Pituitary Apoplexy Caused by Ruptured Internal Carotid Artery Aneurysm

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Background and Purpose—We report the first case of pituitary apoplexy caused by the rupture of an intracavernous carotid artery aneurysm embedded in a pituitary adenoma.

Case Description—A 46-year-old man presented with clinical and CT findings typical of pituitary apoplexy. MRI showed an unusual flow-void protrusion into the intratumoral hematoma, which, however, was not diagnosed as a ruptured aneurysm until severe intraoperative bleeding occurred. Angiography after surgery revealed an intracavernous carotid artery aneurysm.

Conclusions—The possible association of adenoma and aneurysmal rupture should be kept in mind when assessing any case of pituitary apoplexy. (Stroke. 2001;32:567-569.)

Key Words: adenoma • cerebral aneurysm • hematoma • pituitary apoplexy • pituitary neoplasms

Pituitary apoplexy usually results from hemorrhage into a pituitary adenoma either spontaneously or after various precipitating events. However, the exact mechanism of pituitary apoplexy is not always clear. Recently, Provenzale et al reported a case in which pituitary apoplexy occurred probably secondary to internal carotid artery (ICA) dissection. We present a case of pituitary apoplexy caused by an unsuspected aneurysmal bleeding into a pituitary adenoma.

Case Report

A 46-year-old man presented with a sudden onset of headache, vomiting, and visual impairment. The patient had a 1-year period of decreased libido and a 2-month period of mild visual impairment with no diplopia.

On admission, the patient was alert and cooperative. Visual acuity had severely decreased when he attempted to count fingers with 1 eye covered. The visual fields in both eyes were preserved on the confrontation test to finger movement in all quadrants. He exhibited a complete right ptosis, a fixed dilated right pupil, and loss of all extraocular movements of the right eye. On the left side, the abducens nerve function was completely impaired, but the function of oculomotor and trochlear nerves was preserved. There was severe pain on the right side of the forehead and the nasal bridge. The prolactin level was 2200 mg/L (normal range, 9.7). A skull radiography revealed an enlargement of the sella turcica and erosion at the floor of the sella turcica. There was abnormal signal intensity consistent with acute hemorrhage in the tumor. Urgent transsphenoidal surgery was performed to decompress the optic chiasm. After removing the tumor in the sphenoid sinus, the tumor and hematoma in the sella turcica were pulsating. When removal of the hematoma was begun, the hematoma protruded, and profuse arterial bleeding suddenly occurred. Bleeding could not be controlled by the largest sized suction device and was finally stopped by packing with Surgicel (Ethicon Inc) and Biobond (Welfide Co). The total volume of bleeding was >3 L. The histopathologic diagnosis was a prolactinoma.

The angiography performed immediately after the surgery showed a right intracavernous carotid artery aneurysm (Figure 3A and 3B). After an occlusion test using a balloon catheter, endovascular occlusion of the right ICA was performed by use of platinum coils from the distal portion to the proximal portion of the aneurysmal neck (Figure 3C and 3D). An additional latex balloon was detached more proximally in the right ICA.

In a postoperative reevaluation, the preoperative MRI revealed that the right intracavernous carotid artery protruded a small signal void into the intratumoral hematoma, which might indicate a ruptured aneurysm (Figure 2D and 2E). Fortunately, the cranial nerve dysfunctions completely resolved. The prolactin level remained high at 940 mg/L. The
dynamic pituitary function tests revealed a deficiency of growth hormone and gonadotropin secretion. The persisting polyuria was diagnosed as pituitary diabetes insipidus. The patient was discharged receiving bromocriptine mesylate and desmopressin acetate, and he returned to work 3 months after the surgery.

Discussion

The incidence of aneurysms and pituitary apoplexy in patients with pituitary adenoma has been reported as 3.7% to 7.4%4–6 and 0.6% to 12.3%,1,2 respectively. However, the spontaneous rupture of an aneurysm associated with a pituitary tumor4,7 and pituitary apoplexy associated with an unruptured aneurysm8 are very rare. Although several cases of unruptured intrasellar aneurysms contiguous to pituitary adenomas have been reported,5,6,8 the rupture of an aneurysm embedded in a pituitary tumor is rarer9: a review of the literature revealed only 1 previous case,9 in which the rupture of an intratumoral aneurysm caused fatal epistaxis rather than pituitary apoplexy. On the other hand, 2 cases in which a parasellar aneurysm ruptured and mimicked pituitary apoplexy have been reported10,11: one was associated with an endocrine-active adenoma,11 and the other had no coexisting adenoma.10 The clinical presentation in the present case was unusual in that the rupture of an aneurysm caused a hemorrhage into a pituitary adenoma with no subarachnoid hemorrhage, resulting in an acute clinical syndrome of pituitary apoplexy. To our knowledge, this is the first report of a pituitary apoplexy caused by aneurysmal bleeding into a pituitary adenoma.

Whether the pituitary adenoma contributes to aneurysm formation is interesting. The incidence of coexisting aneu-
Aneurysms and pituitary adenomas have been reported to be significantly higher than that found with other brain tumors. Although aneurysms are associated with all types of pituitary tumors, the frequency of this association with growth hormone–secreting adenomas is highest. Moreover, most coexisting aneurysms are from adjacent arteries and are located close to the pituitary tumor. Thus, the following mechanisms of aneurysm formation associated with pituitary adenomas have been proposed: local circulatory mechanisms, endocrinological mechanisms, mechanical effects, and/or direct invasion. In only one series by Pant et al was this association independent of hormone secretion, size, invasive nature of the tumor, and encasement of the vessels by the tumor; therefore, it was considered to be a simple coincidence. The specific causal relationship between prolactinomas and aneurysms is thus far unknown. In the present case, the development of the aneurysm was most likely due to the hemodynamic stress induced by increased blood supply to the tumor, because the tumor was large with no carotid encasement, and the aneurysm was oriented toward the tumor. The tumor might have invaded, weakened the aneurysmal wall, and caused the growth and rupture of the aneurysm into the tumor.

In the MRI era, routine angiography is probably not indicated before transsphenoidal surgery, inasmuch as MRI usually has sufficient resolution to rule out an associated aneurysm. In the present case, in retrospect, we should have suspected that the presence of an unusual flow-void protrusion suggested a vascular component from the ICA. If a pituitary adenoma is present, the possibility of a coincidental aneurysm should always be considered. Our case proves that pituitary apoplexy can be due to the rupture of an adjacent ICA aneurysm into the tumor. This association should be kept in mind when evaluating any case of pituitary apoplexy. Careful MRI interpretation is required with preoperative carotid angiography to follow, if an aneurysm is suspected.

References
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