



Symptomatic Cerebral Vasospasm Following Left Vestibular Schwannoma Surgery – A Case Report.

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KEYWORDS

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ABSTRACT:

Cerebral vasospasm is a well-recognized complication of aneurysmal subarachnoid hemorrhage, but is exceedingly rare following posterior fossa tumor surgery. Only a few cases have been reported in association with vestibular schwannoma resections, making it a clinically significant postoperative concern due to its potential impact on neurological outcomes. We present a case of delayed symptomatic cerebral vasospasm following the resection of a left vestibular schwannoma in a 50-year-old female with a history of hypertension. The patient presented with progressive left-sided hearing loss, tinnitus, imbalance, headaches, and facial numbness. MRI revealed a T2 hyperintense lesion in the left cerebello-pontine angle with cystic and hemorrhagic areas, suggesting an acoustic schwannoma. Surgical resection was performed under intraoperative neuro-monitoring, with minor bleeding and no significant intraoperative complications. Post-operatively, the patient developed extensive subarachnoid hemorrhage (SAH) and subsequently, right-sided weakness and vasospasm on CT angiography. Despite initial management with nimodipine and cerebroselective vasodilators, the patient's condition worsened with declining sensorium and hemiparesis. A ventriculoperitoneal shunt was performed on post-operative day 10. The patient's recovery was slow, with eventual improvement and discharge, though with residual right hemiparesis and impaired ambulatory function. Follow-up MRI showed resolving vasospasm. This case highlights the importance of early recognition and aggressive management of post-surgical vasospasm, a rare but potentially devastating complication in vestibular schwannoma surgery.

Introduction

Vestibular schwannomas are common benign tumors of the cerebellopontine angle, accounting for nearly 8% of all intracranial tumors. Surgical excision remains a key treatment option, especially for symptomatic or enlarging lesions. While well-established postoperative complications include facial nerve palsy, cerebrospinal fluid leakage, and cerebellar dysfunction, cerebral

vasospasm after posterior fossa tumor surgery is exceptionally uncommon. Most documented cases of vasospasm occur following aneurysmal subarachnoid hemorrhage, where the pathophysiology and timelines are well understood. In contrast, vasospasm after tumor resection—particularly vestibular schwannoma excision—has been reported only in a handful of cases, making its true incidence, mechanisms, and optimal management less clearly defined.



Several mechanisms have been proposed to explain this phenomenon. These include subarachnoid blood contamination during surgery, direct manipulation of neurovascular structures, sympathetic irritation from cranial nerve handling, and inflammatory changes in the basal cisterns. The delayed onset of vasospasm in these postoperative settings often makes early recognition challenging. Clinical deterioration—typically manifesting as new focal deficits, altered sensorium, or worsening hydrocephalus—may be mistaken for routine postoperative edema or ischemia, further delaying diagnosis.

Because vasospasm in this context can lead to significant morbidity if not promptly identified, each additional case expands the collective understanding of its clinical spectrum and contributes to improved vigilance. We present a case of delayed symptomatic cerebral vasospasm following resection of a left vestibular schwannoma, highlighting the diagnostic challenges, radiological features, and the need for early intervention to prevent irreversible neurological injury. This case adds to the limited literature and reinforces the importance of postoperative monitoring for vasospasm in posterior fossa tumor surgery.

Case Presentation

A 50-year-old female with a known case of Hypertension on Tab Metoprolol succinate 25 mg BD for 10 years presented with 2 years of progressive left-sided hearing loss, left-ear tinnitus for 1.5 years, imbalance, headaches, and left facial numbness for 6

months. On examination, the Patient was Conscious, oriented with Time, Place, and person, with a Glasgow Coma scale of E4M6V5. The Patient presented with left-sided sensorineural hearing loss, confirmed upon Rinne and Weber tests, with Weber lateralizing to the right ear and Rinne test negative on the left side.. There is subtle left facial weakness, particularly in the lower face, with mild drooping of the mouth corner and flattening of the nasolabial fold, indicating possible facial nerve involvement. The rest of the cranial nerve examination was normal. Additionally, truncal ataxia is noted, with mild unsteadiness during the Romberg test and tandem walking, suggesting cerebellar and vestibular signs. No significant deficits in motor strength, sensation, or reflexes were observed, but slight dysmetria is present on finger-to-nose testing. With findings of hearing loss, facial weakness, and ataxia, we investigated further with neuroimaging. The patient is Overweight, with a BMI of approximately 28 Kg/m². No similar history was noted in any family members. MRI Brain with contrast revealed a T2 hyper-intense lesion in the left cerebello-pontine angle with cystic and haemorrhagic areas, extending into the internal acoustic meatus, causing mild obstructive hydrocephalus, likely an acoustic schwannoma [figure 1a,b]. Pure tone audiometry showed left sensorineural hearing loss and minimal right-sided hearing loss. The patient was evaluated and scheduled for an elective left retro-mastoid sub-occipital craniotomy and tumor excision under intraoperative neuro monitoring (IONM) guidance.

