



# Distal Symptomatic Delayed Vasospasm Following Cerebellopontine Angle Meningioma Resection: An Elucidative Case

Sanjay SV\*, Shivani SV, Aditya SS and Snehal RV

Department of Neurosurgery, BJGMC and Sassoon General Hospital, Pune, Maharashtra

\*Corresponding author: Sanjay S Vohra, Department of Neurosurgery, BJGMC and Sassoon General Hospital, Pune, Maharashtra, India, Email: drsanjayvohra12@gmail.com

Received Date: August 12, 2024; Published Date: September 04, 2024

## Abstract

**Background:** Symptomatic cerebral vasospasm following posterior fossa extraaxial tumor resection is a rare phenomenon, with only 13 cases previously reported in the literature. The condition appears similar to vasospasm following supratentorial tumor resection, intraaxial posterior fossa tumor resection, and aneurysmal subarachnoid hemorrhage (aSAH). The majority of patients were not evaluated for vasospasm prior to symptom onset, leading to a delay in diagnosis.

**Observations:** The authors present their experience in a 55-year-old female who developed delayed cerebral vasospasm after excision of a left sided cerebellopontine angle Meningioma.. Routine postoperative brain computed tomography showed evidence of subarachnoid hemorrhage in the basal cisterns. She developed frontal lobe signs on 7th post-operative day. Angiography confirmed bilateral frontal diffuse cerebral vasospasm. The patient responded to standard hyperdynamic therapy used for vasospasm secondary to aSAH.

**Conclusion:** Symptomatic distant cerebral vasospasm after posterior fossa extraaxial tumor excision is a rare but challenging complication with a very high morbidity rate in our reported case. A high index of suspicion is required for early diagnosis and prompt management even if there is no direct encasement of vasculature for a favorable outcome.

This is the first documented case where excision of extra-axial meningioma was done in posterior fossa and vasospasm occurred in anterior fossa signifying the distal nature of vasospasm.

**Keywords:** Cerebral Vasospasm; Vestibular Schwannoma; Posterior Fossa; Extraaxial Tumor; complication Patient Informed Consent: Patient's informed consent was obtained in this study

## Abbreviations

aSAH: Aneurysmal Subarachnoid Hemorrhage; CSF: Cerebrospinal Fluid; CT: Computed Tomography; MCA:

Middle Cerebral Artery; MRC: Medical Research Council; MRI: Magnetic Resonance Imaging; SAH: Subarachnoid Hemorrhage.

## Introduction

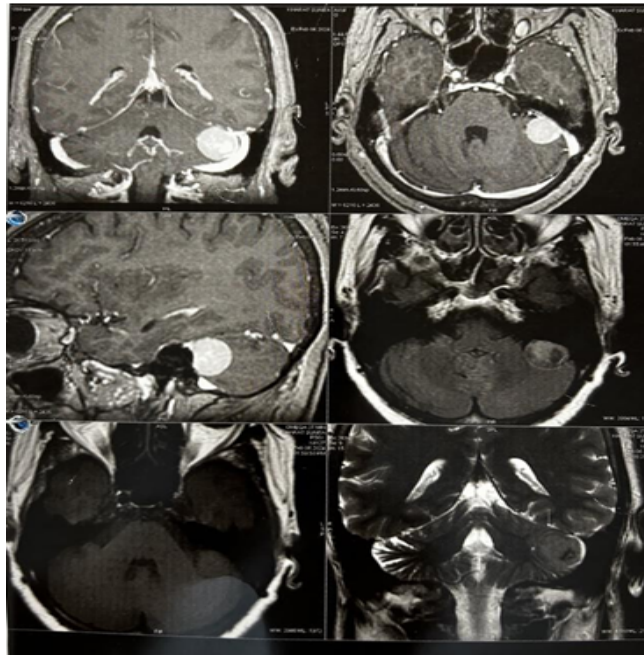
Vasospasm is a dreaded complication of subarachnoid hemorrhage (SAH), which is usually secondary to aneurysm bleeding [1]. It is a rare presentation after tumor resection, especially after excision of a cerebello-pontine angle meningioma, and only a handful of cases have presented with delayed symptomatic vasospasm after tumor resection. We reviewed the literature available on symptomatic vasospasm after tumor resection, especially posterior fossa extraaxial tumors, and were able to identify 14 such cases, including 4 cases of cerebello-pontine angle meningioma. In this article, we describe our experience with an additional case, with the aim of discussing various theories of vasospasm following posterior fossa tumor excision as well as the treatment

strategy that can be provided.

