CASE REPORT
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Vasospasm of Proximal Internal Carotid Artery Following Transcranial Removal of a Pituitary Adenoma

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We report a case of proximal internal carotid arterial spasm following pterional removal of pituitary adenoma. We discuss the possible mechanism of vasospasm associated with tumor resection.

KEY WORDS : Pituitary adenoma · Vasospasm · Transcranial approach.

Introduction

Cerebral vasospasm is well known to occur after various events that cause subarachnoid hemorrhage, such as aneurysmal hemorrhage and neurotrauma[2]. Vasospasm after resection of skull base tumors is rare. Review of the literature demonstrates that most of vasospasms are associated with pituitary adenomas treated by a transcranial approach[1,4-6].

Although there is no consensus concerning its exact etiology, accumulation of blood in the subarachnoid basal cisterns has been postulated to play a major role in vasospasm[1,2,5,6]. But in our case, spasm of cervical, petrous and cavernous ICA proximal to pituitary tumor may provide other possible mechanism of vasospasm.

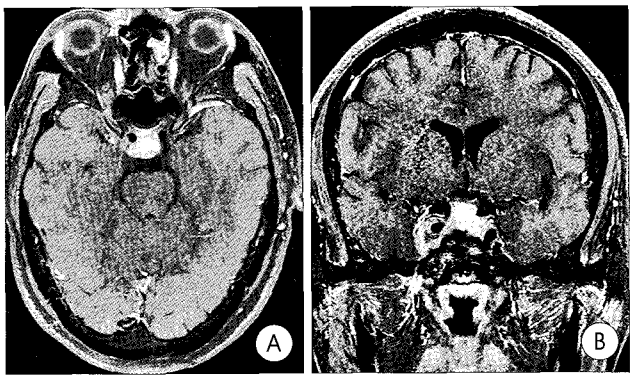

Fig. 1. Preoperative images. T1 weighted gadolinium enhanced axial (A) and coronal (B) images show tumor mass in sellar, suprasellar region and right cavernous sinus. The tumor encases internal carotid artery in the cavernous sinus.

Case Report

A 45-year-old man presented with right visual disturbance and ocular pain for 3 months. The adenohypophyesal hormones were normal. Magnetic resonance imaging(MRI) demonstrated suprasellar and right cavernous mass impinging on the right optic nerve with encasement of right internal carotid artery (Fig. 1). Magnetic resonance angiography(MRA) showed no abnormality (Fig. 2).

The patient underwent transcranial tumor removal. The tumor was in sellar and suprasellar areas and involved right c-

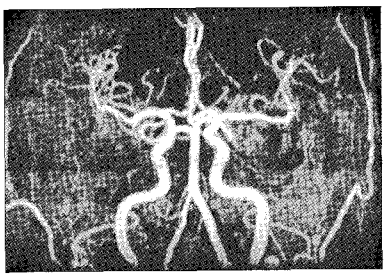

Fig. 2. Magnetic resonance angiography shows no vascular abnormality.

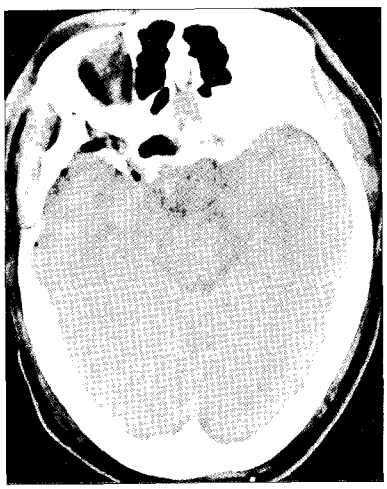

Fig. 3. Postoperative image computerized tomography demonstrates scanty subarachnoid hemorrhage in the basal cistern.

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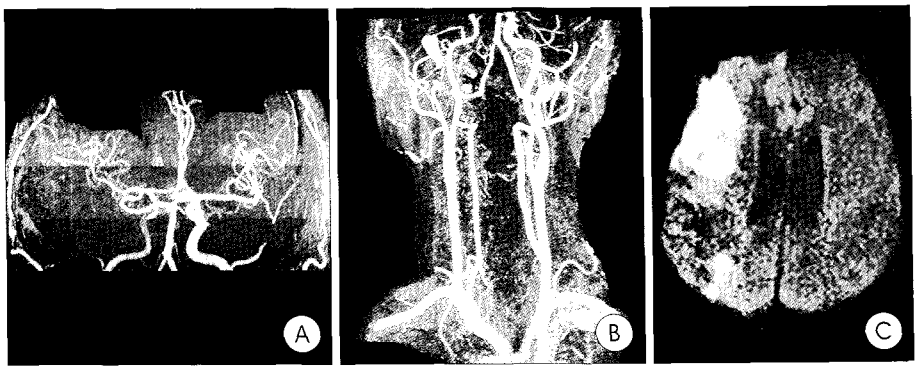

Fig. 4. On postoperative 3 days, Brain (A) and neck (B) shows vasospasm in proximal (cervical, petrous, and cavernous) internal carotid artery. C : Diffusion magnetic resonance image shows high signal intensity on right frontal cortex.