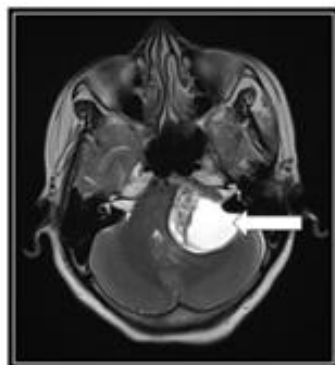


Fig 1a – Showing Axial MRI Brain image showing left CP angle Tumor causing Brainstem Compression.

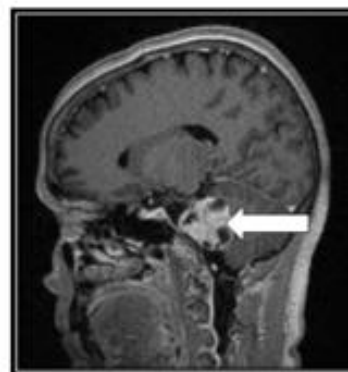


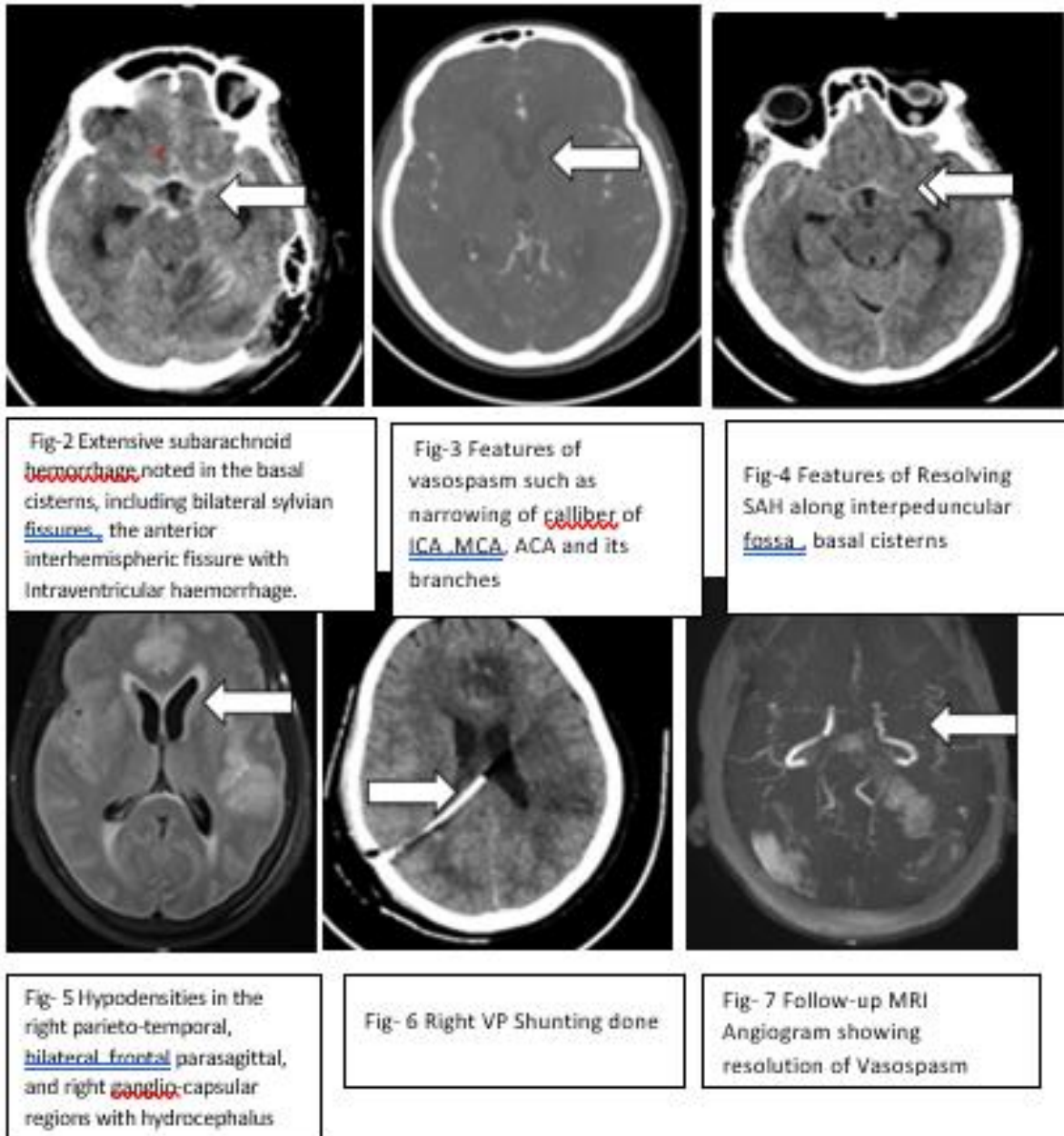
Fig 1b – Sagittal view of MRI Brain image showing left CP angle Tumor causing Brainstem



