

Pituitary Apoplexy Causing Internal Carotid Artery Occlusion

—Case Report—

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Abstract

A 50-year-old man presented with pituitary apoplexy resulting in internal carotid artery occlusion manifesting as sudden onset of severe headache, right ptosis, and left hemiparesis, associated with visual impairment. Computed tomography showed a nodular mass, located in the sellar and suprasellar regions with early signs of acute cerebral ischemia. Magnetic resonance (MR) imaging indicated that the mass compressed the bilateral cavernous sinuses, resulting in obliteration of the cavernous portion of the right internal carotid artery. Right middle cerebral artery territory infarction was also found. Conservative therapy with steroids was given in the acute stage and repeated MR imaging showed recanalization of the internal carotid artery with reduction of the tumor size. The tumor was removed through the transsphenoidal approach to obtain a definitive diagnosis in the chronic stage. The histological diagnosis was consistent with non-functioning pituitary adenoma. Eye movement of this patient showed full recovery after the operation. Pituitary apoplexy resulting in internal carotid artery occlusion is rare. Surgical decompression through the transsphenoidal approach is appropriate, but the optimal timing should consider severe disturbance of visual acuity and visual field in the acute stage.

Key words: pituitary apoplexy, internal carotid artery occlusion, pituitary adenoma

Introduction

Pituitary apoplexy is a clinical syndrome characterized by sudden onset of headache, visual impairment, and ophthalmoplegia caused by enlargement of pituitary adenoma. The incidence of apoplexy in patients with pituitary adenomas is 0.6–10%.²⁾ However, cerebral infarction associated with pituitary apoplexy is very rare. Here, we report a case of pituitary apoplexy with development of massive cerebral infarction due to internal carotid artery occlusion caused by the sudden enlargement of a pituitary tumor.

Case Report

A 50-year-old man presented with acute onset of severe headache, which was especially retro-orbital in location, right ptosis, and left hemiparesis. He was referred to another hospital 6 hours after the onset and neurological examination showed right third cranial nerve palsy with ptosis. Motor strength was grade 2 on the left. Computed tomography (CT) showed an enlarged pituitary fossa consisting of an area of heterogeneous high density with loss of cortical-subcortical discrimination in the right middle

cerebral artery territory and decreasing volume of the sylvian fissure in the right insular region (Fig. 1A). Magnetic resonance (MR) imaging revealed an intra- and suprasellar lesion measuring $3 \times 3 \times 2$ cm without enhancement which caused compression of the optic chiasma and obliteration of the cavernous portion of the right internal carotid artery (Fig. 1B, C). MR imaging also showed focal high intensity around the cavernous portion of the internal carotid artery, suggesting hemorrhage. MR angiography and right common carotid angiography showed right internal carotid artery occlusion and diffusion-weighted MR imaging demonstrated massive cerebral infarction of the right middle cerebral artery territory (Fig. 2).

The patient was initially treated with infusion of steroids and free radical scavenger because CT had already showed early signs of acute cerebral ischemia and visual disturbance was not severe. His level of consciousness improved and headache was relieved by these treatments, but left hemiparesis became gradually worse. He was transferred to our hospital 24 hours after the onset.

On admission, his neurological signs were left hemiparesia with right ophthalmoplegia. Fortunately, visual acuity and the visual field were not remarkably decreased, so we continued intravenous infusion of the free radical scavenger and steroids for massive cerebral infarction. MR imag-

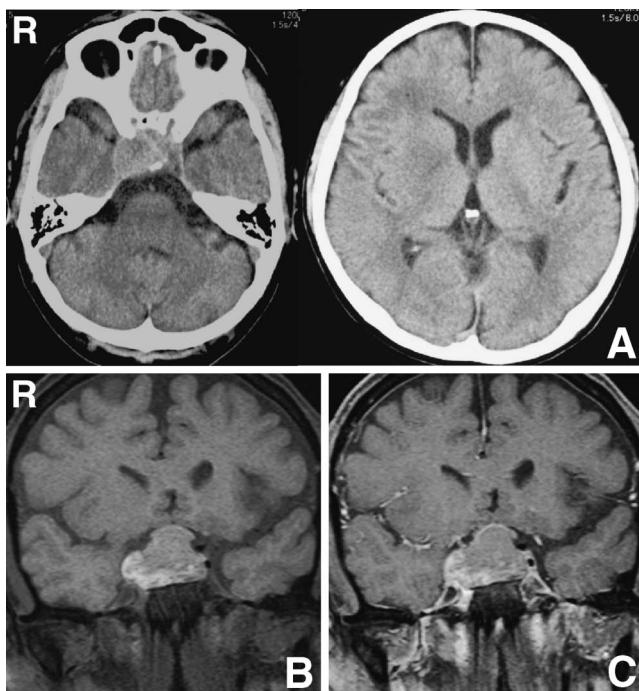


Fig. 1 Computed tomography scans (A) showing a high density lesion in the pituitary fossa with loss of cortical-subcortical discrimination and decreasing volume of the sylvian fissure in the right insular region 6 hours after the onset. Coronal T₁-weighted magnetic resonance images without (B) and with (C) gadolinium showing focal high intensity around the cavernous portion of the internal carotid artery, suggesting hemorrhage.