## Illustrative Case

### First Admission

A 55-year-old female with an unremarkable medical history presented to us with a history of progressive left-sided hearing loss for 6 months, imbalance while walking, and headaches for 3 months. On examination, she had severe hearing loss in her left ear. Magnetic resonance imaging (MRI) of the brain with contrast revealed an approximately 3.6 x 3.1 x 3.5-cm left-sided cerebello-pontine angle meningioma exhibiting significant compression of the left cerebellum (Figure 1).



**Figure 1:** 3.6 x 3.1 x 3.5-cm left-sided cerebellopontine-pontine angle meningioma exhibiting significant compression of the left cerebellum.

## Surgery

With the patient placed in right Parkbench position, a left retrosigmoid craniotomy and Total excision of the tumor were performed under continuous facial nerve monitoring. The tumor was soft. There was some bleeding during decompression of the solid part of the tumor. The left sided Transverse sinus was injured accidentally at the end of the procedure causing a small rent at the inferior edge of the sinus. A bipolar cautery was used initially to stop the bleeding but it could not be stopped so a patty was placed over it for hemostasis. The bleeding continued despite this maneuver. Thus, Ligaclips were used to achieve hemostasis

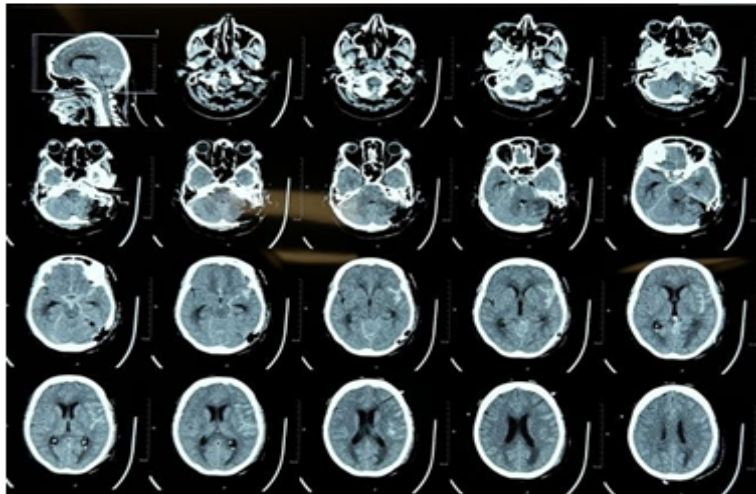
without complete occlusion of the of the sinus. Thereafter, the cerebellum started swelling and to control that the head of the patient was elevated and patient was hyperventilated to reduce the EtCO<sub>2</sub>. The cerebellar swelling subsided after this. There was no hemodynamic instability or any sudden drop in EtCO<sub>2</sub> suggestive of air embolism. The rest of the procedure was uneventful. Total blood loss during the surgery was less than 500 ml.

## Postoperative Course

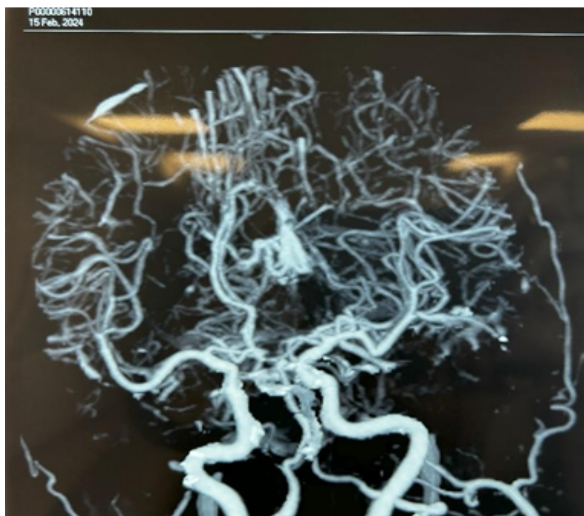
After the operation patient was extubated on table and was completely conscious. There were no signs of any

