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Case Report

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A Rare Case of Brain Arteriovenous Malformation Presenting as Isolated Intraventricular Hemorrhage

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ABSTRACT

Background: Cerebral arteriovenous malformations (cerebral AVMs) are congenital vascular anomalies characterised by direct arteriovenous shunting through a nidus without an intervening capillary bed. Hemorrhage is the most common presentation; however, isolated intraventricular hemorrhage (IVH) without associated intraparenchymal bleed is rare and may obscure the underlying vascular etiology. Early identification is crucial to prevent rebleeding and guide definitive management.

Methods: We report a case of a 50-year-old female with acute neurological symptoms, diagnosed with intraventricular haemorrhage on non-contrast CT (EC-SDUAHER/R&D/CEC/SDUMC-F/71/NF/-2025-26) at the Department of Radiology, Sri Devraj Urs Medical College, Kolar. Hemorrhage involved bilateral lateral, third, and fourth ventricles with ventriculomegaly and periventricular CSF seepage. CTA of the brain and neck was performed to assess vascular abnormality.

Results: CTA revealed a 2.4 × 1.2 cm arteriovenous nidus in the left parietal lobe, supplied by M2–M4 segments of the left middle cerebral artery and drained via an enlarged superior anastomotic vein into the superior sagittal sinus with early arterial opacification. No aneurysm or stenosis was seen. The lesion was classified as Spetzler–Martin Grade I AVM, confirming cerebral AVM presenting as isolated IVH.

Conclusion: Cerebral AVM should be considered in cases of spontaneous isolated intraventricular hemorrhage. CTA plays a critical role in early detection and anatomical characterisation, facilitating timely referral for digital subtraction angiography and therapeutic planning.

Key-words: Cerebral arteriovenous malformation; Isolated intraventricular hemorrhage; CT angiography; Spetzler–Martin grade; Non-traumatic IVH; Cerebral vascular malformation

INTRODUCTION

Arteriovenous malformations (AVMs) are vascular anomalies characterised by direct arteriovenous shunting that bypasses the normal capillary bed, resulting in abnormal hemodynamic stress.

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These lesions predispose patients to serious complications, with intracerebral hemorrhage (ICH) being the most severe manifestation. Spontaneous hemorrhage due to AVM rupture is a major cause of neurological morbidity and mortality, often presenting with acute neurological deficits. The resultant increase in intracranial pressure can lead to significant secondary brain injury [1].

The management of ruptured AVMs requires a multidisciplinary approach involving neurosurgeons, interventional radiologists, and neuroanesthesiologists to ensure comprehensive care. Collaborative decision-

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making enables the selection of appropriate therapeutic strategies, including endovascular embolisation, microsurgical resection, and stereotactic radiosurgery [2]. Treatment modality selection depends on several factors, such as AVM size, location, vascular anatomy, and the patient's clinical stability. Preoperative stabilisation typically includes a waiting period of 2–6 weeks following hemorrhage to allow hematoma resolution and partial neurological recovery, thereby reducing surgical risks [3].

Advancements in neuroimaging and interventional techniques have significantly improved AVM management. Digital subtraction angiography (DSA) remains the gold standard for diagnosis and preoperative planning, providing detailed visualisation of the nidus, feeding arteries, and venous drainage patterns. The introduction of modern embolic agents, such as the Onyx liquid embolic system, has enhanced the efficacy of endovascular embolisation by facilitating effective nidus occlusion and reducing intraoperative bleeding risks [4]. Intraoperative neuromonitoring further improves surgical safety by aiding preservation of eloquent brain regions during nidus resection.

Despite advances in management, many patients experience post-hemorrhagic morbidity and persistent neurological deficits, with an ongoing risk of re-rupture and recurrent hemorrhage. Therefore, consistent long-term follow-up is essential to monitor for residual lesions and potential complications. Variability in outcomes underscores the importance of standardised protocols and evidence-based guidelines to optimise management strategies and improve prognosis [5].

This case report aims to describe a rare presentation of a cerebral arteriovenous malformation manifesting as isolated intraventricular hemorrhage, highlighting the diagnostic challenges, radiological findings, and clinical outcomes to aid early etiological identification, appropriate management, and prevention of rebleeding.

CASE PRESENTATION

A 50-year-old female with no known comorbidities presented to the emergency department with a sudden onset of severe headache. The headache was described as diffuse, intense, and unlike any prior episodes. It was associated with nausea and multiple episodes of non-projectile vomiting. There was no history of preceding trauma, fever, seizure activity, loss of consciousness, focal neurological deficit, or visual disturbance. She had no prior history of hypertension, diabetes mellitus, stroke, anticoagulant use, or known vascular malformations. On arrival, the patient was hemodynamically stable. Her blood pressure was within normal limits, and there were no features suggestive of hypertensive emergency. Neurological examination revealed that she was disoriented to time, place, and person. Her Glasgow Coma Scale (GCS) score was 8/15. Cranial nerve examination was normal, with no significant papilledema on fundoscopy. Motor and sensory examinations did not reveal any focal neurological deficits. There were no signs of meningeal irritation. Given the acute onset of severe headache, disorientation and vomiting, an intracranial hemorrhage was suspected, and urgent neuroimaging was performed.

