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Vasospasm after transsphenoidal pituitary surgery: A case report and review of the literature

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Vasospasm, so commonly described after aneurysmal rupture, is very rare after surgery for brain tumors. Its occurrence after transsphenoidal surgery is extremely uncommon with only three cases reported as per the authors' review of the literature. The authors report a case of pituitary macroadenoma in a 34-year-old female who underwent transsphenoidal tumor decompression and developed angiographically documented vasospasm during the course of her illness. The rarity of the occurrence of vasospasm after transsphenoidal surgery makes its management very challenging, frequently leading to a delay in diagnosis and a poor outcome. The key to successful management is a high index of suspicion and early proactive management.

Key words: Pituitary adenoma, transsphenoidal surgery, vasospasm

Vasospasm, the leading cause of morbidity and mortality after rupture of intracranial aneurysm is very rare after transsphenoidal surgery with only three cases reported as per the authors' review of the literature.^[1-3] Though rare after surgery for brain tumors, the potential to cause significant neurological sequel remains the same as after aneurysmal rupture.^[4] Moreover, the rarity and unpredictability of its occurrence after tumor surgery frequently leads to a delay in diagnosis and a worse outcome.^[1-3] The authors report a case of arterial vasospasm after sublabial transsphenoidal removal of a pituitary adenoma and discuss the possible mechanisms of its occurrence and management. Recognizing this entity as a cause of neurological deterioration with aggressive management of the same may avoid untoward consequences.

Case Report

A 34-year-old female presented with amenorrhea, galactorrhea and dull headache of one year duration along with bilateral diminution of vision. On examination the visual acuity in both eyes was hand movements

close to face with bitemporal field cuts on perimetry. Fundus examination revealed bilateral primary optic atrophy with no other cranial nerve palsy or any motor or sensory deficit on examination. Routine biochemical investigations and complete serum hormonal profile were within normal limits. Magnetic Resonance Imaging (MRI) of the brain showed an isointense sellar suprasellar mass lesion with enlargement of sella [Figure 1].

A diagnosis of pituitary macroadenoma was made and a sublabial transsphenoidal tumor decompression was done. Cerebrospinal fluid (CSF) leak was present for which sellar packing with fat and fascia was done. On the third postoperative day, patient developed deterioration in vision in both eyes. Non-contrast computed tomography (CT) head was done which showed blood in suprasellar cistern/residual tumor. Patient was reexplored transsphenoidally and altered blood present in the operative cavity was evacuated. Sella was repacked and postoperatively lumbar CSF drain was kept. There was no episode of any vascular injury per-operatively. The patient developed high-grade fever during the postoperative course. The CSF

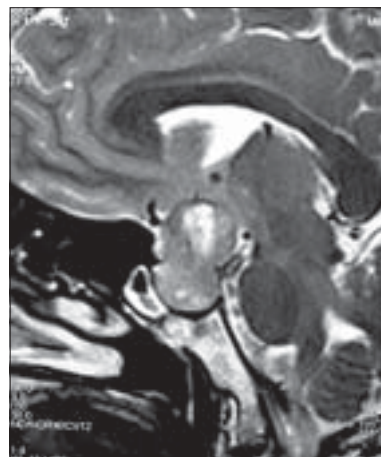


Figure 1: Sagittal MRI brain showing a sellar suprasellar mass lesion isointense to grey matter with enlargement of sella suggestive of a pituitary macroadenoma

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analysis was suggestive of meningitis and culture grew pseudomonas organism. Culture-based antibiotics were started and patient gradually improved. On Day 13 after surgery, patient developed right-sided weakness and focal convulsions involving the right side of the body. Non-contrast CT head revealed a hypodense area in the left caudate nucleus and anterior limb of the internal capsule [Figure 2]. The infarct gradually increased in size on further scans and patient deteriorated neurologically. Transcranial Doppler (TCD) showed increased velocities in middle cerebral arteries with non-insonable anterior cerebral arteries bilaterally. Angiography was done which revealed spasm of the left internal carotid artery (ICA) bifurcation and ICA narrowing in the cavernous region [Figure 3]. Biochemical parameters with electrolyte levels were within normal limits. Hypervolemic therapy was started. Intraarterial papaverine was not given as patient had already developed an infarct. Patient finally died on Day 1 after angiography.



