Journal of Surgery and Surgical Specialities
Volume 1, Issue 3, September 2025; pp: 75-80
eISSN: 2455-2860 DOI: 10.65129/surgical.v1i3.127

RESEARCH ARTICLE

A Study on Clinical Profile and Outcome of Intracranial Cavernoma Patients in Tertiary Care Hospital

V. Velarasan*, S. Balamurugan and R. Raghavendran

Department of Neurosurgery, Madras Medical College, Chennai - 600003, Tamil Nadu, India; velarasan94@gmail.com

Abstract

Aim: Intracranial cavernous malformations are congenital benign hamartomas of the brain. They are more common than generally suspected. They are low flow, low pressure lesions, and their hemorrhage typically displaces and compresses adjacent tissue rather than destroying it. The clinical picture is a result of the hemorrhage in relation to the location and volume of the hemorrhage. We retrospectively reviewed our cases with Intracranial Cavernomas and presented our results. **Methods:** A retrospective analysis of 17 patients with Intracranial cavernous malformations admitted at Institute of Neurosurgery, RGGGH, over a period of 4 years from 2021 till now. The data was obtained from review of medical records and available imaging studies. **Results:** After studying the 17 intracranial cavernoma patients, we found that 12 presented with seizures, 14 of them were supratentorial, 1 was in brainstem. All underwent microsurgical excision and 15 of them improved without deficit. **Conclusion:** In this paper, we evaluated the demographic features, location, clinical features, surgical technique and postoperative follow up of intracranial cavernomas. Microsurgical excision is the gold standard treatment of cerebral cavernous malformations.

Keywords: Cavernoma, Excision, Hemorrhage, Seizures

1. Introduction

Intracranial cavernous malformations, sometimes referred to as cavernous angiomas or cavernous hemangiomas, are congenital lesions involving the brain. They are usually low-flow, low- pressure lesions and can bleed and compress and displace tissue around which a bleed occurs without destroying it. Symptoms related to any interval bleeding depend on the location and amount of the bleed. Cavernomas may be either single sporadic lesions, or groups of related lesions in families, a common occurrence in medical practice. While older beliefs postulated a developmental history, it is now generally accepted that a cavernoma can develop de novo, especially after radiation therapy. Cavernomas aren't fixed, they can enlarge or regress over time, but rarely remain the same size. Cavernomas can also be small or very large, with growth typically occurring due to recurrent small bleeds or the filling of previously occluded channels with clot. These malformations make up 5–13 percent of CNS vascular malformations. Most are located above the tentorium, but can also occur in the brainstem, basal ganglia or the spinal cord. Many cases have multiple lesions. Histologically, these lesions consist of randomly positioned spaces filled with blood, with no smooth muscle or elastic tissue, and no associated feeding arteries, draining veins, or neural connective tissue. Since the introduction of MRI, cavernous malformations are diagnosed more often. Appropriate management of these lesions is therefore crucial. Herewith presenting a case series of surgically managed 17 intra cranial cavernomas with respect to demographic features, varied clinical presentations, radiological imaging and outcome.

*Author for correspondence

Article Received on: 01-05-2025 Revised on: 15-07-2025 Accepted on: 05-09-2025

2. Aim and Objectives

The aim of this study is to analyze the demography, clinical presentation, radiological investigations and surgical outcomes of intracranial cavernous malformations.

