Postoperative Cerebral Vasospasm Following Transsphenoidal Pituitary Adenoma Surgery

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Key words
- Cerebral vasospasm
- Macroadenoma
- Pituitary
- Transsphenoidal
- Treatment

Abbreviations and Acronyms
CSF: Cerebrospinal fluid
DCI: Delayed cerebral ischemia
MCA: Middle cerebral artery
MRA: Magnetic resonance angiography
MRI: Magnetic resonance imaging
SAH: Subarachnoid hemorrhage
TSS: Transsphenoidal surgery

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PURPOSE: **Cerebral vasospasm following a transsphenoidal resection of a pituitary adenoma is a devastating occurrence that can lead to delayed cerebral ischemia and poor neurologic outcome if not diagnosed and treated in a timely manner. The etiology of this condition is not well understood but can lead to significant arterial vasospasm that causes severe ischemic insults. In this paper, we identify common presenting symptoms and essential management strategies to treat this harmful disease.**

METHODS: **A retrospective case report and literature review of presentation, treatment, and outcome of cerebral vasospasm following transsphenoidal surgery.**

RESULTS: **We present 1 case and review 12 known cases in the literature on vasospasm following transsphenoidal surgery. Mean age was 48 (±13.8) years. There were 46.2% male patients. Factors associated with vasospasm, such as cerebral spinal fluid leaks following surgery, were seen in 38.5% of cases, and postoperative subarachnoid hemorrhage (SAH) was seen in 84.6% of cases. Hemiparesis was the presenting symptom of delayed cerebral ischemia in 61.5% of cases. For management, maintaining at least a euvolemic volume status was used in 76.9%, induced hypertension was used in 61.5%, and nimodipine was administered in 46.2% of cases. Patients returned to their neurologic baseline in 61.5% of cases, had new permanent deficits in 7.7% of cases, and died in 30.8% of cases.**

CONCLUSION: **Cerebral vasospasm following transsphenoidal surgery is a dangerous disease that can lead to a high likelihood of mortality if not identified and treated. Early postoperative events, such as peritumoral subarachnoid hemorrhage and hemiparesis, may be factors associated with post-transsphenoidal surgery vasospasm. Effective treatment options used in patients that regained complete neurologic recovery were by inducing hypertension, maintaining euvolemia, and administering nimodipine.**

INTRODUCTION

Cerebral vasospasm following transsphenoidal surgery (TSS) of a pituitary tumor is a rare occurrence that can result in long-term morbidity and death.[1] The pathophysiology of cerebral vasospasm following a TSS is not well understood and has mainly been described in the literature through case reports.[2-7] In accordance with the Neurocritical Care Society's multidisciplinary consensus conference, the term *vasospasm* refers to radiographic evidence of arterial narrowing following subarachnoid hemorrhage (SAH), while delayed cerebral ischemia (DCI) refers to neurologic deteriorations that results from ischemia.[8] The mechanism of cerebral vasospasm following transsphenoidal surgery is likely multifactorial and may result when an intraoperative tear of the arachnoid leads to SAH in the basal cistern and eventually arterial narrowing in the circle of Willis that can lead to devastating ischemic insults.[2,4,5,9] The rarity of this condition makes it difficult to predict and manage, which can lead to a worse outcome.[2,5,7] The appropriate management for this condition is also poorly understood.[10] The case of a patient with cerebral vasospasm with delayed cerebral ischemia following a transsphenoidal resection of a large pituitary macroadenoma is presented, and we evaluate 12 other known cases of cerebral vasospasm following TSS in the literature. We present the largest comprehensive review and analysis in the literature of all the reported cases on this topic. The etiology, presentation, and management of postoperative TSS vasospasm are also discussed.

METHODS

Our case report is based on 1 patient with postoperative cerebral vasospasm following TSS that was treated in 2014 at Johns Hopkins Hospital. We reviewed the medical records for the purpose of this study. For the literature review, we searched the database of PubMed using search terms: vasospasm, pituitary,

transsphenoidal, and adenoma in all combinations. All original articles reviewed were published between 1980 and 2013 and included 12 cases of transsphenoidal surgeries for pituitary lesions. We performed an analysis on all 13 patients regarding age, gender, preoperative symptom presentation, postoperative cerebral spinal fluid (CSF) leak, postoperative SAH, delayed cerebral ischemia, duration of vasospasm, management, and clinical outcome.

CASE

A 43-year-old otherwise healthy female, with no history of migraines, presented to her primary care physician with headache, fatigue, and blurred vision for several days. Physical examination revealed a bitemporal hemianopsia and a left optic nerve atrophy, with no other neurologic deficits or symptoms of Cushing disease. Endocrinology studies showed elevated 8 AM cortisol and adrenocorticotropic (ACTH) levels (Table 1).

