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Pablo Alfredo Vargas-Ardila,
María Camila Bonilla-Lersundy,
Andrés Felipe Vargas-Ardila,
Leonardo Andrés Chacón-
Zambrano



Early postoperative vasospasm and cerebral infarction after endoscopic endonasal approach for pituitary macroadenoma resection

P. A. Vargas-Ardila^{2,3}, M. C. Bonilla-Lersundy¹, A. F. Vargas-Ardila¹,
L. A. Chacón-Zambrano²

¹ School of Medicine, Universidad Militar Nueva Granada, Bogotá, COLOMBIA

² Department of Neurosurgery, Hospital Militar Central, Bogotá, COLOMBIA

³ Department of Neurosurgery, School of Medicine, Universidad Militar Nueva Granada, Bogotá, COLOMBIA

ABSTRACT

Cerebral vasospasm leading to delayed cerebral ischemia is an uncommon but life-threatening complication of the endoscopic endonasal approach (EEA), which could cause devastating neurological sequelae. Herein, we report the case of a 59-year-old female with a medical history of poorly controlled type 2 diabetes mellitus, high blood pressure, dyslipidemia, and allergy to penicillin, who presented with left eye amaurosis and right temporal hemianopsia, for which a magnetic resonance imaging (MRI) was requested, revealing a pituitary macroadenoma of approximately 3.39 x 3.43 x 3.15 cm. Surgical management was indicated for resection of the tumoral lesion by endoscopic endonasal approach. A postoperative MRI and computed tomography (CT) were performed due to severe deterioration of visual acuity and disorientation in time and place, showing supratentorial subarachnoid hemorrhage in addition to vasogenic edema in the perioperative frontal region. A subsequent magnetic resonance angiography (MRA) revealed acute fronto-cingulate ischemia in the territory of both anterior cerebral arteries (ACA). Management with nimodipine and triple H therapy was initiated, in addition to strict neurological follow-up. The patient also required follow-up and management by endocrinology due to the development of diabetes insipidus. Subsequent imaging confirmed reperfusion of both ACAs; however, the patient was discharged 40 days after surgery with persistent impairment in higher mental functions, including temporal disorientation, bradyphrenia, and operative and episodic amnesia. This is a rare complication, but strict postoperative follow-up aids in timely suspicion to provide indicated treatment, reducing sequelae and improving neurological outcomes.

INTRODUCTION

Pituitary adenomas rank as one of the most encountered primary brain tumors among adults, with an approximate prevalence of 16.7% (5).

Keywords

pituitary neoplasms,
vasospasm, intracranial,
postoperative complications,
brain ischemia,
natural orifice endoscopic
surgery



Corresponding author:
Pablo Alfredo Vargas-Ardila

Hospital Militar Central, Bogotá,
Colombia

pablovargas.medicina@gmail.com

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A common procedure for removing pituitary tumors is the endoscopic endonasal approach (EEA), which is a reasonably safe and effective approach for reaching parasellar and suprasellar tumors (2), however like any surgical intervention, it has complications that in these cases vary between 8.4 and 10.2% with the most common being cerebrospinal fluid leakage, cranial nerve palsy, intracranial hematoma, or meningitis (7,13). Cerebral vasospasm is an extremely rare complication of EEA that typically has its symptomatic onset between 2 to 10 days after surgery. Early symptomatic cerebral vasospasm (within 2 days) is sparse. Clinically it presents with signs and symptoms associated with ischemia in the territory of the implicated vessels (2). In one of the largest single-center retrospective series, only 4 of 1,997 patients (0.2%) who underwent transsphenoidal surgery over an 8-year period developed symptomatic postoperative vasospasm, underscoring how uncommon this complication

truly is despite the large surgical volume required to observe it (13). When it does occur, postoperative subarachnoid hemorrhage or tumor bed hematoma appears to be the most consistently identified risk factor, and the anterior cerebral artery is disproportionately affected compared with other vessels, with unilateral involvement being more frequent than bilateral involvement (5). Due to its rarity, there is not much known about the prevalence, etiology, and management of this condition, that is why vasospasm associated with EEA, in general, is understood and managed using the principles of vasospasm associated with non-traumatic subarachnoid hemorrhage (SAH) (10), as no standardized therapeutic protocol specific to postsurgical vasospasm currently exists. Here we present the case of a patient with early both anterior cerebral arteries vasospasm with posterior ischemia in this territory associated with the resection of a pituitary macroadenoma using a EEA.