A non-contrast computed tomography (CT) scan of the brain demonstrated acute intraventricular hemorrhage involving the body and occipital horns of the bilateral lateral ventricles, as well as the third and fourth ventricles, as shown in Fig. 1. There was associated dilatation of the bilateral lateral ventricles and third ventricle, consistent with obstructive hydrocephalus. Periventricular hypodensities were noted around the left occipital horn, suggestive of transependymal cerebrospinal fluid (CSF) seepage. Mild effacement of cortical sulci in the bilateral cerebral hemispheres was observed, consistent with early raised intracranial pressure. Importantly, there was no associated intraparenchymal hematoma identified; there was a small intraparenchymal mildly hyperdense area noted in the left parietal lobe. In the absence of trauma and given the pattern of hemorrhage, a vascular etiology such as aneurysm or arteriovenous malformation (AVM) was suspected. The patient was admitted for close neurological monitoring, head elevation, and supportive management, including antiemetics and measures to prevent secondary brain injury.

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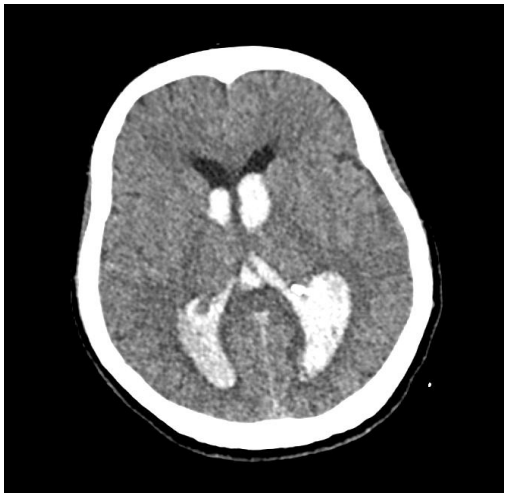

Fig. 1: Axia non-contrast CT of the brain reveals acute intraventricular hemorrhage with hyperdense blood products filling the bilateral lateral ventricles, involving the frontal and occipital horns. Mild diffuse sulcal effacement is present. Periventricular hypodensity suggests transependymal CSF permeation.

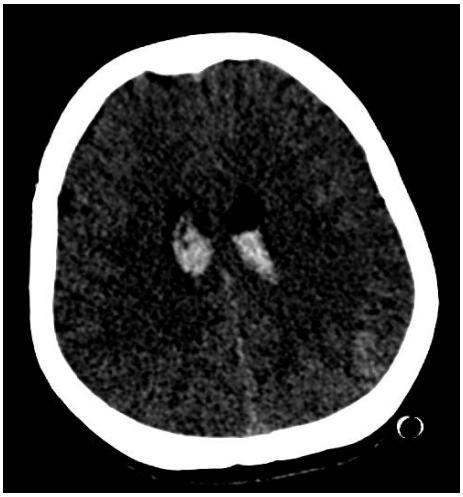

Fig. 2: Non-contrast CT Brain — Axial non-contrast CT image demonstrates a subtly hyperdense focus in the left parietal lobe, raising suspicion for an underlying arteriovenous malformation. There is a persistent hyperdense intraventricular hemorrhage within the bodies of the bilateral lateral ventricles.

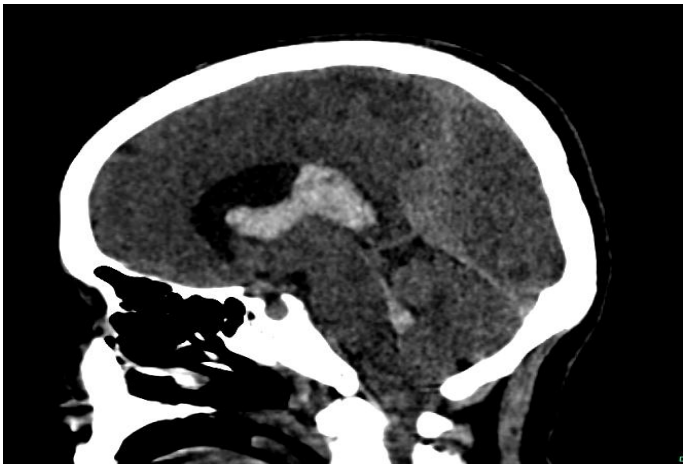

Fig. 3: Sagittal Reformatted Non-contrast CT Brain — Sagittal reformatted non-contrast CT image demonstrates hyperdense intraventricular hemorrhage involving the third ventricle and the body of the lateral ventricle, with inferior extension into the fourth ventricle. Findings consistent with extensive intraventricular hemorrhage.