neurological deficit and was kept for observation in the neurosurgical ICU. On post-operative day 2 (POD) in the evening she became drowsy and had 2 episodes of vomiting, so a CT scan was done revealing hemorrhage in the basal cisterns with mild hydrocephalus with total resection of the tumor (Figure 2). The scan also showed subarachnoid hemorrhage (SAH) along the cerebellar folia and along the left cerebral convexity in left front-temporal region. Extra-axial hemorrhage was also seen along the anterior falx cerebri. Patient also underwent CT Angiography of brain and no angiographic abnormality was detected (Figure 3).

Patient was considered for a VP shunt procedure coming morning (Post-operative day-3), however she improved significantly and became completely conscious and a repeat CT was done, which showed resolution of hydrocephalus. On Post-Operative Day-4 patient was shifted from Neurosurgical ICU to private room. The patient's recovery was uneventful till Post-Operative Day -7, when she became irritable and had signs suggestive of Frontal lobe syndrome. This was accompanied with behavioral changes, loss of inhibition and emotional lability.

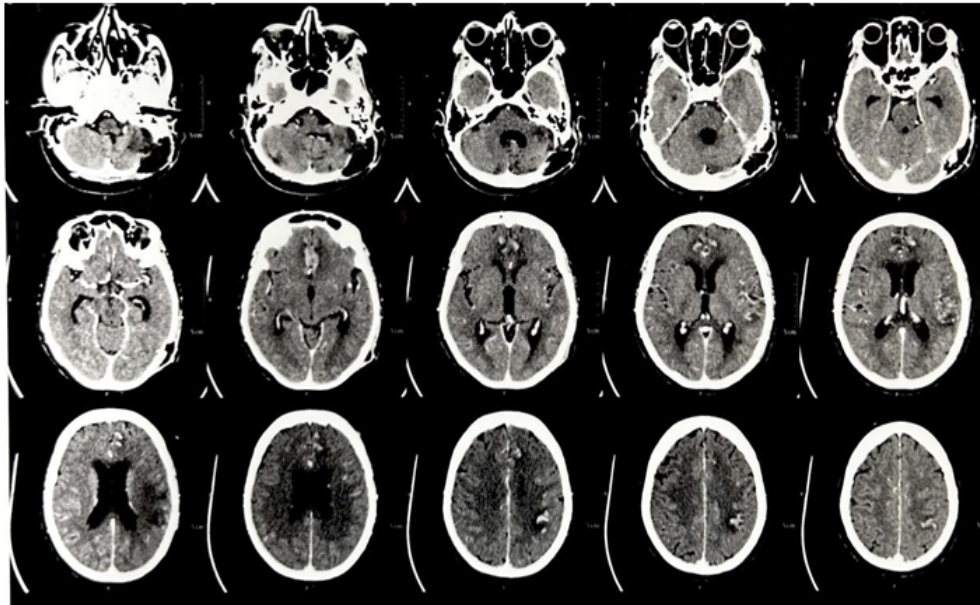


**Figure 2:** Hemorrhage in the basal cisterns with mild hydrocephalus with total resection of the tumor; showed subarachnoid hemorrhage (SAH) along the cerebellar folia and along the left cerebral convexity in left front-temporal region. Extra-axial hemorrhage was also seen along the anterior falx cerebri. (POD2).