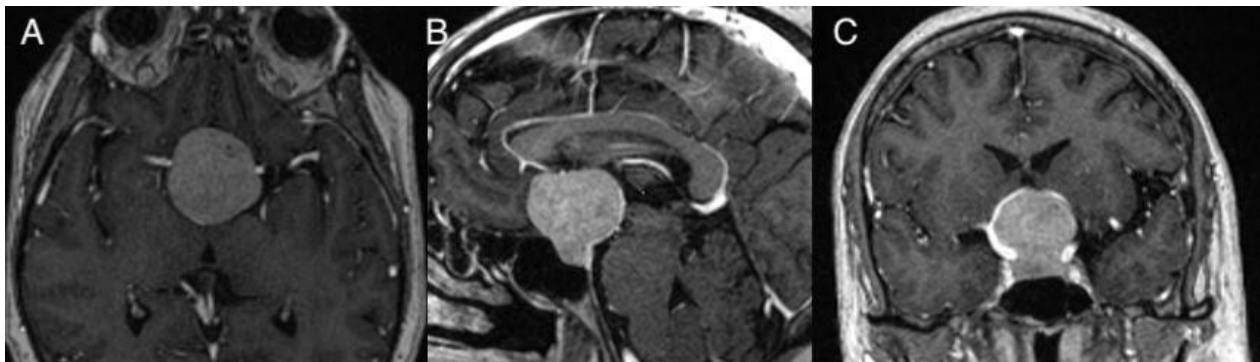


Figure 1. Preoperative magnetic resonance imaging with (A) axial, (B) sagittal and (C) coronal sequences T1 with contrast, showing a large heterogeneously enhanced mass with suprasellar extension to the floor of the third ventricle, approximately $3.39 \times 3.43 \times 3.15$ cm.

CASE DESCRIPTION

A 59-year-old female patient with a history of poorly controlled diabetes mellitus, high blood pressure and dyslipidemia presents with symptoms of left amaurosis and right temporal hemianopsia. A cerebral magnetic resonance imaging (MRI) (Figure 1) reveals a tumoral lesion at the sellar level with homogeneous enhancement on contrast of approximately $3.39 \times 3.43 \times 3.15$ cm, extending to the floor of the third ventricle and the medial wall of both cavernous sinuses, closely adjacent to both internal carotid arteries (supraclinoid segment), anterior cerebral arteries, and anterior communicating artery, radiologically compatible with a pituitary macroadenoma (Knosp

Classification: Grade 2 and Wilson-Hardy: Grade II-D). Serum levels of hypothalamic-pituitary axis hormones showed no elevation. The clinical picture is consistent with a chiasmal syndrome secondary to a non-functioning pituitary macroadenoma, and surgical management via endoscopic endonasal transsphenoidal approach is recommended. Surgical intervention results in wide subtotal resection of the tumoral lesion, without cerebrospinal fluid leakage or excessive bleeding during surgery.

Early postoperatively, the patient experiences somnolence, disorientation, mental confusion, polyuria, and hypernatremia, interpreted as signs and symptoms of diabetes insipidus, initiating active

medical management. Despite medical management, the patient continues to experience fluctuating consciousness. A follow-up MRI of the sella turcica (Figure 2) on the 4th postoperative day reveals significant intratumoral cytorreduction with residual hemorrhagic components, associated with cytotoxic edema and signs of ischemia in the medial and basal surfaces of both frontal lobes related to the territory of both anterior cerebral arteries, with minimal subarachnoid hemorrhage in the occipital horns of the lateral ventricles. Magnetic resonance angiography confirms the absence of flow through both anterior cerebral arteries (Figure 3).

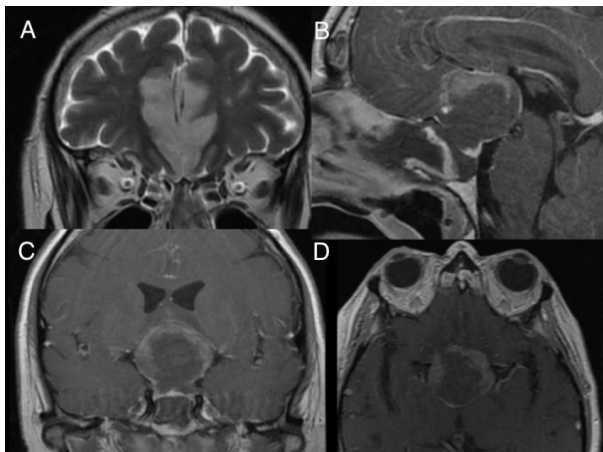


Figure 2. Postoperative magnetic resonance imaging with (A) coronal T2 sequence and (B) sagittal and (C) coronal and (D)

axial T1 with contrast sequence, showing cytotoxic edema in the medial contour of the anterior component of the frontal lobes.