The patient was positioned in the park bench position for a left retro-sigmoid suboccipital craniectomy and near-total tumor excision under IONM monitoring of cranial nerves V, VII, IX, X, XI, and XII. The tumor had both soft and solid-cystic characteristics. The tumor capsule was carefully dissected from the cranial nerves, cerebellum, and brainstem. Minor bleeding occurred while decompressing the solid portion of the tumor. There was no significant intraoperative bleeding. The total blood loss was approximately 300 ml.

On POD 1, the patient was intubated and connected to a mechanical ventilator. By POD 2, she was extubated, conscious, oriented, and able to move all four limbs. CT Brain plain was done on POD 2, revealing Extensive subarachnoid hemorrhage in the basal cisterns, including bilateral sylvian fissures and the anterior interhemispheric fissure. Intraventricular hemorrhage was noted with clots in the anterior horns of bilateral lateral ventricles, 3rd and 4th ventricles. Minimal blood-fluid level in the occipital horns of bilateral lateral ventricles. Diffuse sulcal effacement was noted. Mild hydrocephalus. (Fig 2). The patient was started on cerebroselective vasodilators. Although the patient was conscious, oriented, and ambulant with oral feeds, she developed Grade 1 Left Lower Motor Neuron Facial Palsy and mild cerebellar signs. On POD 05, the patient developed right-sided weakness. A CT cerebral angiogram was done. The intracranial arteries exhibit diffuse mild narrowing of the vessel caliber, with normal opacification observed in the internal carotid arteries. Similarly, the bilateral anterior cerebral arteries, middle cerebral arteries, and their branches show diffusely mild narrowing of the vessel caliber, with normal opacification. The vertebro-basilar arteries, posterior cerebral arteries, and their branches also demonstrate diffusely mild narrowing of the vessel caliber, with normal opacification suggestive of Vasospasm (Fig. 3). The duration from surgery to the development of Vasospasm was 05 days. The patient was maintained on hydration, avoided hypotension, and continued on nimodipine.

By POD 8, the patient developed upward rolling of the eyeballs, accompanied by a decline in sensorium (GCS E4V1M5). An urgent CT Brain revealed resolving SAH along the interpeduncular fossa, basal cisterns, Sylvian fissures, and para-sagittal frontal lobe sulci (Fig 4). Injection Methylprednisolone 1gram IV BD for 2 days, followed by Injection Methylprednisolone 500mg IV BD for 3 days, and oral Tab Nimodipine 60mg 6 times a day for 21 days was given. On POD 9, despite no significant improvement in sensorium and weakness in the right upper and lower limbs, an MRI Brain showed multiple ill-defined hypodensities in the right parieto-temporal, bilateral inferior frontal parasagittal, and right ganglio-capsular regions, along with multifocal subarachnoid hemorrhage and mild ventriculomegaly (Fig 5). As the patient had no signs of Clinical deterioration, it was decided to continue the conservative line of treatment for hydrocephalus. On POD 10 The patient's conscious level deteriorated, right hemiparesis worsened, GCS was E2M4V1, and was desaturating, SPO2 on room air was 86%, Hence was intubated and connected to mechanical ventilation, Based on clinical deterioration and MRI image showing Hydrocephalus, decision of emergency VP shunt was decided, and right VP shunt done on POD 10 (Fig 6). She was placed on ventilatory support with tracheostomy, antibiotics, Tab nimodipine 60 mg 6 times a day, iv mannitol 100ml tds and inj.Frusemide 20mg BD for 10 days, and intensive physiotherapy. Gradually, she was weaned from the ventilator on POD 16 post VP shunting. Patient slowly recovered, Tracheostomy closed after 1 month, was under treatment for 2 months and patient got discharged with GCS E4M6V5 with right hemiparesis and is Ambulant with support after 2 months. MRI angiogram done on follow-up 15 days post discharge, shows resolving features of Vasospasm. [Fig 7]



Discussion

Although rare, cerebral vasospasm represents a significant postoperative complication in neurosurgery, particularly in procedures involving cranial base lesions such as vestibular schwannomas.⁽¹⁾ . Originating from Schwann cells of the vestibular nerve, these benign lesions frequently require extensive posterior fossa surgery. The delayed onset of symptomatic vasospasm after such procedures presents a notable challenge, as it

can produce significant neurological deficits or ischemic injury.^(1,6,8)