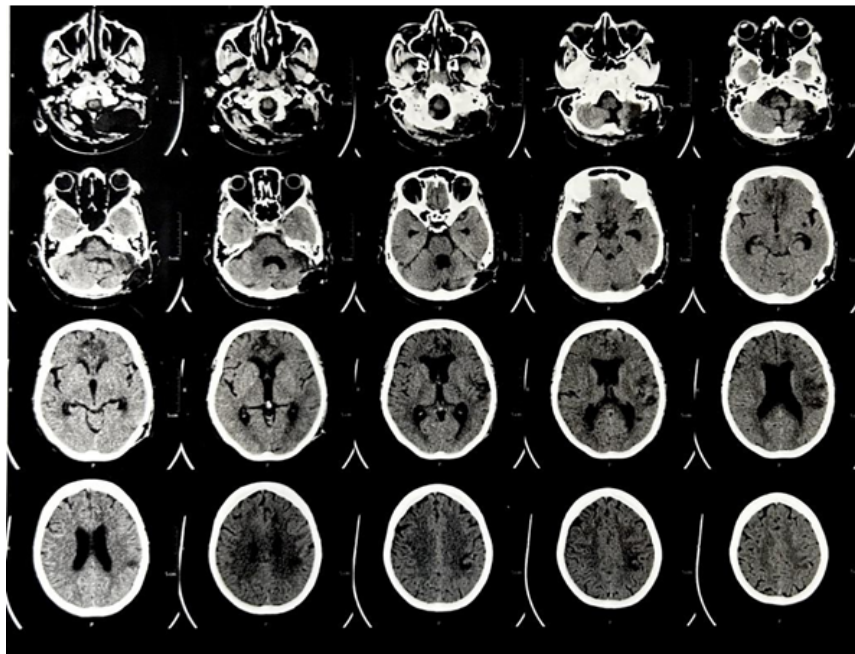


**Figure 3:** CT Angiography of brain and no angiographic abnormality was detected. No evidence of any Aneurysm.

A CT head Plain scan was done to reveal a hypodensity in the bilateral frontal lobes suggestive of ischemia (Figure 4). The scan was discussed with a Neurologist, her symptoms and the radiological picture of bilateral frontal hypodensities grew our suspicion of a bilateral ACA vasospasm. She was started on triple H therapy (Hypertension, hypervolemia and hemodilution) [2], tablet Nimodipine [3] tablet Aspirin, and tablet Atorvastatin via nasogastric tube. Muscle physiotherapy, speech therapy and swallowing therapy were also instituted. CT scanning of the brain on the 9th postoperative day confirmed the disappearance of the previously seen SAH (Figure 5). The patient demonstrated gradual neurological improvement. At the 1-month follow-up, the patient had significant recovery from frontal lobe signs, she was having normal behavior and her MMSE score was 28/30 which had significantly improved. Follow-up MRI of the brain showed decrease in the ischemic zone along with significant clinical improvement in patient's condition. Despite significant improvement in the patient, she was still drowsy and apathic. Patient was discharged for better care at home.



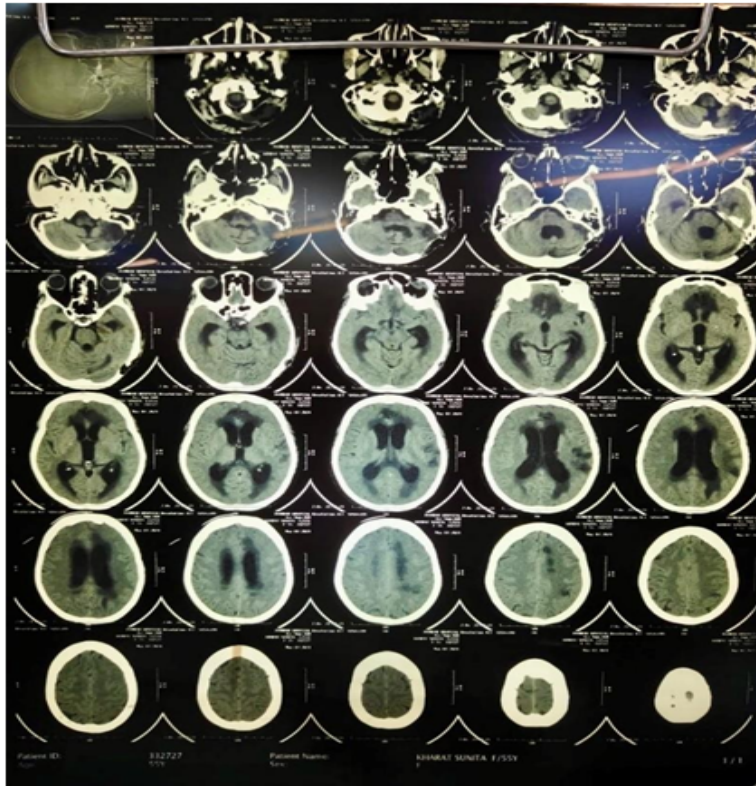
**Figure 4:** CT head (contrast) scan was done to reveal a hypodensity in the bilateral frontal lobes suggestive of ischemia of bilateral frontal lobes.