Based on these findings, the patient is diagnosed with secondary cerebral ischemia due to severe vasospasm of both anterior cerebral arteries, with the onset time unclear (intraoperative or early postoperative). Medical management with triple H therapy (hypervolemia, hemodilution, and hypertension) and oral nimodipine (60 mg every 4 hours) for 21 days is initiated. The patient's clinical course shows endocrinological stability but persists with impairment in higher mental functions (temporal disorientation, bradyphrenia, operative, and episodic amnesia). Subsequent radiological studies (angio-CT) demonstrate reperfusion of both anterior cerebral arteries (Figure 4). The histopathological study confirmed the diagnosis of pituitary adenoma (Figure 5). The patient is discharged 40 days after surgery with the described impairments in higher mental functions.



Figure 3. Postoperative magnetic resonance angiography showing (A) vasospasm due to the decrease in the signal magnitude of A1 segments bilaterally, and (B) decrease in the cerebral blood flow volume in the frontal lobes.

Timeline

Day 0: Endoscopic endonasal transsphenoidal resection of pituitary macroadenoma; wide subtotal

resection achieved without CSF leak or excessive bleeding.

Early postoperative period: Onset of somnolence,

disorientation, mental confusion, polyuria, and hypernatremia; diabetes insipidus diagnosed and medical management initiated.

Day 4: Persistent fluctuating consciousness despite treatment; follow-up MRI reveals intratumoral cytorreduction with hemorrhagic residue, cytotoxic edema, and ischemic changes in the medial-basal frontal lobes; MRA confirms absence of flow in both anterior cerebral arteries, consistent with severe bilateral vasospasm.

Day 4 onward (21 days): Initiation of triple H therapy (hypervolemia, hemodilution, hypertension) and oral nimodipine (60 mg every 4 hours). Follow-up: Endocrinological stability achieved; persistent higher mental function impairment (temporal disorientation, bradyphrenia, operative and episodic amnesia). Follow-up angio-CT demonstrates reperfusion of both anterior cerebral arteries.

Day 40: Patient discharged with residual cognitive impairment as described.

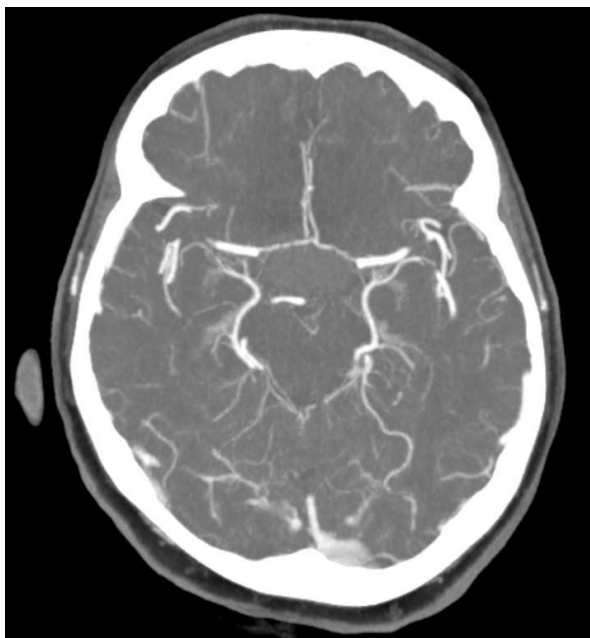


Figure 4. Follow-up axial angio-CT demonstrating reperfusion of both anterior cerebral arteries.