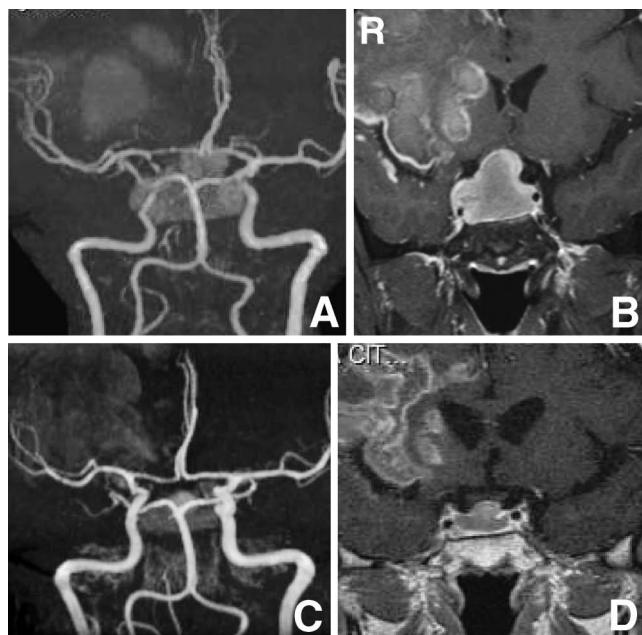


Fig. 3 T₁-weighted magnetic resonance (MR) images with gadolinium (B, D) showing homogeneous enhancement in the pituitary fossa and suprasellar lesion and MR angiograms (A, C) revealing partial recanalization of the right internal carotid artery one week after the onset (A, B), and the size of the tumor was decreased and the internal carotid artery was completely patent one month later (C, D).

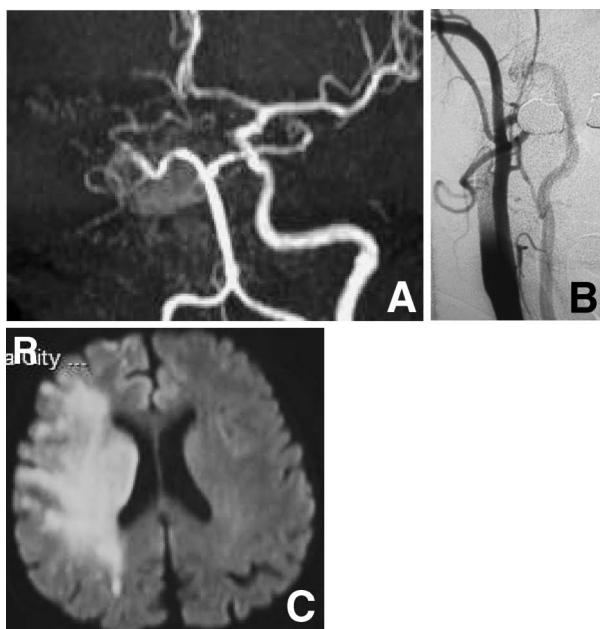


Fig. 2 Magnetic resonance (MR) angiogram (A) and right common carotid angiogram (B) showing the right internal carotid artery was occluded 6 hours after the onset and diffusion-weighted MR image (C) showing high signal intensity in the right middle cerebral artery territory.

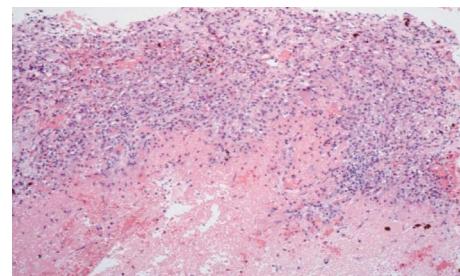


Fig. 4 Photomicrograph showing infarction with necrotic cells. Hematoxylin and eosin stain, $\times 100$.

ing was performed to evaluate recanalization of the internal carotid artery and hemorrhagic infarction in the right cerebral hemisphere one week later (Fig. 3A, B). The tumor was reduced in size and the right internal carotid artery was well visualized as the normal shape one month later (Fig. 3C, D). Fortunately, no neurological deterioration was found during this acute stage. The patient underwent tumor removal through the transsphenoidal approach in the chronic stage to obtain a definitive diagnosis.

After cutting the dura mater of the sella floor, no bleeding occurred. The coagulated blood, not serum, was evacuated by microcurettage. After subtotal hematoma removal, pulsation of the diaphragm was confirmed. The

postoperative course was uneventful. Right ptosis and total ophthalmoplegia were improved 2 weeks later. Histological examination revealed pituitary adenoma with some infarction (Fig. 4).