Figure 2: Non-contrast CT head revealing a hypodensity in the basal ganglia and anterior limb of the internal capsule

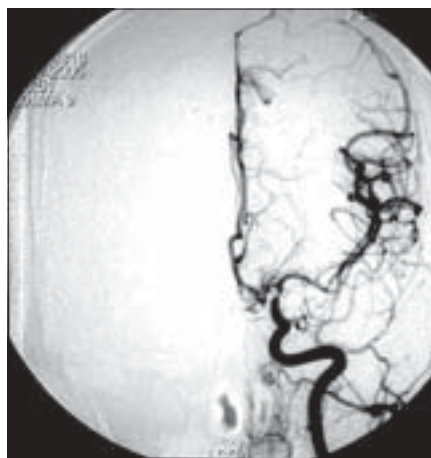


Figure 3: Antero-posterior projection of the left internal carotid artery (ICA) angiogram showing ICA narrowing in the cavernous region and spasm at left ICA bifurcation

Discussion

Transsphenoidal surgery has become the preferred approach for most of the cases of pituitary adenoma. Vascular injuries though rare, form a well-recognized complication of transsphenoidal surgery. However, cerebral vasospasm following transsphenoidal pituitary surgery has been extremely infrequent with only three cases described as per the authors' review of the literature.^[1-3] Of these, two patients died and only one of them had a successful recovery following onset of vasospasm.^[1-3]

Cerebral vasospasm and its management has remained a great challenge for the entire neurosurgical community ever since its description for the first time by Ecker *et al.* in 1951. Cerebral vasospasm after aneurysmal subarachnoid hemorrhage (SAH) is a well-known entity responsible for significant neurological deterioration and morbidity.^[5] Vasospasm can be associated with other conditions like trauma, tumors or arteriovenous malformation bleed and can equally complicate the management of these patients as in cases of aneurysmal SAH.^[5] There are very few cases of vasospasm after surgery for brain tumors.^[1-3,6] The pathogenesis of vasospasm after tumor surgery is not very well understood. Though pituitary adenomas are the most frequent tumors in which vasospasm is described, most of it is associated with transcranial approaches; vasospasm being very rare after transsphenoidal approach.^[1-3,6] Various possible causes for vasospasm after tumor surgery include direct mechanical damage to arterial walls, deposition of blood in the basal cisterns, meningitis, hypothalamic damage or release of some chemical substances during tumor removal.^[4,6-8] In an experimental study by Kamal *et al.* craniopharyngioma fluid was shown to cause vasospasm of the femoral vessels in rat and its spillage during surgery can be the cause of vascular complications after surgery for craniopharyngioma.^[9] Vasospasm was similarly shown after injection of hypothalamic extract in a dog model by Wilson *et al.*,^[7] hypothesizing the role of hypothalamic injury in cases of vasospasm around the sellar region. The exact cause of vasospasm in our case could not be determined; however, it could be attributed to unrecognized hemorrhage during surgery, previous pituitary apoplexy or meningitis. A review of the literature does reveal the association of meningitis with vasospasm.^[10] Cerebrospinal fluid leak also occurred during transsphenoidal surgery in two reported cases of vasospasm as in our case and thus the role of underlying subclinical meningitis due to the CSF leak can't be ruled out conclusively even in those cases, making meningitis a likely contributing factor towards the development of vasospasm.^[1,2,10]

Various new strategies are available for the prevention of vasospasm after subarachnoid hemorrhage like subarachnoid clot removal, lysis with fibrinolytic

agents or in some experimental studies by intrathecally administered slow-release vasodilators and endothelin antagonists. However, considering the very low incidence of vasospasm after surgery for skull base tumors and after transsphenoidal surgery, the use of such agents seems controversial. Various forms of treatment are available for the treatment of vasospasm after SAH which include triple H therapy, intraarterial papaverine administration and percutaneous transluminal balloon angioplasty. The use of all the three modalities has been described in cases of vasospasms after brain tumors.^[1,4,6] Nishioka *et al.* used triple H therapy and intravenous thromboxane A2 antagonists and reported a good outcome in vasospasm after transsphenoidal surgery.^[3] Intraarterial papaverine could not be used in our case as the patient had already developed infarct. However, it would have been a viable option if the vasospasm was diagnosed earlier.

To conclude, though vasospasm is rare after transsphenoidal surgery, with exact pathogenesis still obscure, its existence has been well described as in this case. The management can be very challenging and the key to success is a high index of suspicion and early proactive management. Though there were multiple factors in our case which were responsible for the ultimate demise of the patient, vasospasm did play a critical role in contributing towards the terminal outcome. Vasospasm may be considered as a cause of neurological deterioration after transsphenoidal surgery and should be diagnosed and managed aggressively,

especially if the patient develops any focal neurological signs or evidence of meningitis in the postoperative period.

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