To further evaluate for an underlying vascular abnormality, CT angiography (CTA) of the brain and neck was performed using a pressure injector. CTA revealed abnormal dilatation of the M2, M3, and M4 segments of the left middle cerebral artery (MCA). These arterial feeders were seen converging into a compact vascular nidus measuring ~2.4 × 1.2 cm, located in the left parietal lobe. The nidus demonstrated early venous drainage through an enlarged superficial cerebral vein, identified as the superior anastomotic vein. This vein showed early arterial-phase enhancement and drained into the superior sagittal sinus.

Additionally, a few trans-cerebral veins were noted, which were also dilated and communicated with the enlarged superficial cerebral vein and the ependymal surface of the left lateral ventricle, suggesting a possible anatomic pathway for intraventricular hemorrhage. The bilateral internal carotid arteries were normal in course and calibre with no evidence of stenosis or aneurysmal dilatation. The right middle cerebral artery, anterior cerebral arteries, vertebral arteries, basilar artery, and posterior cerebral arteries were all unremarkable. No saccular aneurysm or other vascular malformation was identified.

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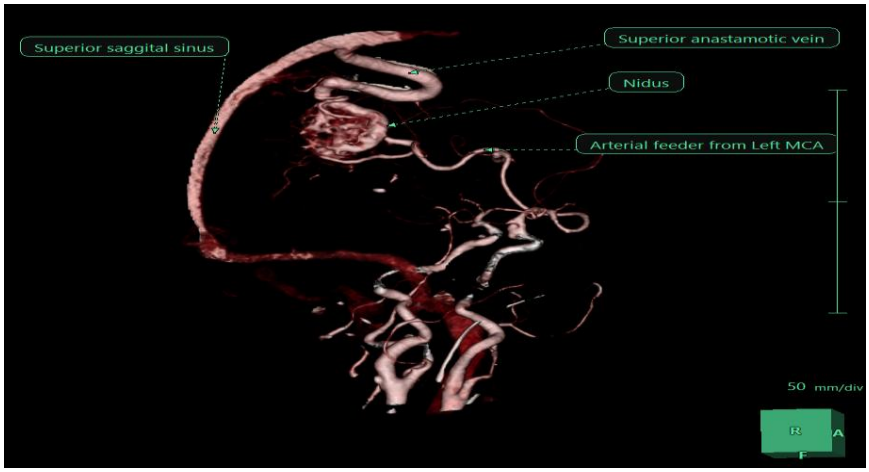

Fig. 4: 3D Volume-rendered CT Angiography — A 3D volume-rendered image demonstrates an arteriovenous malformation in the left cerebral hemisphere, supplied by branches of the left middle cerebral artery. The nidus shows venous drainage via a prominent superior anastomotic vein, ultimately draining into the superior sagittal sinus.

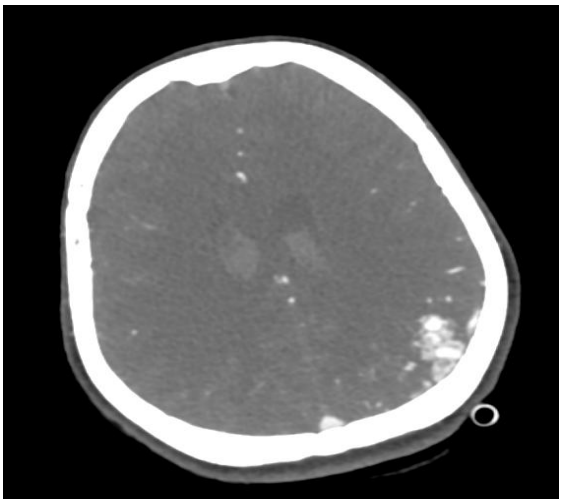

Fig. 5: Axial Contrast-Enhanced CT Angiography — Axial CTA image demonstrates a compact tangle of enhancing serpiginous vascular channels in the left parietal region, consistent with an arteriovenous malformation. The nidus is supplied by arterial feeders from the left middle cerebral artery, with early opacification of draining cortical veins, indicative of arteriovenous shunting.