**Figure 5:** 9<sup>th</sup> postoperative day confirmed the disappearance of the previously seen SAH.

After 3 weeks of discharge, she came back in a state of drowsiness. A CT brain plain was done to reveal a communicating hydrocephalus (Figure 6). A VP shunt was

done immediately. Patient became fully conscious on the next day and recovered well. She resumed her work as before.



**Figure 6:** 6 weeks after surgery, development of communicating hydrocephalus.

POD	Clinical presentation	Radiological findings	Intervention	Outcome
2	Drowsiness and vomiting	CT head (plain)- Mild hydrocephalus with 4th ventricle hemorrhage, SAH left cerebral convexity SDH along anterior Falx Angiogram Normal MR venogram- no evidence of aneurysm	Considered for VP shunt coming morning	-
3	Significant improvement in previous status Fully conscious and oriented	Resolution of hydrocephalus	VP shunt was avoided	-
4	Neurologically stable patient	-	Shifted to Privated room from ICU	-
7	Irritable behavior Loss of Inhibition Emotional lability	Plain CT- bilateral frontal Hypodensity- Ischemia	Suspected vasospasm- Started on- Hyperdynamic therapy, Nimodipine, Aspirin and Atorvastatin. Shifted to Neurological ICU	Proactive treatment
9	Mild improvement in behaviour	Plain CT- disappearance of previous SAH	Continued Hyperdynamic therapy	Mild Improvement

1 month	Significant improvement in behaviour but continues to be apathic. MMSE score 28/30	MRI- mild resolution of ischemic zone.	Patient discharged for home care	
2 months	Came to Emergency in a drowsy state.	CT head plain- Communicating hydrocephalus	VP shunt done	Regained complete consciousness next day
3 months	Fully conscious, walking			Resumed her work.

**Table 1:** Post-operative course Discussion.

## Discussion

Cerebral vasospasm is common after aneurysmal subarachnoid hemorrhage (aSAH). However, it has been documented as a rare complication following intracranial posterior fossa tumor removal. There were only 40 cases found in a 2013 systematic analysis [4]. Since then, more cases have been reported, and there has been an improvement in the understanding of this phenomenon.

## Observations

Vasospasm after intra-axial tumor excision has always been re-ported as bilateral, distant and diffuse. Such an event is rare after extra-axial tumor resection. Pituitary tumor and the sellar area are the most common pathology and location associated with cerebral vasospasm. Only 14 cases of symptomatic cerebral vasospasm have been detected following extra-axial posterior fossa tumor excision [5]. Out of the 5 posterior fossa schwannomas, only 2 cases were confirmed by the authors for intraoperative bleeding as a cause of distant cerebral vasospasm. Other than vestibular schwannomas, 8 cases of posterior fossa extra-axial tumor excisions leading to cerebral vasospasms were identified. Tumors in these cases included 4 meningiomas, epidermoid cyst, medulloblastoma, chordoma, and chondrosarcoma [1,2,6-8] ours is the 5th case of distant vasospasm after Cerebellopontine angle extra-axial Meningioma excision secondary to hemorrhage. Kraysenbuehl, et al. [9] were the first to report a case of vasospasm following tumor resection, which occurred after the excision of a pituitary macroadenoma. Various theories have been proposed for this rare occurrence of vasospasm following tumor excision. The literature indicates the high arterial blood load in the subarachnoid space [10], hypothalamic dysfunction [11], and/or vascular injury or manipulation [1] as the causative factors. Vascular encasement and manipulation are thought to be more pertinent to the excision of extra-axial tumors. Hypothalamic injury is thought to play a major role in the development of vasospasm after the removal of suprasellar

