Stroke Associated with Severe Cerebral Vasospasm after Petroclival Meningioma Resection

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Objective: We report a rare case of stroke associated with delayed severe vasospasm following resection of petroclival meningioma

Background: Diffuse cerebral vasospasm is a frequent complication following aneurysmal subarachnoid hemorrhage or after severe traumatic brain injury; however, symptomatic cerebral vasospasm following intracranial tumor resection is a rare and under recognized condition.

Design/Methods:A 55-year-old woman with a petroclivalmeningioma, presented with intractable seizures and decreased right hand dexterity. She underwent a nine hour-long procedure for resection of the mass and was discharged home on POD# 7 with mild slurred speech and IV nerve palsy. She was readmitted on POD#11 for evaluation of four-day history of slurred speech and lethargy.

Results: MRI showed acute infarcts in the left posterior frontal, parietal and occipital lobes, perisylvian region, corpus callosum splenium, mesial temporal lobe, hypothalamus, and bilateral pontomesencephalic junctions. MRA was remarkable for diffuse vasospasm, including: 1) severe stenosis of terminal ICAs bilaterally, 2) significant stenosis in the proximal ACA A1, MCA M1, and ACA A2 segments bilaterally 3) moderate-severe stenosis in both vertebral arteries and severe stenosis in mid basilar artery and proximal PCA. Four-vessel angiogram confirmed the findings. Patient was subsequently treated with balloon angioplasty and intrarterial nicardipine, with marked improvement in symptoms.

Conclusions: Vasospasm is a rare but important cause of neurological deterioration following brain tumor resection, and warrants prompt diagnosis and aggressive management.Purported mechanisms for the development of the vasospasm include presence of blood in the basal cisterns, vessel manipulation, and tumor-related inflammatory mediators.

Introduction

Cerebral vasospasm (CV) is a well-recognized complication of aneurysmal subarachnoid hemorrhage (SAH). Although the presence of blood in subarachnoid space has been implicated as the strongest predictor of vasospasm, the exact pathophysiology remains elusive. CV has been reported in other pathological states such as traumatic SAH , intraventricular hemorrhage from non-aneurysmal ruptu0re, head trauma without SAH , periprocedural clipping or coiling of unruptured aneurysms, meningitis, and preeclampsia . Recently, there is a small body of literature reporting cerebral vasospasm after tumor resection[1,2]. Reports of CV in the setting of atypical etiologies are essential to better understanding this serious condition, which may lead to stroke, and permanent disability. This is particularly important since the index of suspicion for vasospasm following brain tumor resection, is extremely low. We present a case of delayed severe symptomatic diffuse vasospasm following meningoma resection, which required aggressive management with induced hypertension and intraluminal balloon angioplasty.

Case Report

A 55 yr old woman with a known left petroclival mass, followed conservatively over years, presented with left facial twitching and difficulty writing with her right hand. As the MRI of the brain revealed an interval increase in the size of the mass from the previous scan, and the patient was now symptomatic, the decision was made to resect the lesion. Preoperatively, the patient had a non-focal neurologic exam. The patient underwent a nine-hour craniotomy, using a left posterior transpetrosal approach. A gross total resection was performed, with only a small cuff of tumor left surrounding the left 5th and left 6th cranial nerves to limit cranial neuropathy. The total estimated blood loss intraoperatively was 350ml. Pathology was consistent with meningioma with atypical features (WHO grade I).

On post-operative Day 1 (POD1), the patient was oriented to self and place, and had left CN VI and VII deficits. CT head showed mild homogenous hyperdensity within the basal cisterns and lateral ventricles, indicating blood.(Fig-1)

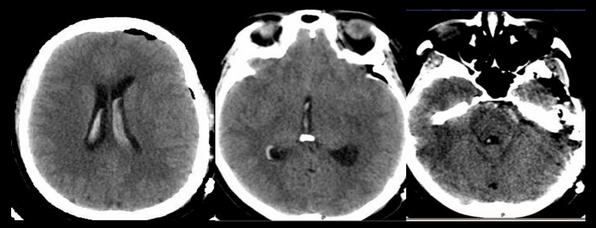

Fig-1

On POD 2 the neurological exam was without focal deficits. On POD3 to POD 8, the patient was noted to have mild slurred speech and CN VI palsy, but was not significantly changed, and she was discharged home. On POD 12, the patient was brought to the emergency room after two days of progressively decreasing consciousness and mutism. On exam, the patient was lethargic and oriented only to self but was not following commands. The patient had bilateral VI and VII nerve palsies. She was intubated for airway protection, and admitted to the neuro-ICU. Stat brain MRI revealed superimposed patchy acute infarcts in the left posterior frontal, parietal, and occipital lobes, perisylvian region, splenium of the corpus callosum, mesial temporal lobe, hypothalamus, and bilateral pontomesencephalic junctions(Fig2a-h).MRA showed diffuse vasospasm in the anterior and posterior circulation. (Fig-3)

Given the constellation of symptoms and radiographic changes the patient underwent urgent four vessel cerebral angiogram revealing extensive bilateral vasospasm in the anterior and posterior circulation territories.

