

# Vasospasm following transcranial removal of large pituitary adenomas

## Report of three cases

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✓ The authors report three cases of cerebral vascular spasm following transfrontal removal of large pituitary neoplasms. One patient awakened from surgery hemiparetic, but has since recovered. A second patient developed hemiparesis with confusion on the fifth postoperative day and eventually died. The third patient developed hemiplegia on the tenth postoperative day, but recovered completely. Vascular spasm was documented angiographically in all three cases. Possible mechanisms underlying this unusual complication are discussed.

**KEY WORDS** • pituitary neoplasm • vasospasm • hypophysectomy

THE past 5 years have seen increasing popularity of the transsphenoidal approach to pituitary neoplasms. As endocrinological and neuroradiological sophistication have increased, the number of patients presenting with large pituitary adenomas has decreased. There is general agreement that transsphenoidal surgery is the safest and simplest alternative for microadenomas or for neoplasms of modest size.<sup>2</sup> Huge pituitary tumors with significant suprasellar extension, on the other hand, are probably best managed by the traditional transcranial, subfrontal approach. In the last 4 years, we have seen three patients who developed cerebral arterial spasm after transcranial resection of large chromophobe adenomas. We are unable to find reports of this unusual complication elsewhere in the English literature.

### Case Reports

#### Case 1

This 15-year-old girl was admitted to the hospital for evaluation of headaches, recent weight gain, and primary amenorrhea. She denied visual loss.

**Examination.** The patient was markedly obese and displayed an inappropriate affect. There was mod-

erate truncal ataxia. A complete left homonymous hemianopsia was demonstrated. Routine chemistries and a complete blood count (CBC) were normal. Serum cortisol, 17-ketosteroids, follicle-stimulating hormone (FSH), and growth hormone (HGH) were all normal. Although the thyroid-stimulating hormone (TSH) level was normal, the thyroxine ( $T_4$ ) was low. A nuclear brain scan demonstrated a large suprasellar mass. A right carotid angiogram showed lateral displacement of the internal carotid artery and stretching of the anterior cerebral artery (Fig. 1 left).

**Operation.** A right frontal craniotomy was performed. A large, soft tumor was found arising from the sella, extending forward into the superior orbital fissure, posteriorly onto the clivus, and superiorly into the region of the hypothalamus. A gross total removal was accomplished, but one tiny branch of the middle cerebral artery was encased by tumor and had to be sacrificed. No vascular spasm was evident at the time of closure. Pathological examination of the specimen demonstrated a chromophobe adenoma with scattered eosinophilic cells.

**Postoperative Course.** The patient awakened promptly, but she had a dense left hemiparesis and a right third nerve palsy. She remained alert for the first

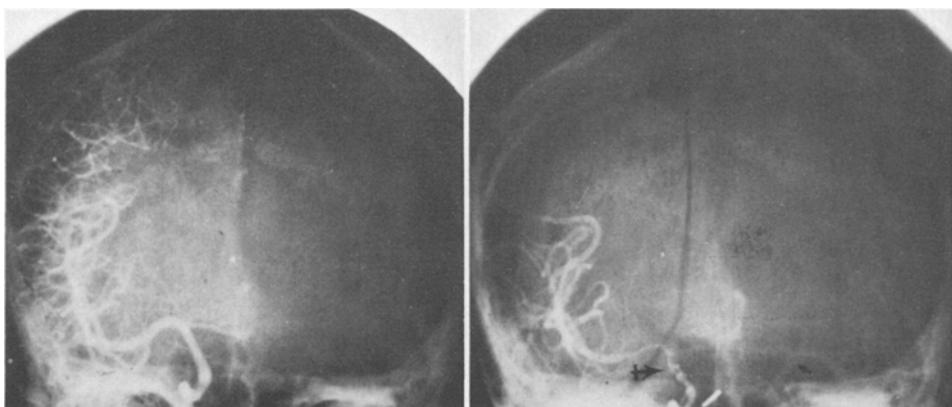


FIG. 1. Case 1. *Left:* Preoperative angiogram showing lateral displacement of the internal carotid artery and stretching of anterior cerebral artery. *Right:* Postoperative angiogram showing moderate spasm of proximal anterior and middle cerebral arteries (*crossed arrow*); in spite of this, there is spontaneous crossover of the contrast material (*arrow*).

4 postoperative days, but then developed lethargy and diabetes insipidus. A right carotid angiogram performed on the sixth postoperative day showed marked vascular spasm (Fig. 1 *right*).

The patient underwent radiation therapy. Her left hemiparesis has gradually improved. The third nerve palsy resolved. Although she continues to have a mild affectual disturbance, there is no evidence of tumor recurrence after 4 years.

#### Case 2

This 42-year-old man presented with a 15-year history of headache. Progressive loss of vision in the left eye had been ignored; disturbance of vision in the right eye caused the patient to seek help. The patient also complained of a markedly decreased libido.