and middle cranial fossa tumors [4]. The release of arterial vasoactive blood products following hemorrhage in the subarachnoid space is thought to irritate local as well as distant vasculature [12]. The direct mechanical trauma to the arterial wall during tumor resection can induce local artery vasoconstriction, which results in an intrinsic myogenic response, upregulated norepinephrine levels or increased cerebrovascular reactivity to norepinephrine and serotonin [13]. Young people have enhanced contractility and elasticity of their arterioles, which can make them more susceptible to direct mechanical stress. In fact, the incidence of symptomatic vasospasm is lower among patients with aSAH who are older [14]. Studies have also implicated direct stimulation of the A2 nucleus or the release of vasoactive substances from the tumor bed, [15-17]. Which may affect the postganglionic trigeminal system, the A2 nucleus and the median eminence and this in turn may lead to cerebral vasospasm, particularly after pituitary tumor and ventral medullary tumor removal [18-20].

Table 2 summarizes the clinical details of 5 reported cases of symptomatic cerebral vasospasm after the excision of posterior fossa schwannoma, amongst them most patients were young, with 4 of 5 being younger than 40 years of age [9,21-23]. However, as in our case, 1 patient with vestibular schwannoma excision was 69 years old, and the author also described the tumor as having a cystic component [23]. Two cases were reported to have undergone a cerebrospinal fluid (CSF) diversion procedure before the tumor excision and in 1 of the cases, the authors suggested a relative paucity of CSF in the cisterns as a predisposing factor for vasospasm [22,23]. Out of 14 cases of posterior fossa extraaxial tumors that led to cerebral vasospasm, perioperative hemorrhage or imaging confirmation of SAH was reported in only 4 cases [7,8,21]. In the remaining 9 cases, although the presence of hemorrhage was not explicit, only 2 had evidence of a lack of hemorrhage. One case reported minor intraoperative bleeding [22], whereas the other presented postoperative MRI showing minimal hemorrhage [2]. The latency between the day of tumor excision and vasospasm ranged from 3 to

14 days, resembling aSAH-related vasospasm. However, in 1 case of petroclival and cavernous meningioma, vasospasm was recorded on the 1st postoperative day, and the author mentioned major vessel encasement of the tumor causing vessel manipulation and vasospasm.1 Similarly, 1 patient had symptom onset at the 3rd day after surgery, where the direct mechanical trauma to the arterial wall during tumor resection was postulated as the cause [13].

Our patient had a moderately sized soft solid tumor and there was indeed some bleeding during decompression

of the tumor and craniotomy. Total blood loss was less than 500ml. The blood seeping into the subarachnoid space due to the lateral position [3] would have been the cause of the documented evidence of postoperative SAH along the cerebellar folia and along the left cerebral convexity in left front-temporal region. The release of vasoactive materials from the tumor might have played a role. Last, the presence of blood in the subarachnoid space after surgery seems to be the most likely causative factor of vasospasm. Prompt initiation of cerebral perfusion-directed hyperdynamic therapy can help in the reversal of vasospasm.