The patient subsequently underwent angioplasty and received intra-arterial nicardipine. Concomitantly, patient was started on hypertensive therapy. Her exam gradually improved, and blood pressure allowed to normalize. The patient was extubated on POD 14, and subsequently transferred to the floor. She was discharged to inpatient rehabilitation a week later, and had marked improvement by discharge with near complete resolution of aphasia and only slight weakness of right upper extremity and left VI nerve palsy.

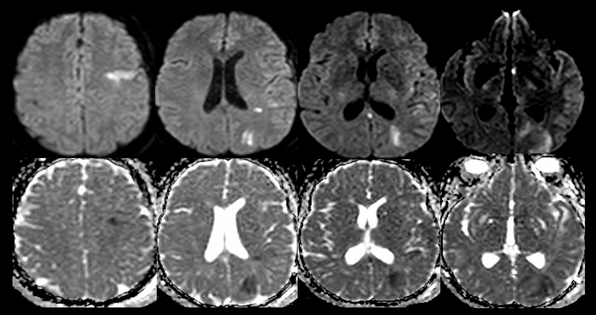

Fig-2. upper Row : DWI; Lower ROW : ADC

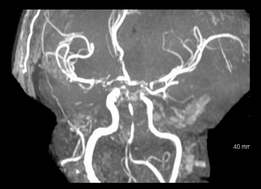

Fig-3

Discussion:

Vasospasm, commonly described after aneurysmal SAH, is very rare following tumor resection. The overall incidence is 1.9%.[2]This explains why the index of suspicion is low and the diagnosis is often missed..

The pathophysiological basis of vasospasm in the setting of tumor resection is obscure. In a large series of skull-base surgeries [2], the authors found nine patients with evidence of post-operative vasospasm; eight of whom were symptomatic. After correlating several variables, they concluded that patients with large tumors (more than 4 cm), with vascular component with encasement and narrowing of major vessels, or that took longer to resect, were more likely to develop vasospasm. The possibility that manipulation of the major vessels of the basal cisterns produced the observed phenomenon cannot be ruled out; however, it is unlikely that this mechanism led to vasospasm in our patient, since there was no significant vessel encasement in this patient, and thus little vessel manipulation occurred in this patient. Additionally, there was diffuse rather than regional vasospasm which would suggest diffuse inflammation rather than focal vessel spasm from mechanical manipulation of arteries. Of note, authors [2] failed to mention whether vasospasm developed in the vessel that was encased or in the distant vessels.

Most prevailing hypotheses clearly relate the development of vasospasm to presence of blood in the subarachnoid space[3]. Direct relationship between the amount of blood and subsequent development of vasospasm has been demonstrated in both animal [10] and human studies[11]. Although meningiomas are prone to bleed intraoperatively, in our case preoperative embolization was not performed since the suspicion for encountering major bleeding was considered low. Estimated blood loss was only 250 ml. Further measures were implemented to minimize spillage of blood in the subarachnoid spaces, gelfoam was used to isolate the surgical area from the remaining cisterns, repeatedly thorough washing of the cisterns was performed, and rundown of the epidural blood during dural closure was prevented. We believe that blood leakage into the basal cisterns is a likely contributor in this case; however, the amount of vasopasm was out of proportion to the amount of blood in the basal cisterns as seen on post-operative head CT. Therefore, we suspect another mechanism.

Some have speculated that vasoactive materials liberated from the tumor bed, either at the time of surgery, or post-operatively, related to tumor necrosis may induce cerebral vasospasm. [4] We speculate that release of tumor-derived inflammatory mediators may contribute to vasospasm, and needs to be further studied in future studies.

While one may argue that blood in perimesencephalic cisterns could independently induce vasospasm, this is extremely unlikely since the clinical course of perimesencephalic nonaneurysmal SAH usually follows a benign course [5]. In a study by Rinkel et al[5], authors compared the clinical course of 65 patients with perimesencephalic non-aneurysmal SAH to 49 patients with aneurysmal SAH who presented with a similar clinical grade and similar amount of cisternal subarachnoid blood on head CT. Patients with perimesencephalic nonaneurysmal SAH had a better clinical course than those with aneurysmal SAH and none had vasospasm-induced ischemic stroke, unlike our patient who suffered multiple ischemic strokes.

In conclusion, although CV is relatively uncommon following tumor resection, its existence has been well documented in an increasing number of cases. There is no clear inciting factor for this phenomenon; but it is evident that multiple factors may be implicated in its development. Regardless of the mechanisms, it appears that the interval for the occurrence of CV after tumor resection tends to parallel that which is seen after aneurysmal SAH. Thus, one should have a high index of suspicion that vasospasm may be the cause of neurological deterioration post-operatively after tumor resection, and this should lead to prompt diagnosis and aggressive management.

References:

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