3. Review of Literature

Cerebral Cavernous Malformations (CCMs) are clusters of thin-walled vascular channels lacking smooth muscle and elastic tissue, predisposing them to recurrent microhemorrhages and gliosis. They constitute 5–15 % of intracranial vascular malformations and may present with seizures, focal neurological deficits, or symptomatic hemorrhage. While many cases remain asymptomatic, the risk of clinical events is influenced by lesion location—particularly the brainstem—and prior hemorrhage[6]. Familial CCMs exhibit autosomal dominant inheritance involving CCM1, CCM2, or CCM3 mutations, often presenting with multiple lesions and greater clinical penetrance[2]. The molecular mechanisms, emphasizing RhoA–ROCK pathway dysregulation, endothelial barrier dysfunction, and emerging roles of MAP3K3 and PIK3CA mutations in sporadic lesions. These discoveries support new therapeutic avenues targeting PI3K/mTOR signaling and β-blockers such as propranolol, which early trials suggest may reduce symptomatic hemorrhage. Seizures constitute a major manifestation of supratentorial CCMs. Pure lesionectomy demonstrates high seizure-freedom rates, reported between 70% and 90%[9,10]. Early surgery improves outcomes, particularly when epilepsy duration is short or when hemosiderin-laden tissue is completely removed[4]. Management of brainstem lesions remains challenging due to eloquent location, though large series show favorable outcomes with meticulous microsurgical techniques[5]. Diagnostic advances, particularly MRI with SWI and T2*GRE sequences, have improved detection of small or multiple lesions[3]. Conservative management is generally preferred for asymptomatic lesions, whereas microsurgical excision or, in selected cases, stereotactic radiosurgery is recommended for symptomatic CCMs[7,8]. Overall, recent literature reflects substantial progress in understanding CCM genetics, molecular pathways, imaging, and evolving therapeutic strategies, supporting increasingly individualized patient management.

4. Material and Methods

This is a retrospective study of 17 patients who underwent surgery between January 2021 to July 2025 admitted in Institute of Neurosurgery, Madras Medical College/RGGGH. Patient demographics, clinical presentation, types and surgical outcomes is analyzed. All patients in this study underwent MRI (MRI 1.5 TESLA, plain).

5. Results (Including Observations)

5.1 Demographics

In this study 8 of them were female and 9 were male as shown in Table 1. Most common age group was between 11-20 years (35.2%) 6 cases as shown in Table 2.

Almost, every patient presented with hemorrhage and 70% had seizure as the first symptom.

There was no side predilection; with right sided lesions 60% and left sided lesions 40%.

Supratentorial location is most common accounting about 84%.

5.2 Case Reports

CASE 1

16-year-old female, presented with seizure; Microsurgical excision done. Patient improved, with no post op deficit and no further seizure episode. MRI shows T1 heterointense lesion in left frontal lobe with various stages of hemorrhage (Popcorn appearance).

CASE 2

6-year-old female, child presented with gaze palsy. Microsurgical excision done. Patient improved, with no post op deficit. MRI shows well defined irregular lobulated predominantly T1 hyper intense, T2 hetero intense with T2 hyperintense rim in dorsal aspect of pons causing compression of fourth ventricle.

Table 1. Sex distribution

Gender	Number (%)
Male	9(53%)
Female	8(47%)

Table 2. Age distribution

Age groups (years)	Number (%)
0-10	4 (23.5%)
11-20	6 (35.2%)
21-30	1 (5.8%)
31-40	3 (17.6%)
41-50	2 (11.7%)
51-60	1(5.8%)

Table 3. Clinical presentation

SYMPTOM	PERCENTAGE
SEIZURE	12(70.5%)
HEADACHE	5(29.4%)
WEAKNESS	2(11.7%)
GAZE PALSY	2(11.7%)

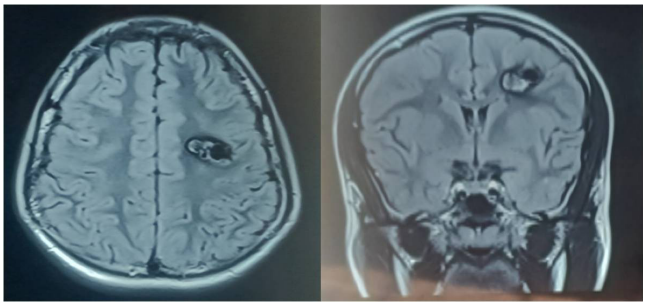

Figure 1. MRI brain showing lesion in left frontal lobe.