Author	Tumor type	Age	Sex	Post-operative day at Vasospasm Diagnosis	Location of Vasospasm as Identified by Angiogram (unless otherwise specified)	Vascular Manipulation or Tumor Encasement of Vessels Found to Undergo Vasospasm	Intraoperative or Early Postoperative Bleeding as Evidenced by Postoperative CT Imaging or Author Report	Relative Morbidity at Last Follow up
Bejjani, et al. [1]	Lt foramen magnum meningioma	65	F	15	Rt VA & BA.	VBA encased by tumor.	Insufficient information to comment.	Disability
Bejjani, et al. [1]	Rt petro-clivus & cavernous sinus meningioma	57	M	1	Rt MCA (M1 segment).	Tumor encased ICA & VBA bilaterally. ICA narrowing bilaterally	Insufficient information to comment.	Insufficient information to comment.
Cervoni, et al. [7]	Lt anterior, middle & posterior fossae meningioma	51	M	4	Bilateral MCA vasospasm identified using Transcranial doppler.	No comment made by authors.	Copious intraoperative blood loss from cavernous sinus. No further distinction provided by authors.	Disability
Pan, et al. [8]	Lt middle & posterior cranial fossa clinoidal meningioma	53	F	6	Lt ACA & MCA.	Tumor encased lt ICA-MCA & ACA.	Intraoperative ACA perforator bleed requiring clipping. 2cm hematoma removed intraoperatively.	Insufficient information to comment.
						Extensive manipulation was involved.		
Current case	Lt CPA Meningioma	55	F	7	Bilateral Anterior cerebral arteries	No encasement	Intra-op bleeding from left sigmoid sinus, controlled with partial clipping of sinus	Resumed her work, no disability

**Table 2:** clinical details of 5 reported cases of symptomatic cerebral vasospasm after the excision of posterior fossa schwannoma.

## Conclusion

Blood in the subarachnoid space can contribute to symptomatic vasospasm. This is proven time and again by similar phenomenon seen in Aneurysmal Subarachnoid hemorrhage. This knowledge can be applied in our case and it can be postulated that the degree and extent of vasospasm can be affected by following factors:

- Presence and degree of bleeding prior to vasospasm
- Duration of bleeding during surgery
- Distribution of blood in the subarachnoid space and
- Release of arterial vasoactive blood products, stimulation of A2 nucleus and median eminence.

Recognizing this relationship and inducing efforts to minimize above factors may reduce the possibility of post-operative vasospasm.

It is also critical to keep a vigilant eye on the post-operative clinical progress of the patient to diagnose vasospasm in its initial stage, so as to initiate the therapy proactively [24].

To achieve the expected results even in established vasospasm, we must lower our threshold for initiating Hyperdynamic therapy for distal vasospasm in operated cases of posterior fossa meningiomas.

Despite all the speculations, the cause of SAH in supratentorial compartment following a posterior fossa extra-axial tumor resection was unclear. There was minimal bleeding at operative site except accidental injury to left transverse sinus, which was controlled immediately. This is the first documented case where excision of extra-axial meningioma was done in posterior fossa and vasospasm occurred in anterior fossa signifying the distal nature of vasospasm [25].