Discussion

The predisposing factors of pituitary apoplexy have been frequently described, including pituitary irradiation, minor head trauma, hormone therapy, pregnancy, diabetes mellitus, cerebral angiography, anticoagulant medication, dynamic study of the pituitary gland, hemodialysis, and various types of surgery, including lumbar laminectomy and cardiac surgery. Intratumoral hemorrhage and hemorrhagic infarction in pituitary tumors are known to be the main causes of pituitary apoplexy. However, the mechanism of infarction in pituitary tumors has still not been verified. Pituitary apoplexy results in a rapid increase in intrasellar contents, with consequent sudden rise in intrasellar pressure (ISP). ISP was markedly elevated in patients with pituitary apoplexy, with a medial value of 47 mmHg.¹⁵⁾ Normal ISP is not known, but is believed to be similar to or less than the normal intracranial pressure of 7–15 mmHg.⁶⁾ The sharp increase in ISP leads to disturbance of anterior pituitary cell viability, which could increase the risk of ischemic necrosis and limit the potential for recovery of pituitary function. In our patient, MR imaging showed focal high intensity around the cavernous portion of the internal carotid artery, suggesting hemorrhagic infarction and not massive bleeding. Therefore, we judged that the pituitary apoplexy was caused by hemorrhagic infarction and ISP was suddenly elevated to cause occlusion of cavernous portion of the internal carotid artery.

The mechanism of cerebral ischemia has been suggested to involve either mechanical obstruction of the internal carotid artery by the enlarging mass or vasospasm. Pituitary apoplexy may occur first leading to internal carotid artery occlusion.^{5,14)} Vasospasm may be caused by extravasation of blood from the pituitary tumor into the subarachnoid space or the vasoactive agent released from the pituitary tumor into the hypophyseal area.¹⁰⁾ We speculate that if the vasoactive agent is the main factor to cause acute internal carotid artery occlusion, the bilateral inter-

nal carotid arteries should be occluded. On the other hand, internal carotid artery occlusion was the primary event leading to hemorrhagic infarction in pituitary macroadenoma.⁹⁾ Our patient did not have any past history of risk factors such as hypertension and diabetes mellitus causing arteriosclerotic change in the internal carotid artery. Therefore, we speculate that the internal carotid artery was easy to displace and was occluded by the acute enlarged suprasellar mass infiltrating directly to the cavernous sinus. Massive cerebral infarction following mechanical compression of the internal carotid artery is known^{1,3,7,8,11–14)} (Table 1). The internal carotid artery was occluded in the cavernous sinus or supraclinoid portion by the enlarged tumor in most cases.

The optimal timing for the surgical treatment of the tumor should be considered. Surgical decompression may or may not restore flow in the occluded vessels. Surgical decompression in the acute stage via the transsphenoidal route was performed in one case,⁷⁾ but the patient died of a large cerebral infarction in the right internal carotid artery territory. Early decompression surgery is often advocated for pituitary apoplexy.⁴⁾ However, pituitary apoplexy with massive cerebral infarction, as in the present case, is associated with high mortality due to early decompression of the tumor for two reasons, risk of hemorrhagic infarction due to recanalization of the obstructed internal carotid artery by removing the pituitary tumor, and hemodynamic changes in the cerebral hemisphere due to the occluded internal carotid artery during the acute period.

Our patient did not complain of significant visual acuity or visual field disturbance. Considering these risk factors and the visual function of the patient, we judged that decompression surgery in the acute stage could be high risk. We planned to perform tumor removal and ligation of the internal carotid artery at the cervical portion in the chronic stage. However, recanalization of obstructed internal carotid artery occurred within one week from the onset, and caused massive hemorrhagic infarction in the cerebral hemisphere. Therefore, we performed the transsphenoidal surgery without perioperative complications in the chronic stage so that the cerebral circulation would be stable.

Pituitary apoplexy causing massive cerebral infarction

Table 1 Reported cases of mechanical compression of major vessels following pituitary apoplexy

Author (Year)	Age (yrs)/Sex	Symptom	Radiological finding	Tumor removal	Outcome
Schnitker and Lehnert (1952) ¹³⁾	65/M	lt hemiplegia	ND	—	death
Sakalas et al. (1973) ¹²⁾	9/M	drowsy, loss of vision	infarction	OS	good
Rosenbaum et al. (1977) ¹¹⁾	77/M	stupor, lt hemiparesis	infarction	OS	death
Majchrzak et al. (1983) ⁸⁾	29/M	confused, lt hemiparesis	infarction	OS	good
Bernstein et al. (1984) ¹⁾	48/M	confused, lt hemiparesis	ND	TSA	good
Clark et al. (1987) ⁹⁾	40/M	dysphagia, rt hemiplegia	infarction	—	good
Lath and Rajshekhar (2001) ⁷⁾	40/M	lt hemiplegia	infarction	TSA	death
Yang et al. (2008) ¹⁴⁾	43/M	lt hemiplegia	infarction	TSA	good
Present case	50/M	lt hemiplegia	infarction	TSA	good

lt: left, M: male, ND: not described, OS: open surgery, rt: right, TSA: transsphenoidal approach.

due to internal carotid artery occlusion by sudden enlargement of a pituitary tumor is rare. Mechanical obstruction of the internal carotid artery by the acute enlarging mass is likely. Surgical decompression through the trans-sphenoidal approach is appropriate. However, we should take into consideration the optimal timing of surgical resection if patients do not present with severe disturbance of visual acuity and visual field in the acute stage.

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