

ILLUSTRATIVE ORIGINAL SURGICAL VIDEO

## Microsurgical resection of a huge pontine cavernoma with bulbar disturbance

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saparovnurgeldimd@gmail.com**Received:** 14 August 2025; **Accepted:** 05 September 2025; **Published:** 30 September 2025

Pontine cavernous malformations are rare vascular lesions that carry a significant risk of hemorrhage and neurological deficits because of their location within the brainstem. We describe the case of a young patient who presented with bulbar symptoms secondary to a pontine cavernoma. Alongside the clinical details, we review similar cases reported in the literature, including presentations such as trigeminal neuralgia and other cranial nerve syndromes arising from pontine lesions. The report outlines the surgical approach, intraoperative techniques, and postoperative management in detail. Advanced neuromonitoring and careful selection of safe entry zones were critical to the operative plan. The patient experienced a favorable recovery, with improvements noted during rehabilitation and on follow-up imaging. In discussing this case, we highlight the balance between achieving maximal safe resection, often subtotal in brainstem surgery, and minimizing the risk of rehemorrhage while preserving neurological function.

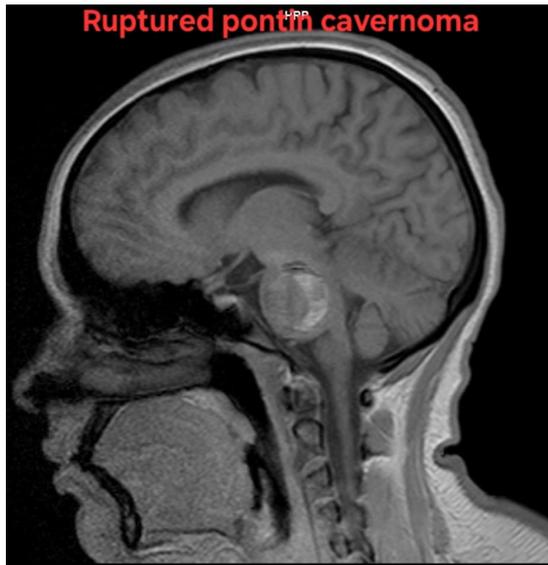
**Keywords:** pontine cavernoma, brainstem ruptured cavernoma, safe entry zone, intra-op neuromonitoring, brainstem surgery, retrosigmoid craniotomy, peritrigeminal area

### Introduction

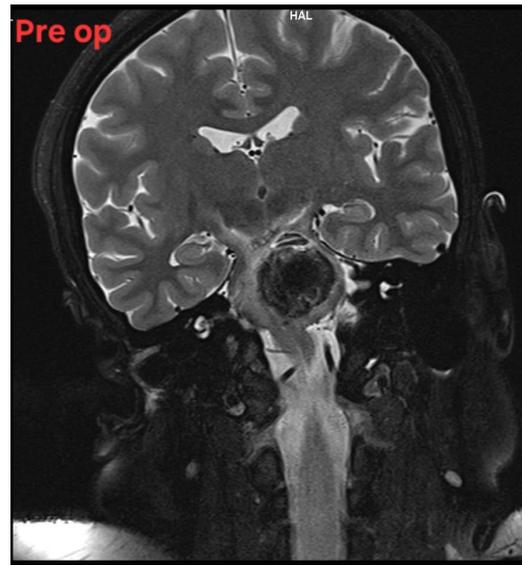
Cerebral cavernous malformations (cavernomas) are slow-flow vascular anomalies, occurring in approximately 0.4–0.8% of the population (1). They commonly present in the 2nd to 5th decades of life. Supratentorial lesions often cause seizures or focal deficits, whereas infratentorial lesions (e.g., brainstem) typically manifest with ataxia, cranial nerve signs, or sudden hemorrhage. Approximately 20% of cavernomas are located in the brainstem; pontine cavernomas are the most frequent brainstem subtype. Brainstem cavernomas have an especially high propensity to bleed: meta-analyses and cohort studies report 5-year hemorrhage risks on the order of 8% if asymptomatic, rising to >30% if there has been a prior bleed or focal deficit. One prospective series found an annual hemorrhage rate of ~7.0% for brainstem cavernomas (and even higher if multiple prior hemorrhages