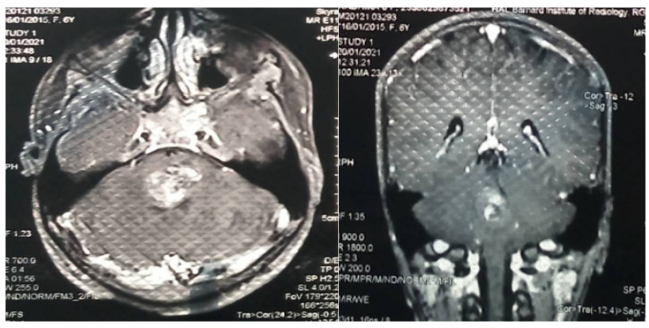

Figure 2. MRI brain with contrast showing lesion in brainstem

Table 4. Frequency of side of lesion

SIDE	PERCENTAGE
RIGHT	9(60%)
LEFT	6(40%)

Table 5. Frequency of location of lesion

SITE/LOBE	PERCENTAGE
FRONTAL	8(47%)
TEMPORAL	4(23.5%)
PARIETAL	1(5.8%)
OCCIPITAL	1(5.8%)
PONTINE	2(11.7%)
CEREBELLUM	1(5.8%)

Table 6. Frequency of outcome

OUTCOME	PERCENTAGE
IMPROVED	15(88%)
NO IMPROVEMENT	1(6%)
EXPIRED	1(6%)

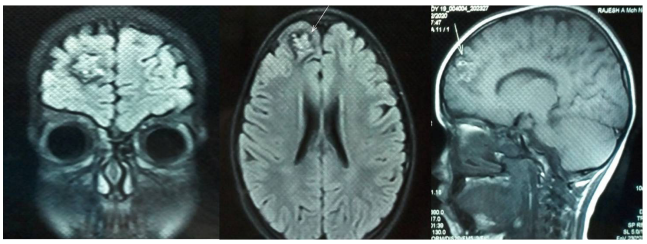

Figure 3. MRI brain showing lesion in right frontal lobe.

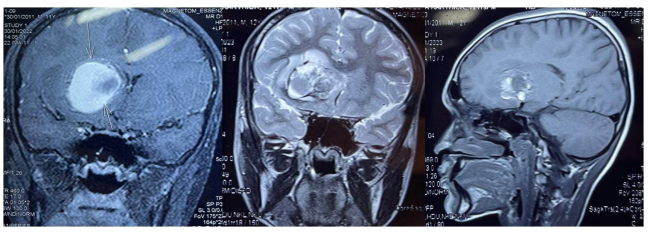

Figure 4. MRI brain showing lesion in right GC region with popcorn appearance.

Table 7. Zabramski radiological Classification

Type	MR Signal Characteristics	Pathological Characateristics	Risk of Hemorrhage
IA	**T1:** hyperintense **T2:** hyperintense or hypointense extending through at least one wall of the hypointense rim that surrounds the lesion, focal edema* may be present	"Overt" extralesional focus of hemorrhage extending outside the lesion capsule.	Almost all lesions are symptomatic. High risk of recurrent symptomatic hemorrhage of up to 60%/yr for brainstem lesions (25%-30%/yr).
IB	**T1:** hyperintense **T2**: hyperintense or hypointense surrounded by a hypointense rim.	Subacute focus of intralesional hemorrhage	Risk of symptomatic hemorrhage related to presentation and location. Higher for symptomatic lesions in the brainstem and basal ganglia (5%-10%/yr). Lower in asymptomatic lesions (0.5%-1%/yr).
II	**T1:** reticulated mixed signal core **T2**: reticulated mixed signal core surrounded by a hypointense rim	Loculated areas of hemorrhage and thrombosis of varying age surrounded by gliotic, hemosiderin-stained brain; in large lesions, areas of calcification may be seen. (Popcorn appearance)	Risk of symptomatic hemorrhage related to presentation. In symptomatic patients risk of recurrent hemorrhage 4%-5%/yr. Low risk for asymptomatic lesions (0.5%-1%/yr).
III	**T1:** isointense or hypointense **T2:** hypointense with hypointense rim that magnifies the size of lesion **GE:** hypointense with greater magnification than T2	Chronic resolved hemorrhage with hemosiderin staining within and around the lesion.	Rarely symptomatic. Lesions have a low risk of hemorrhage (<0.5%/yr).
IV	**T1:** poorly seen or not visualized at all **T2**: poorly seen or not visualized at all **GE:** punctate hypointense lesions	Two lesions in the category have been pathologically documented to be telangiectasias.	Never symptomatic. Very low risk of hemorrhage.