A subsequent preoperative magnetic resonance imaging (MRI) revealed a 2.9 × 3.1 × 3.9 cm (in the transverse, anteroposterior, and craniocaudal dimensions) enhancing pituitary mass with suprasellar extension and compression of the optic chiasm (Figure 1). The mass also extended laterally into the right cavernous sinus. No dedicated vascular imaging was done preoperatively, but a review of MRI showed normal flow voids of the supraclinoidal internal carotid arteries and proximal middle cerebral arteries (MCAs) bilaterally. Given the large size of the pituitary tumor and the symptoms of the patient, a surgical resection was pursued.

The patient underwent an endoscopic endonasal transsphenoidal approach for resection of a pituitary tumor. She was intubated under general anesthesia and sedated using fentanyl and propofol infusions. The suprasellar component of the tumor was fibrous and extended into the third ventricle, pushing up against the hypothalamus. An extensive resection was safely conducted with preservation of the normal pituitary gland. CSF leak was seen intraoperatively after the resection, which required a comprehensive skull base reconstruction using a fat graft, polymer hemostatic agents (Surgicel, Ethicon, Somerville, NJ, USA), absorbable gelatin sponges (Gelfoam, Pfizer, New York City, NY, USA), and fibrin glue (DuraSeal, Covidien, Dublin, Ireland). No complications were incurred during the procedure, and the pathology specimen showed a pituitary adenoma, positive for ACTH immunoreactivity.

The patient tolerated the procedure well and postoperatively remained without any new neurologic deficits. A postoperative MRI showed a small amount of residual tumor along the suprasellar surgical bed with some hemorrhage in the tumor bed (Figure 2). The patient spent 6 days in the neurocritical care unit for management of diabetes insipidus, which required close monitoring of volume status and was treated with vasopressin and free water repletion. Upon transferring to the neurosurgical floor, she continued to experience a milder form of headache that would self-resolve, as well as diabetes, which required a low dose of nightly desmopressin upon discharge on postoperative day 10. Two days after discharge, the patient experienced an acute onset of left-sided paresthesia, left arm weakness, and expressive aphasia while at home. She returned to the hospital, and given the concern for stroke, magnetic resonance angiography (MRA) was obtained. It showed narrowing of the bilateral supraclinoid internal carotid arteries and M1 segments of the MCAs consistent with vasospasm (Figure 3). A small focus of restricted diffusion was also found in the right insular region. Transcranial Dopplers (TCDs) showed elevated cerebral blood flow velocities of 312 cm/seconds on the right M1 MCA and 265 cm/seconds on the left M1 MCA with Lindegaard ratios of 8.9 and 7.6, respectively. The patient was admitted to the neurocritical care unit and hemodynamic management using induced hypertension and euvolemia was started with saline boluses, as well as a phenylephrine infusion to help meet mean arterial pressure goals. Oral nimodipine was also administered, and no signs of hypotension were observed. Neurologically, her examination began to improve 48 hours after beginning the hemodynamic therapy, and there was gradual resolution of her vessel narrowing on serial imaging and TCDs over the course of 16 days, at which point she returned to her neurologic baseline. She was discharged home and postoperative hormonal profile normalized (Table 2).

Table 1. Preoperative Hormonal Profile Taken at 8 AM

Hormone (Normal)	Serum Level	Hormone	Serum Level
Free T4 (0.82–1.77)	0.83 ng/dL	LH (1.7–11.2)	1.7 mIU/mL
TSH (0.45–4.5)	1.430 mIU/L	FSH (1.5–12.4)	5.3 mIU/mL
Cortisol (2.3–19.4)	22.4 mcg/dL	ACTH (6–50)	63 pg/mL
Prolactin (4.8–23.3)	10.3 ng/mL	IGF-I (62–204)	162 ng/mL

LH, luteinizing hormone; TSH, thyroid-stimulating hormone; FSH, follicle-stimulating hormone; ACTH, adrenocorticotropic hormone; IGF, insulinlike growth factor.

RESULTS

Cases were searched for on Pubmed.org using the key words vasospasm, pituitary, transsphenoidal, and adenoma in all combinations. We identified all cases of cerebral vasospasm following transsphenoidal surgery, identified by digital subtraction angiography (DSA) or MRA, between 1980 and 2013, which comprised 12 cases that were reported as case reports. Table 3 summarizes the demographics, presenting symptoms, management, and outcome of all 13 patients, including our case report.

The mean age at presentation was 48 ± 13.8 years (range 30–74), and 46.2% of the patients were male. Preoperative symptom presentation consisted of headaches (38.5%), vision changes (69.2%), galactorrhea (23.1%), amenorrhea (15.4%), decreased libido (7.7%), and fatigue (7.7%). Vision loss was the first presenting symptom in 53.8% of the patients (Table 4).

Following the surgical procedure, CSF leak was seen in 38.5% of cases and postoperative subarachnoid hemorrhage