were present) (2). Infratentorial and deep location, young age, female gender, and prior hemorrhage are known risk factors for rebleeding. The natural history of untreated brainstem cavernomas is thus aggressive, and repeated bleeds can lead to permanent deficits due to disruption of critical fiber tracts. Current consensus recommends individualized management, often favoring surgical intervention for lesions that are accessible and have bled or caused neurologic deficits.

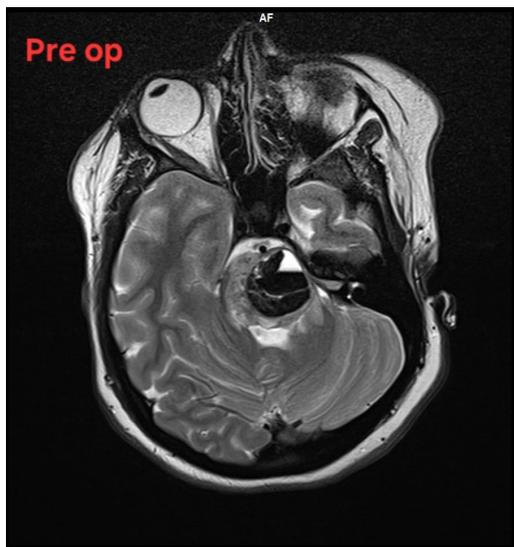
Early reports of pontine cavernoma surgery showed substantial technical challenge and nontrivial morbidity, but surgical outcomes have improved with modern microsurgical techniques. Large series suggest that complete resection is achieved in the majority of cases, with long-term functional status preserved or improved in over 80% of patients. However, new postoperative deficits are common (often transient), underscoring the difficulty of brainstem surgery. These factors, together with the inherent risk of hemorrhage,



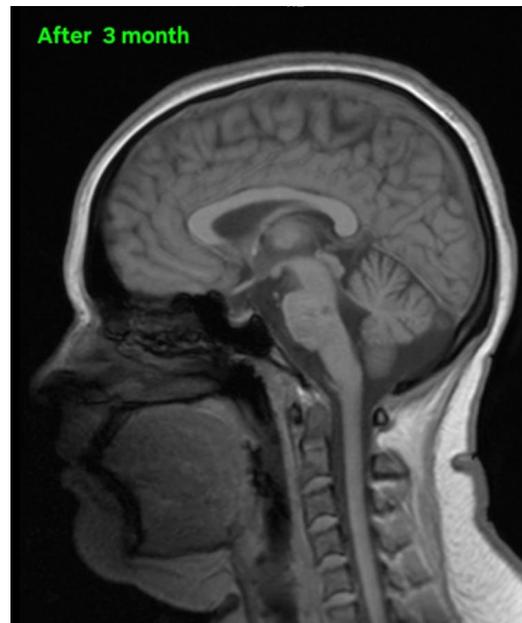
**FIGURE 1** | Pre operatively T1 MRI on sagittal plane, hemorrhagic lesion with hyperintensity component.