## References

1. Bejjani GK, Sekhar LN, Yost AM, Bank WO, Wright DC (1999) Vasospasm after cranial base tumor resection: pathogenesis, diagnosis, and therapy. *SurgNeurol* 52(6): 577-584.
2. Rao VK, Haridas A, Nguyen TT, Lulla R, Wainwright MS, et al. (2013) Symptomatic cerebral vasospasm following resection of a medulloblastoma in a child. *Neurocrit Care* 18(1): 84-88.
3. Pasricha P, Alay V, Khandhar, Basant K, Misra (2024) Delayed symptomatic cerebral vasospasm following vestibular schwannoma resection: illustrative case. *J Neurosurg Case Lessons* 7(9): CASE23745.
4. Alotaibi NM, Lanzino G (2013) Cerebral vasospasm following tumor resection. *J Neurointerv Surg* 5(5): 413-418.
5. Hiwase AD, Kalyanasundaram K, Bak VS, Laden SM, Ovenden CD, et al. (2022) Symptomatic cerebral vasospasm following posterior fossa hemangioblastoma resection: illustrative case. *J Neurosurg Case Lessons* 3(13): CASE21492.
6. Aw D, Aldwaik MA, Taylor TR, Gaynor C (2010) Intracranial vasospasm with delayed ischaemic deficit following epidermoid cyst resection. *Br J Radiol* 83(991): e135-e137.
7. Cervoni L, Salvati M, Santoro A (1996) Vasospasm following tumor removal: report of 5 cases. *Ital J Neurol Sci* 17(4): 291-294.
8. Pan J, Levitt MR, Ferreira M, Sekhar LN (2021) Symptomatic cerebral vasospasm following resection of skull base tumors: case report and literature review. *Clin Neurol Neurosurg* 202: 106482.
9. Kravenbuehl H (1960) A contribution to the problem of cerebral angiospastic insult. *Schweiz Med Wochenschr* 90: 961-965.
10. Fisher CM, Kistler JP, Davis JM (1980) Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. *Neurosurgery* 6(1): 1-9.
11. Wilkins RH (1975) Hypothalamic dysfunction and intracranial arterial spasms. *Surg Neurol* 4(5): 472-480.
12. Allen GS, Gross CJ, French LA, Chou SN (1976) Cerebral arterial spasm. Part5: in vitro contractile activity of vasoactive agents including human CSF on human basilar and anterior cerebral arteries. *J Neuro surg* 44(5): 594-600.
13. Qi J, Zhang L, Jia W, Zhang J, Wu Z (2015) Diffuse cerebral vasospasm after resection of schwannoma: a case report. *Neuropsychiatry Dis Treat* 11: 317-320.
14. Torbey MT, Hauser TK, Bhardwaj A, Williams MA, Ulatowski JA (2001) Effect of age on cerebral blood flow velocity and incidence of vasospasm after aneurysmal subarachnoid haemorrhage. *Stroke* 32(9): 2005-2011.
15. Mawk JR (1983) Vasospasm after pituitary surgery. *J Neurosurg* 58(6): 972.
16. Mawk JR, Ausman JI, Erickson DL, Maxwell RE (1979) Vasospasm following transcranial removal of large pituitary adenomas. Report of three cases. *J Neurosurg* 50(2): 229-232.
17. Wilson JL, Feild JR (1974) The production of intracranial vascular spasm by hypothalamic extract. *J Neurosurg*



- 40(4): 473-479.
18. Chang SD, Yap OW, Adler JR (1999) Symptomatic vasospasm after resection of a suprasellar pilocytic astrocytoma: case report and possible pathogenesis. *Surg Neurol* 51(5): 521-527.
  19. Aoki N, Oritano TC, Mefty OA (1995) Vasospasm after resection of skull base tumors. *Acta Neurochir (Wien)* 132(1-3): 53-58.
  20. Atalay B, Bolay H, Dalkara T, Soylemezoglu F, Oge K, et al. (2002) Transcorneal stimulation of trigeminal nerve afferents to increase cerebral blood flow in rats with cerebral vasospasm: a noninvasive method to activate the trigeminovascular reflex. *J Neurosurg* 97(5): 1179-1183.
  21. Almeida GMD, Bianco E, Souza AS (1985) Vasospasm after acoustic neuroma removal. *SurgNeurol* 23(1): 38-40.
  22. Afshari FT, Fitzgerald JJ, Higgins JN, Garnett MR, Fernandes HM, et al. (2014) Diffuse cerebral vasospasm following resection of a hypoglossal schwannoma in a child. *Br J Neurosurg* 28(4): 541-543.
  23. Roux PDL, Haglund MM, Mayberg MR, Winn HR (1991) Symptomatic cerebral vasospasm following tumor resection: report of two cases. *Surg Neurol* 36(1): 25-31.
  24. Brian LH, Nerissa UK, Hanjani MD, Yi CSH, Flores SC, et al. (2023) A guideline from American Heart Association/American Stroke Association. 54(7).
  25. Tyree HK (2023) Pharmacologic options for prevention and Management of cerebral vasospasm in Aneurysmal Subarachnoid Hemorrhage. *Hosp. Pharm* 48(5): S2-S9.