Risk Factors in Posterior Fossa Surgery

Several intraoperative factors increase the risk of vasospasm in posterior fossa tumor resections:

- Increasing tumor size often necessitates more extensive operative handling, which in turn heightens local tissue stress and predisposes surrounding vessels to irritation.^(1,8)



- Suboptimal patient positioning in posterior fossa procedures may alter the orientation or tension of vascular structures, creating conditions that favor vasospasm.⁽¹⁾
- When bleeding is excessive, transient drops in cerebral blood flow and endothelial irritation may occur, and these changes can trigger postoperative vasospasm^(1,9).
- Direct surgical handling of sensitive vessels and nerves in the posterior fossa may induce localized inflammatory reactions that increase the risk of vasospasm^(1,2,4).
- Endothelial injury disrupts nitric oxide release, impairing vasodilation and creating conditions that maintain ongoing vasospasm^(1,8).
- The delayed onset of vasospasm reflects progressive alterations in vascular tone and the slow buildup of vasoactive mediators, which together make early diagnosis challenging^(5,7,10).

Diagnosis

Accurate diagnosis of vasospasm relies heavily on imaging. CT and MR angiography offer noninvasive visualization of arterial constriction, whereas digital subtraction angiography remains the definitive modality for comprehensive assessment.^(1,8)

Prevention and Management

Effective prophylaxis involves maintaining steady hemodynamics, providing adequate hydration, and preventing intraoperative hypoxia or hypotension.^(1,8) Agents like nimodipine, which act as calcium channel blockers, have demonstrated effectiveness in reducing the risk of vasospasm^(1,6,8).

Vasospasm is managed with vasodilators and calcium channel blockers, while refractory cases may need balloon angioplasty or intra-arterial vasodilator therapy.^(1,2,7,8)

Pathophysiology

The pathogenesis of postoperative cerebral vasospasm is multifactorial:

- Tissue trauma and postoperative bleeding initiate inflammatory signaling that disrupts endothelial integrity and promotes vasoconstriction. Cytokines, including interleukins and TNF, heighten smooth-muscle contraction and limit normal vasodilation.^(1,3,9)
- Trauma to the vestibular nerve can trigger sympathetic activation, leading to heightened vascular constriction. Its proximity to major neurovascular structures makes this mechanism particularly relevant in posterior fossa surgery.^(1,6)

Table summarizes the clinical details of 6 reported cases of symptomatic cerebral vasospasm after the excision of posterior fossa schwannoma

<u>Authors</u>	<u>Age/ Sex</u>	<u>Location</u>	<u>CSF Divers ion</u>	<u>Tum or Size (cm)</u>	<u>Cys tic</u>	<u>Blo od Lo ss</u>	<u>SAH Evide nce</u>	<u>Sympt om Onset</u>	<u>Vess els</u>	<u>Treat ment</u>	<u>Outcom e</u>
Krayenbuehl, 1960 [3]	39, M	Lt Vestibular	No	NA	No	NA	NA	NA	ICA	NA	NA
LeRoux et al., 1991 [7]	69, M	Lt Vestibular	Yes	3	Yes	Yes	No	7	B/L VA, BA Rt	HHH therapy, nimodi	Disability (mild gait)



									ICA	pine	ataxia)
Qi et al., 2015 [5]	16, M	Lt Ventral Medulla	No	2.5	No	No	No	3	B/I ICA & BA	HHH therapy, decompressive craniectomy	Disability (Dependent for daily activities)
Pasricha, P et al., 2024 [10]	56, F	Rt Vestibular	No	4.6	Yes	Yes	Yes	6	Rt MCA, Proximal M1 segment of Lt MCA, Distal BA	Hyperdynamic therapy, nimodipine, anticoagulants	Disability (Residual hemiparesis)
Our case	50, F	Lt Vestibular	Yes	4.5	Yes	No	Yes	8	Left MCA, Left ACA	Hyperdynamic therapy, nimodipinsteroids, and antioedema measures	Disability (Ambulant with support)

ACA = anterior cerebral artery; BA = basilar artery; FU = follow-up; HHH therapy = hypertensive, hypervolemic, hemodilution therapy; ICA = internal carotid artery ; MCA = middle cerebral artery; NA = not available; VA = vertebral artery, GOS- Glasgow Outcome Score

Conclusion

Delayed symptomatic cerebral vasospasm following vestibular schwannoma resection is a rare but potentially debilitating complication. Early detection

and timely intervention are critical to improving outcomes. Further research and case documentation will enhance understanding and treatment strategies for this challenging complication.



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