**FIGURE 3** | Pre operatively T2 MRI on coronal plane, size 2.1\*1.8\*1.7 hemorrhagic lesion in the left dorsal pons.



**FIGURE 2** | Pre operatively T2 MRI on axial plane, size 2.1\*1.8\*1.7 lesion with hypointense hemosiderin rim.

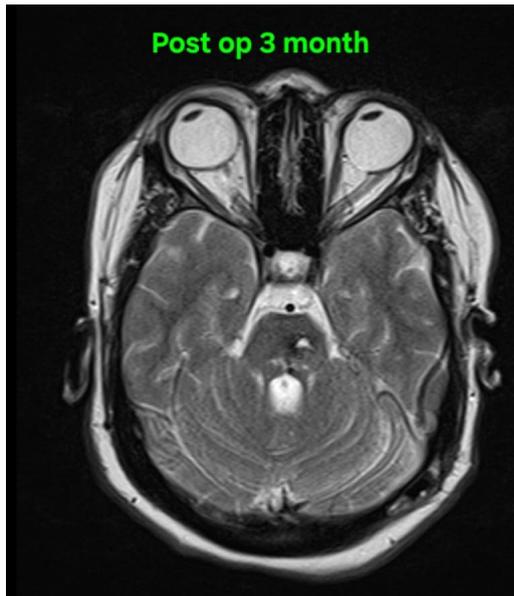


**FIGURE 4** | Post operatively after 3 month T1 MRI on sagittal plane.

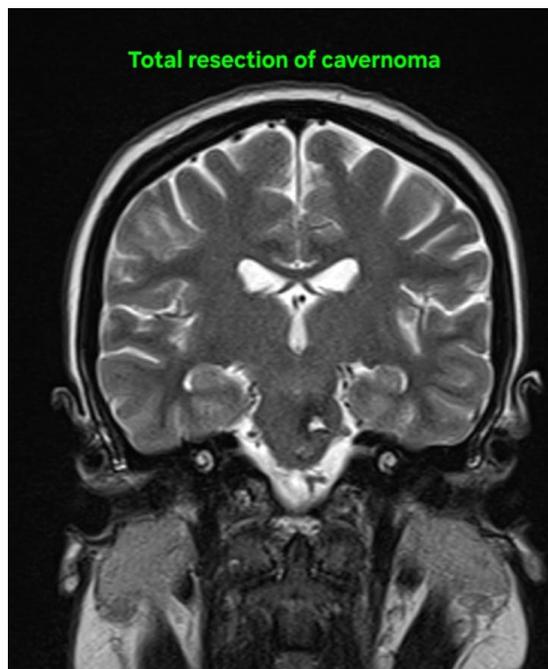
guide the decision-making process when considering resection, radiosurgery, or conservative observation for pontine lesions.

Reports in the literature further illustrate the diverse clinical presentations of pontine cavernomas and emphasize the need for an individualized approach to treatment. For example, Cenzato et al. reported a patient with intractable trigeminal neuralgia (V1–V2 distribution facial pain) caused by a left lateral pontine cavernous angioma. Surgical excision of the cavernoma resolved the neuralgia without new deficits. Other reports describe complex brainstem syndromes from pontine cavernomas: a recent case of “eight-and-a-half syndrome” (a horizontal gaze palsy plus ipsilateral facial palsy) was traced to a pontine cavernoma (3–5). In a

pediatric series of lateral pontine cavernomas, transient trigeminal hypesthesia/dysesthesia occurred in 2 of 8 patients after resection. Conversely, cavernoma hemorrhage has even produced Millard-Gubler syndrome (ipsilateral VI and VII palsies with contralateral hemiparesis) (6–8). Together, these cases demonstrate that pontine cavernomas can present with sensory symptoms (facial numbness or pain) or other cranial nerve deficits and that surgical management can alleviate symptoms. We therefore review these analogous cases to contextualize our patient’s presentation.



**FIGURE 5** | Post operatively after 3 month T2 MRI on axial plane.



**FIGURE 6** | Post operatively after 3 month T2 MRI on coronal plane.

## Case Presentation

A previously healthy, 30-year-old man presented with a two-month history of progressive numbness involving the left trigeminal V2 (maxillary) and V3 (mandibular) dermatomes, accompanied by occasional mild dizziness (9–12).