Table 8. Frequency based on Zabramski classification

Types	Number(%)
IA	6(35)
IB	6(35)
II	5(30)

CASE 3

8-year-old male child, presented with seizure. Microsurgical excision done. Patient improved, with no post op deficit and no further seizure episode. MRI shows heterogenous T1/ T2 and FLAIR signal intense lesion with T2 hypo intense blooming on GRE in right frontal region.

CASE 4

16-year-old male, presented with complaints of headache, vomiting on and off for years. Well preserved, no deficit. MRI showed heterogeneously enhancing mixed intensity lesion in right anterior GC region and frontal periventricular region with various stages of hemorrhage (popcorn appearance). Patient given option of surgery or observation and chose to go for a wait and watch approach.

CASE 5

40-year-old female, presented with seizure and headache. Microsurgical excision done. Patient symptomatically improved, with no further seizure episode and no post op deficit. MRI shows ill- defined T2 hetero intense lesion in right frontal lobe with surrounding edema with low ADC values and partial diffusion restriction.

6. Discussion

All patients were symptomatic pre operatively, underwent pre operative evaluation with cerebral MRI. One patient was managed conservatively; however, he

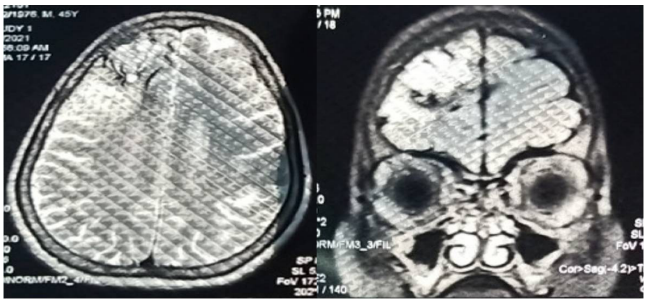

Figure 5. MRI brain showing lesion in right frontal lobe.

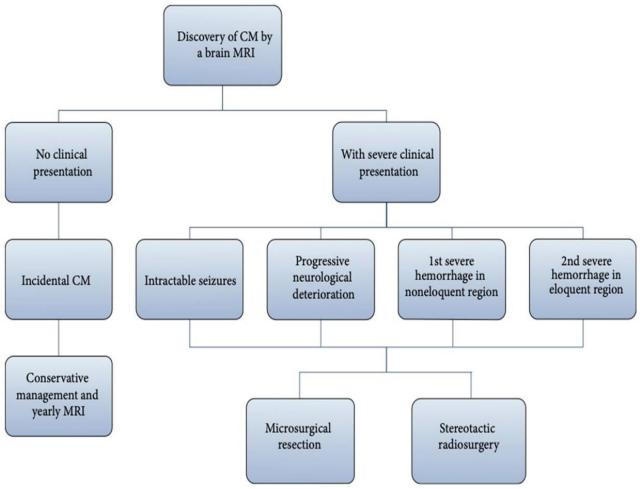

Figure 6. Flow chart depicting the management protocol.