DISCUSSION

The rate of post-EEA vasospasm is unknown; the evidence suggests it is an extremely rare complication, with the first angiogram-confirmed case reported in 1980 and not many cases reported since then (2). Recent systematic reviews confirm

that symptomatic cerebral vasospasm after transsphenoidal pituitary surgery remains an exceedingly rare complication, with a reported incidence of approximately 0.2-0.6% across large surgical series (3,17). The most consistently identified risk factor is postoperative subarachnoid hemorrhage or sellar hematoma, present in 75-94% of reported cases, followed by large tumor size (mean diameter of 39 mm, with tumors ≥ 4 cm conferring greater risk), intraoperative cerebrospinal fluid leakage (present in 84% of cases), intraparenchymal hemorrhage, and parasellar tumor location (3,18). A recently proposed risk stratification score assigns points for dense subarachnoid hemorrhage, intraparenchymal hemorrhage, parasellar location, and tumor size ≥ 4 cm, which may help identify patients warranting closer postoperative surveillance (18). It has been described that post-EEA vasospasm is more frequent in the anterior circulation, with the ACA being the most involved artery (72%). It is also known that unilateral vasospasm is more frequent than bilateral (64% vs. 36%) (5). Common symptoms reported after this complication include aphasia, hemiparesis, weakness, cranial nerve palsies, seizures, and cognitive decline (5,11).

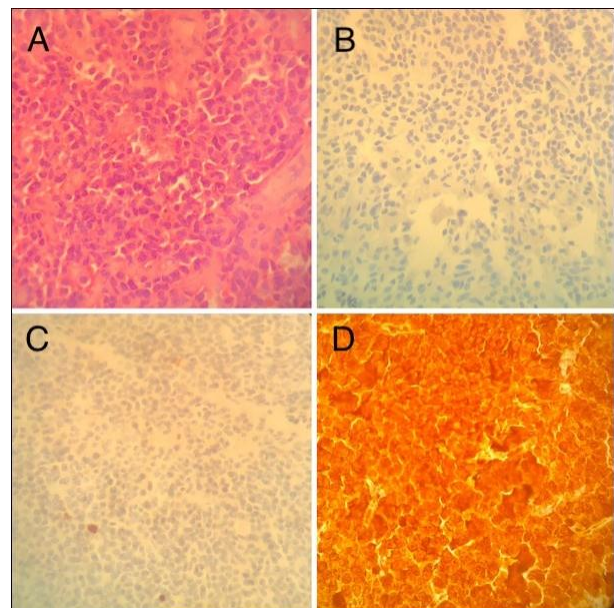


Figure 5. A) A sheet-like proliferation of monomorphic cells with round or oval nuclei and moderate amount of eosinophilic cytoplasm is observed (hematoxylin-eosin 200 $\times$). B) Reticulin histochemical technique demonstrates quenching of the usual adenohypophyseal acinar architecture (Gomori reticulin 200 $\times$).

C) Immunohistochemical study for pituitary hormones (200×).
D) Ki-67 cell proliferation index (200×).

This case is notable for several features that distinguish it from most previously reported cases. First, bilateral involvement of both anterior cerebral arteries is markedly less common than unilateral vasospasm, reported in only 36% of cases in the largest pooled series (5). Second, the exact timing of onset could not be established with certainty, raising the possibility of an intraoperative origin, which is considerably less frequently documented than the more typical delayed presentation between postoperative days 5 and 12 (3,17). Finally, despite prompt recognition and aggressive medical management with triple H therapy and nimodipine, the patient's neurological recovery remained incomplete at discharge, underscoring that even early intervention does not guarantee full reversal of ischemic injury once established. The long-term neurological prognosis after postsurgical cerebral vasospasm is generally unfavorable. Pooled data from recent systematic reviews report complete recovery without sequelae in only 45% of patients, permanent neurological deficits in 32.5-36.7%, and mortality rates between 19.4-22.5% (3,17), although smaller series have reported somewhat better outcomes, with up to 61.5% of patients returning to their neurological baseline (4). Symptom onset typically occurs between postoperative days 5 to 12, with a peak around day 7, and predominantly affects the anterior circulation in more than 90% of cases, often involving multiple vessels (3,17). Although no specific predictors of reversibility have been established, patients who achieved full neurological recovery were more frequently those who received induced hypertension, maintenance of euvolemia, nimodipine administration, and early aggressive treatment (4), which underscores the importance of prompt recognition, as occurred in our patient.

Regarding the pathophysiology of postoperative vasospasm, it remains uncertain; however, several mechanisms seem to be involved. Blood leakage after direct trauma due to manipulation of vessels during the resection is strongly associated with the presence of vasospasm. It has been reported in the literature that about 85% of patients post EEA for resection of pituitary adenomas had subarachnoid hemorrhage (SAH) (1,11,13), all of which are associated with the close spatial relationship between the sellar tumors and the circle of Willis (2).