Neurological examination:

- Sensory loss in the left trigeminal V2-V3 dermatomes
- Mild truncal ataxia

- No facial weakness, limb weakness, or dysarthria
- Left side hemiparesis
- Visual decrease
- Diplopia

Imaging Findings: MRI demonstrated a  $2.1 \times 1.8 \times 1.7$  cm lesion in the left dorsal pons with mixed signal intensities ('popcorn' appearance) and hypointense hemosiderin rim on T2-weighted imaging (Figures 1 to 3).

The patient consented to surgery. His preoperative mRS (modified Rankin Scale) was 2 (slight disability). Routine pre-op workup was normal. Intraoperative neurophysiological baselines (motor evoked potentials, somatosensory evoked potentials, brainstem auditory evoked responses, and cranial nerve EMG) were obtained. Preoperative planning used neuro-navigation to map the lesion's relationship to the fourth ventricle and cranial nerve entry zones.

## Surgical Management

The patient was placed in the lateral (park bench) position with the head fixed in a Mayfield head clamp and slightly flexed and rotated to the right.

### Incision and Exposure

A left retrosigmoid/suboccipital approach was used. A curvilinear incision was made posterior to the mastoid. Subcutaneous tissues and musculature were dissected in layers to expose the occipital bone and posterior edge of the mastoid. A left retrosigmoid craniectomy was performed, exposing the transverse-sigmoid sinus junction. The dura was opened in a curvilinear fashion based on the sigmoid sinus and reflected anteriorly.

### Microsurgical Approach

Cerebrospinal fluid was released from the cisterna magna for brain relaxation. Microsurgical dissection was carried out using the operating microscope. The cerebellum was gently retracted medially to expose the cerebellopontine angle. The VII-VIII nerve complex and lower cranial nerves were identified and preserved (Video 1).

**VIDEO 1** | <https://youtu.be/MWGEWHFUKwE>

## Lesion Identification

Using neuronavigation and intraoperative neurophysiological monitoring, the lateral pontine surface was identified at the peritrigeminal zone, which served as the safe entry corridor (13, 14).

## Resection of Cavernoma

Using a small pial incision was made at the predetermined safe entry point. The cavernoma was carefully dissected circumferentially and removed piecemeal until the appearance of the gliotic margin of the brainstem white matter, taking care to preserve the surrounding parenchyma. The associated hemosiderin rim and clot were removed as safely as possible without injuring eloquent tissue. Hemostasis was secured with gentle bipolar coagulation and hemostatic agents. Some abnormal perilesional veins were coagulated and divided. Following piecemeal removal, panoramic inspection was performed to confirm the absence of residual cavernoma (Figures 4 to 6).

## Closure

The cavity was inspected for residual lesions or bleeding. The dura was closed in a watertight fashion with a dural graft as needed. The bone defect was left (craniectomy) and fixed with mini plates. Muscle and subcutaneous tissue were reapproximated, and the skin was closed in layers.

## Postoperative Care

The patient was extubated and transferred to the neuro-ICU for close monitoring with neurological assessment and imaging follow-up. The patient awoke without new deficits. MRI confirmed complete resection. At the 3-week follow-up, sensory function improved, and no recurrent bleeding was observed.

## Discussion

Pontine cavernomas, once symptomatic, carry high rebleeding rates. Early surgery is indicated in selected cases where lesions are surgically accessible. The peritrigeminal zone provides a safe lateral entry corridor to pontine lesions, minimizing injury to corticospinal tracts and vital nuclei. In this case, isolated trigeminal sensory loss served as the key presenting feature, highlighting the importance of maintaining a high index of suspicion when cranial nerve

symptoms persist without a clear cause. Our experience demonstrates that meticulous microsurgical planning, combined with intraoperative neuromonitoring, can enable complete resection while preserving neurological function.

## Conclusion

Pontine cavernomas may initially present with subtle sensory disturbances. When carefully selected, patients can benefit from microsurgical resection through well-defined brainstem safe entry zones, achieving durable outcomes with preservation of neurological function.

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