA and MCA. Similar MR imaging findings have been described in a case of vasospasm caused by chemical meningitis after the spontaneous rupture of a craniopharyngioma cyst.<sup>14)</sup> Therefore,

the same etiological factor may be involved in the present case. Future findings of this type should be carefully noted in cases of pituitary apoplexy.

Cerebral infarction following pituitary apoplexy is rare, but clinicians should be aware of the possibility as treatment as early as possible is necessary to obtain favorable therapeutic results. Cerebral angiography should be performed in patients who develop neurological deterioration to determine the cause as vasospasm or other factors. MR imaging detection of contrast enhancement around the vessels may be a helpful clue in the diagnosis of reactive processes of the vessels, as in our patient. Early treatment may be also possible if such findings are recognized on the initial MR imaging.

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- Address reprint requests to: H. Akutsu, M.D., Department of Neurosurgery, Tsukuba Medical Center, 1-3-1 Amakubo, Tsukuba, Ibaraki 305-8558, Japan.  
e-mail: akutsuh@zf6.so-net.ne.jp