**Examination.** The left eye was blind and there was a temporal field cut in the right eye. Both discs were

pale. The remainder of the examination was normal. Serum chemistries, a CBC, urinalysis, thyroid index, triiodothyronine ( $T_3$ ) and  $T_4$ , HGH, cortisol, and testosterone levels were all normal. Skull films showed a normal-sized sella with some thinning of the dorsum. A computerized tomographic (CT) scan revealed a large suprasellar mass. Carotid angiography (Fig. 2 *left*) demonstrated a large, relatively avascular suprasellar mass lesion.

**Operation.** A right frontal craniotomy was carried out. There was minimal retraction of the brain. A large, pink suprasellar neoplasm was encountered beneath the chiasm and both optic nerves. The tumor was quite soft and was removed with minimal bleeding. Gelfoam was placed into the tumor bed. The neuropathologist interpreted the tissue as typical chromophobe adenoma.

**Postoperative Course.** The patient awakened promptly without neurological deficit and without any

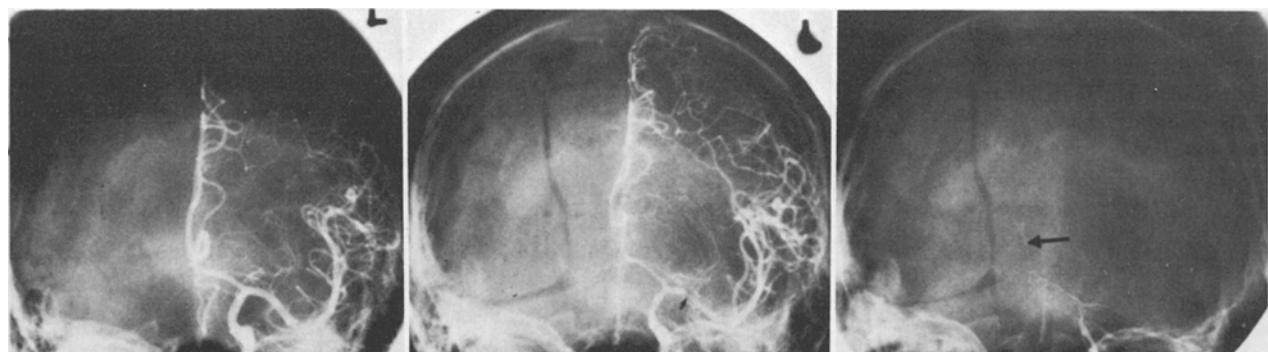


FIG. 2. Case 2. *Left:* Preoperative angiogram showing superior displacement of the anterior cerebral artery. *Center:* First postoperative angiogram demonstrates spasm in the proximal middle cerebral artery (*arrow*). *Right:* Second postoperative angiogram shows extremely severe spasm and displacement of the anterior cerebral artery (*arrow*).

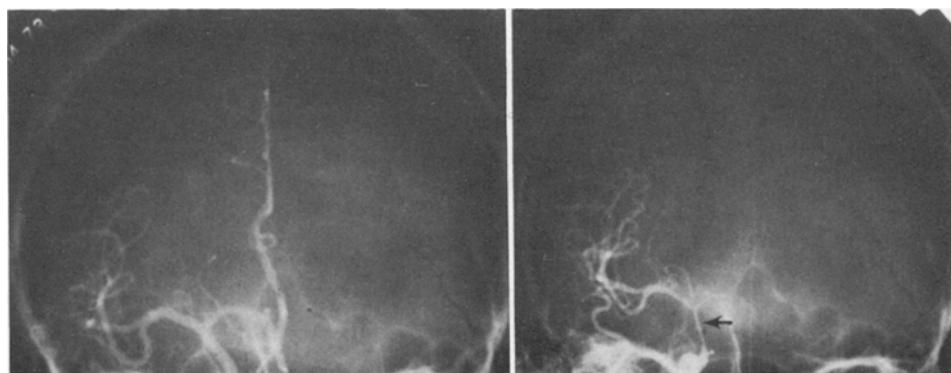


FIG. 3. Case 3. *Left:* Preoperative angiogram showing lateral displacement of the internal carotid artery by an avascular suprasellar mass. *Right:* Angiogram done on the tenth postoperative day shows marked spasm of the internal carotid artery (arrow) extending into the proximal middle and anterior cerebral arteries.

worsening of vision. On the third postoperative day he became slightly confused, but this was attributed to mild hyponatremia. This state persisted until the fifth postoperative day, when he developed a right hemiparesis and became obtunded. A left carotid angiogram demonstrated spasm of the anterior and middle cerebral arteries (Fig. 2 center). He was treated with dexamethasone, but continued to deteriorate. He was re-explored on the eighth postoperative day. The right frontal lobe was tremendously swollen and softened. There was no significant clot in the tumor bed. He continued to deteriorate and died on the eleventh postoperative day. An angiogram done just prior to his death demonstrated intense vascular spasm with flash-filling of the intracranial circulation (Fig. 2 right).