developed, new onset seizure during the observation period. Hence all 17 patients, including the one managed conservatively, underwent microsurgical excision of the cavernous. Headache improved in all patients. 2 patients had seizures in the immediate post-operative period, had no further episode, gaze palsy in 2 patients with pontine cavernous, improved post operatively. One among the 2 patients with hemiparesis did not show any improvement after surgery and one patient expired in the post-operative period. As a first-line treatment for patients with CCM and a single episode, the use of antiepileptic drugs is preferred rather than going straight to surgery. Antiepileptic medications were found to be 47%-60% effective in controlling newly diagnosed cavernoma-related epilepsy. Regular follow-up is suggested. Early surgery should be indicated for patients with a greater risk of bleeding or who cannot comply with AED therapy. A complete removal of the lesion is necessary to prevent recurrent haemorrhagic events. The hemosiderin ring should also be removed if the surgery is for seizure. After surgery, re-bleeding occurs in 40% of cavernoma remnants therefore, a postoperative MRI within 72 hours is encouraged. If there are remnants, surgical intervention is required as early as possible. The complication risk associated with surgical intervention is location dependent of posed location of the CM lesion. One study showed 97% of lobar, 87.5% of cerebellar, 64% of brainstem improved. With the help of intraoperative neuronavigational techniques, diffusion-tensor and functional MR imaging, neurosurgeons are able to resect deep-seated lesions in eloquent areas of the brain with minimal neurologic deficit associated with low mortality and morbidity rates. The precise location of the lesion in the brainstem, and the experience of the neurosurgeon is the key to limiting the extent of excision. The risks of complications and the appearance of neurological deficits postoperatively. Moreover, the natural history of cavernomas requires further investigation because it is important when evaluating the methods of treatment applied.

7. Summary and Conclusion

Microsurgical excision is the gold standard treatment of cerebral cavernous malformations. Proper patient selection, proper diagnosis, meticulous surgical technique combined with proper post-operative care and punctual follow up, all lead to reduce morbidity and mortality. Goal of surgical intervention is to achieve the total resection of the lesion, without any neurological impairment.

8. References

1. Youmans JR, Winn HR. Youmans neurological surgery. 8th ed. Philadelphia: Elsevier; 2022.
2. Zabramski JM, Wascher TM, Spetzler RF, *et al.* The natural history of familial cavernous malformations: results of an ongoing study. J Neurosurg. 1994; 80(3):422-32. https://doi.org/10.3171/jns.1994.80.3.0422 PMID: 8113854.
3. Mouchtouris N, Chalouhi N, Chitale A, Starke RM, Tjoumakaris SI, Rosenwasser RH, *et al.* Management of cerebral cavernous malformations: from diagnosis to treatment. Sci World J. 2015; 2015:808314. https://doi.

org/10.1155/2015/808314 PMID: 25629087 PMCID: PMC4300037.
4. Khallaf M, Abdelrahman M. Supratentorial cavernoma and epilepsy: experience with 23 cases and literature review. Surg Neurol Int. 2019; 10:117. https://doi.org/10.25259/SNI-178-2019 PMID: 31528453 PMCID: PMC6744744.
5. Wang CC, Liu A, Zhang JT, Sun B, Zhao YL. Surgical management of brainstem cavernous malformations: report of 137 cases. Surg Neurol. 2003; 59(6):444-54. https://doi.org/10.1016/S0090-3019(03)00187-3 PMID: 12826334
6. Kondziolka D, Lunsford LD, Kestle JRW. The natural history of cerebral cavernous malformations. J Neurosurg. 1995; 83(5):820-4. https://doi.org/10.3171/jns.1995.83.5.0820 PMID: 7472549.
7. Chalouhi N, Dumont AS, Randazzo C, *et al*. Management of incidentally discovered intracranial vascular abnormalities. Neurosurg Focus. 2011; 31(6):E1. https://doi.org/10.3171/2011.9.FOCUS11200 PMID: 22133178.
8. D'Angelo VA, De Bonis C, Amoroso R, Cali A, D'Agruma L, Guarnieri V, *et al*. Supratentorial cerebral cavernous malformations: clinical, surgical, and genetic involvement. Neurosurg Focus. 2006; 21(1):E9. https://doi.org/10.3171/foc.2006.21.1.10 PMID: 16859262.
9. Englot DJ, Han SJ, Lawton MT, Chang EF. Predictors of seizure freedom in the surgical treatment of supratentorial cavernous malformations. J Neurosurg. 2011; 115:1169-74. https://doi.org/10.3171/2011.7.JNS11536 PMID: 21819194.
10. Ferroli P, Casazza M, Marras C, Mendola C, Franzini A, Broggi G, *et al*. Cerebral cavernomas and seizures: a retrospective study on 163 patients who underwent pure lesionectomy. Neurol Sci. 2006; 26:390-4. https://doi.org/10.1007/s10072-006-0521-2 PMID: 16601930.