Other proposed mechanisms point to the size of the tumor as a risk factor. Additionally, the hypothalamic direct damage during surgery or its dysfunction caused by sympathetic activation can lead to the secretion of spasmogenic factors (6). It is also known that tumors produce vasogenic substances such as calcium or lipids, which could induce vasospasm (2,11). Finally, the fluid disorders common in the postoperative period of this surgery lead to microcirculation disturbances, resulting in cerebral salt wasting syndrome or hyponatremia, which are known as risk factors for vasospasm (11).

Regarding treatment, management of postsurgical vasospasm is extrapolated from the principles established for vasospasm following aneurysmal subarachnoid hemorrhage, as no specific guidelines exist for the postsurgical setting (11,16,17). Current evidence favors maintenance of euvolemia rather than hypervolemia, as the latter has been associated with worse outcomes and increased complications, while induced hypertension remains a reasonable strategy for patients with symptomatic delayed cerebral ischemia (9,8). Oral nimodipine, as used in our patient, has been shown in meta-analyses of aneurysmal subarachnoid hemorrhage to reduce the risk of unfavorable outcomes by approximately one third (9), and remains a cornerstone of therapy per the 2023 American Heart Association/American Stroke Association guidelines (8). For refractory cases, more invasive endovascular strategies include intra-arterial calcium channel blockers and balloon angioplasty; a recent meta-analysis found that intra-arterial nicardipine achieved the highest rates of clinical improvement (73.5%) and favorable outcomes (85%) with the lowest mortality (6%), while balloon angioplasty offered superior radiological improvement (93%) but was associated with higher complication and mortality rates (20%) (12). These endovascular options should be considered when induced hypertension fails to reverse symptomatic vasospasm (8).

An emerging area of interest is the use of artificial intelligence and machine learning models for the early prediction of cerebral vasospasm and delayed cerebral ischemia, although to date these have been developed almost exclusively in the context of aneurysmal subarachnoid hemorrhage rather than postsurgical vasospasm. Deep learning models have demonstrated the highest sensitivity (mean 97.6%,

AUC-ROC 0.97), while ensemble methods such as random forest and XGBoost have shown the best balance between sensitivity and specificity (14,15). Logistic regression models remain the most common and interpretable approach, with a pooled C-index of approximately 0.77 in validation cohorts (21). A dynamic model incorporating daily transcranial Doppler parameters, mean arterial pressure, cerebral perfusion pressure, and serum sodium has shown promising accuracy for predicting angiographic vasospasm at any future time point (19). Although these models require external validation and prospective clinical integration before routine adoption, their future application to postsurgical settings such as endoscopic endonasal pituitary surgery could allow earlier identification of high-risk patients and timelier intervention.

Limitations

This report has several limitations inherent to single case descriptions. The exact timing of vasospasm onset (intraoperative versus early postoperative) could not be definitively established, limiting causal inference regarding the triggering mechanism. Diagnosis and follow-up relied on magnetic resonance angiography and angio-CT rather than digital subtraction angiography, which remains the gold standard for vasospasm characterization. Additionally, as an isolated case, findings cannot be generalized, and the relative contributions of tumor size, intraoperative manipulation, and postoperative fluid disturbances to the development of vasospasm in this patient cannot be disentangled from the available data.

CONCLUSIONS

In this case, we highlight that cerebral vasospasm following endoscopic endonasal surgery, while rare, can present with early and bilateral involvement of the anterior cerebral arteries, a pattern less commonly described than the more typical unilateral, delayed presentation. Although the precise pathophysiology remains uncertain, the close spatial relationship between sellar tumors and the circle of Willis, together with subarachnoid hemorrhage secondary to intraoperative vascular manipulation, appears to be the most consistently implicated mechanism. This complication carries substantial risk of permanent neurological morbidity, and in our patient, higher mental function

impairment persisted despite prompt recognition and aggressive medical management. This underscores that even timely diagnosis and treatment do not guarantee complete neurological recovery once ischemic injury has occurred. Given the absence of standardized management protocols specific to this setting, a high index of suspicion, close postoperative neurological surveillance, and early initiation of therapy extrapolated from aneurysmal subarachnoid hemorrhage management remain the cornerstone of care. Future research, including the potential application of predictive models, may help identify high-risk patients before clinical deterioration occurs.

Abbreviations

EEA: Endoscopic Endonasal Approach; SAH: Subarachnoid Hemorrhage; MR: Magnetic Resonance; CT: Computed Tomography; MRA: Magnetic Resonance Angiography; ACA: Anterior Cerebral Arteries.

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