### Case 3

This 23-year-old man was admitted to the hospital because of rapidly progressive visual loss. He had first noted blurring of vision in the left eye 3 months before admission. He denied headaches. He complained of a recent decrease in libido, but stated that sexual function was intact.

**Examination.** The patient was very obese. Visual acuity was 20/30 on the right and worse than 20/200 on the left. There was a bitemporal field deficit. His examination was otherwise normal. A CBC revealed a hypochromic, microcytic anemia. The peripheral white count was 14,000, with numerous immature forms. Although the TSH and  $T_3$  resin uptake were normal, the  $T_4$  was low. Determinations of FSH, LH, prolactin, testosterone, and cortisol were all normal. Skull x-ray films showed a doubled-floored sella. A CT scan showed a huge suprasellar mass largely obliterating the third ventricle, and spilling into both middle fossae and along the upper clivus. An

angiogram demonstrated displacement of the anterior cerebral and internal carotid arteries by an avascular mass (Fig. 3 left).

**Operation.** A right frontotemporal craniotomy was performed. A large, soft, partially necrotic tumor was encountered beneath the chiasm. It was subtotaly resected with minimal retraction of the brain. Gelfoam was left in the tumor bed. The pathological diagnosis was chromophobe adenoma.

**Postoperative Course.** The patient did well except for the development of diabetes insipidus, which was easily controlled with pitressin in oil. On the tenth day after surgery, over a period of about 2 hours, the patient developed a dense left hemiparesis. An emergency angiogram demonstrated severe spasm of the right middle and anterior cerebral arteries (Fig. 3 right). He was treated with dexamethasone. Over the next 2 weeks, his hemiparesis cleared completely. He subsequently underwent radiotherapy and is now doing well without clinical or radiographic evidence of tumor recurrence, 22 months after his surgery.

### Discussion

We are intrigued by this unusual complication of pituitary surgery. Although we have been unable to find mention of any similar cases in the English literature, a comparable case was reported by Krayenbühl in 1960.<sup>1</sup> His patient, a 40-year-old man with an intrasellar chromophobe tumor, did well during the first 4 postoperative days. Sudden onset of hemiparesis prompted angiography, which demonstrated marked vasospasm. The patient was treated with intravenous papaverine and recovered. Krayenbühl thought that the precipitating event in his case was postoperative hypotension.

In our Case 1, severe neurological deficit was apparent immediately after surgery; this deficit was likely due to destruction of the small perforating

vessel which arose from the middle cerebral artery. It is possible that her further deterioration was due to the observed arterial spasm. Our Cases 2 and 3 were well after their surgery, and did not develop focal neurological deficit until 5 and 10 days later, respectively. We believe that their clinical deterioration was directly attributable to cerebral vasospasm.

Several hypotheses can be advanced to explain the occurrence of vasospasm after pituitary surgery. Simple touching or stroking of the cerebral vessels is known to produce transient spasm. While all three of our patients did have bulky adenomas, the operating surgeons in each case were impressed with the relative ease of tumor removal. A simple mechanical explanation is inconsistent with the diffuse and progressive character of spasm seen in Case 2, and does not explain the time-course of clinical events in Cases 2 and 3.

The possibility that blood in the subarachnoid space produced the observed phenomenon cannot be discounted. If this were true this complication should occur with much greater frequency. Only postoperative angiography in symptomatic patients would reveal this, and such a situation is infrequent in our experience. The presence of Gelfoam in the tumor bed might relate to the development of spasm but this has not been reported.

Wilson and Feild<sup>3</sup> were able to produce vasospasm in dogs using extracts obtained from the hypothalamus and pituitary stalk of pigs. Of interest is their observation that hypothalamic extract which was incubated at 37° C for 4 days was more effective in producing arterial spasm than nonincubated (fresh) extract. If spasm in our clinical series were due to the same agents, then delayed inception of spasm might be predicted. This was indeed observed in two of our cases.

Thus, the following hypothesis seems most attractive to us: Vasoactive materials are liberated from the

tumor, either at the time of surgery, or later, after portions of the tumor have undergone necrosis. These chemical substances might then diffuse into the basal subarachnoid spaces and interact with blood vessel walls in such a way as to produce vascular spasm.

### Summary

The authors present three cases of arterial spasm which occurred after transcranial removal of chromophobe adenomas. One case resulted in death, another patient developed hemiparesis which cleared incompletely, and the remaining patient manifested a transient hemiplegia. Vasospasm was documented in all three cases by angiography. Liberation of chemical agents from the tumor bed into the basal subarachnoid space might produce spasm such as we have observed.

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