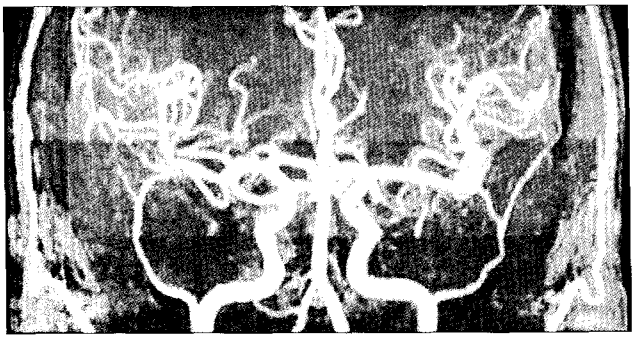

Fig. 5. On postoperative 14 days (at discharge), magnetic resonance angiography shows resolution of internal carotid arterial vasospasm.

avernous sinus. We performed optic nerve decompression by removing the supresellar mass. We did not open the cavernous sinus. The tumor was somewhat hemorrhagic, but bleeding was easily controlled. No vascular spasm was evident at the time of closure. Pathological diagnosis of the specimen was nonfunctioning pituitary adenoma. The patient awakened promptly. Initial postoperative neurological condition was not changed from his preoperative status except subjective improvement of right visual acuity. Immediate postoperative CT scan revealed residual cavernous sinus mass and scanty hemorrhage in basal cisterns (Fig. 3). On the third postoperative day, he suddenly developed left arm weakness (Grade II). CT scan failed to demonstrate any change compared with initial postoperative CT. MRA and diffusion MRI showed spasm of right proximal (cervical, petrous and cavernous) ICA and high signal intensity in right frontal area respectively (Fig. 4). Active hypertensive hypervolemic hemodilution therapy was initiated immediately. Five days later, arm weakness was improved (Grade IV). On the postoperative 14th day, followup MRA revealed resolution of arterial spasm (Fig. 5).

Discussion

Cerebral vasospasm occurs most often in the setting of aneurysmal subarachnoid hemorrhage. Significant morbidity is associated with clinical vasospasm. Therefore, cerebral vasospasm remains one of the most difficult conditions confronted by neurosurgeons. However, both exact pathophysiology and its treatment remain unclear[10]. Although much less frequent than aneurysmal SAH, symptomatic vasospasm may occur in association with head trauma, infection or tumor surgery[10]. Bejjani et al suggested that large tumor, increased tumor vascularity, prolonged operation time, and preoperative vessel encasement and narrowing were significantly correlated with a higher incidence of postoperative vasospasm[2]. Vasospasm occurred often after resection of anterior skull base tumors than posterior skull base tumors. Among them pituitary adenomas were the most frequent[1,5,9].

Several hypotheses may be suggested explaining the occurrence of vasospasm after pituitary surgery. First, blood spillage in the basal cistern is a potential offender[2]. Zabramski et al have demonstrated that there is direct relationship between the amount of blood injected in the subarachnoid cistern and severity of the vasospasm[13]. Second, manipulation or direct damage of the blood vessels is anther factor. As is often the case, simple touching or stroking of cerebral vessel is known to produce transient spasm. However, if the mechanical trauma is primary cause of vasospasm, one would expect the resultant vasospasm to occur immediately after surgery, not after a delay of several days[3]. Third, hypothalamic dysfunction was also cited in the literature as an etiologic factor of vasospasm. In vivo experiments demonstrate hypothalamic extracts produce diffuse vasospasm[12]. Fourth, vasoactive materials are liberated from the tumor, either at the time of surgery, or later, after portion of the tumor have undergone necrosis. These chemical substances might then diffuse into basal subarachnoid space, and interact with blood vessel wall in basal cistern and produce vasospasm[9]. Fifth, the following hypothesis seems most attractive to us : An imbalance of vascular tone seems to contribute to vasospasm.

The physiologic stress of surgery may increase cerebral vasoreactivity to noradrenalin, serotonin or prostaglandins[7,8]. Shigeno et al have shown increased levels of norepinephrine in the cerebrospinal fluid of patients with vasospasm; the degree of vasospasm is dose-related to the level of these vasoactive substances[11]. In this case presented, because long segment vasospasm developed the cervical, petrous and cavernous ICA proximal to pituitary tumor, authors suggested that increased cerebral vasoreactiviey to vasoactive substance other than dif-

ferent mechanisms may be reliable in causing the vasospasm suffered by this patient.

Conclusion

Although vasospasm associated with tumor resection is infrequent complication, one must take it into consideration if there is any delayed deterioration in a patient who has undergone cranial base tumor surgery, especially pituitary tumor surgery. And if vasospasm was confirmed by angiography or MRA, aggressive and prompt management is a key element for improving patient's outcome.

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