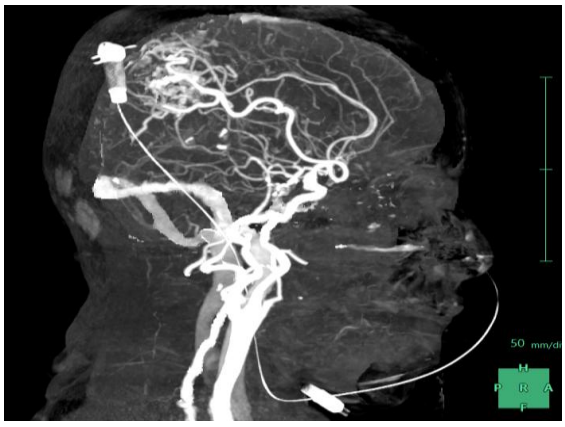

Fig. 6: Maximum Intensity Projection CT Angiography — MIP CTA image demonstrates a compact vascular nidus in the left parietal lobe, supplied by branches of the left middle cerebral artery, with prominent venous drainage into the superior sagittal sinus, consistent with an arteriovenous malformation.

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Based on imaging characteristics, the lesion was diagnosed as a cerebral arteriovenous malformation located in the left parietal lobe. According to the Spetzler-Martin grading system, the AVM was classified as Grade I, given its small size (<3 cm), superficial venous drainage, and location in a non-eloquent brain region. The final radiological diagnosis was a left parietal cerebral arteriovenous malformation presenting with isolated intraventricular hemorrhage. Notably, there was no associated intraparenchymal hemorrhage or aneurysm, making this an uncommon presentation of AVM rupture.

Following the diagnosis, the patient was managed in a monitored setting with close neurological observation. Blood pressure was carefully controlled within normal limits to reduce the risk of rebleeding while maintaining adequate cerebral perfusion. Serial neurological assessments were performed to detect any deterioration. Given the presence of hydrocephalus and intraventricular hemorrhage, the patient was closely monitored for signs of increased intracranial pressure. However, as she remained neurologically stable with preserved consciousness and no progressive ventricular enlargement on follow-up imaging, invasive CSF diversion was not immediately required.

In view of the identified AVM, digital subtraction angiography (DSA) was recommended for detailed angioarchitectural evaluation and to plan definitive management. Treatment options discussed in a multidisciplinary neurovascular team meeting included microsurgical resection, endovascular embolisation, and stereotactic radiosurgery. Considering the low Spetzler-Martin grade (Grade I), small nidus size, and superficial location, microsurgical resection was considered a favourable option with a high likelihood of complete obliteration and low operative risk. The patient and her family were counselled regarding the nature of the lesion, the risk of rebleeding, and available treatment modalities. After stabilisation of the acute hemorrhagic event, definitive treatment planning was initiated.

The case highlights the importance of prompt vascular imaging in patients presenting with non-traumatic intraventricular hemorrhage, even in the absence of parenchymal hematoma or aneurysm. Early identification of an underlying AVM allows timely intervention and reduces the risk of catastrophic rebleeding.

DISCUSSION

Arteriovenous malformations (AVMs) of the brain are uncommon congenital vascular anomalies characterised by a complex nidus of abnormal arteriovenous shunting without an intervening capillary bed. Their estimated prevalence in the general population ranges between 10 and 18 per 100,000 individuals, with an annual hemorrhage risk of approximately 2–4% [6]. Hemorrhage remains the most frequent and clinically significant presentation, particularly in younger patients. However, AVMs most commonly manifest with intraparenchymal hemorrhage, often accompanied by subarachnoid or intraventricular extension. Isolated intraventricular hemorrhage (IVH) without associated parenchymal bleeding, as observed in the present case, is distinctly rare and diagnostically challenging. Intraventricular hemorrhage accounts for a small proportion of spontaneous intracranial hemorrhages in adults. When IVH occurs without trauma or adjacent intraparenchymal hematoma, the differential diagnosis typically includes aneurysmal rupture, vascular malformations, neoplasms, coagulopathy, or hypertension-related deep hemorrhage extending into the ventricular system [7]. Among vascular causes, ruptured aneurysms—particularly of the anterior communicating artery or distal anterior cerebral artery—are more commonly implicated than AVMs [8].

The present case is notable because the AVM presented solely with intraventricular bleeding, without detectable intraparenchymal hematoma. This pattern suggests that hemorrhage may have originated from fragile periventricular draining veins or small intranidal vessels adjacent to the ependymal surface. The identification of trans-cerebral venous channels communicating with the ependyma of the lateral ventricle supports this mechanism. Similar cases described in the literature propose that deeply located venous drainage or periventricular extension of the nidus increases the likelihood of ventricular rupture [9]. Although IVH secondary to AVM rupture is recognised, isolated IVH remains uncommon, and its rarity can delay identification of the underlying vascular pathology. This underscores the importance of early vascular imaging in cases of spontaneous IVH, even when the initial non-contrast CT does not reveal a parenchymal focus.

Prompt identification of an underlying vascular lesion is essential in spontaneous intracranial hemorrhage. Digital subtraction angiography (DSA) remains the gold standard

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for diagnosing AVMs due to its superior spatial and temporal resolution [10]. However, CT angiography (CTA) has emerged as a highly sensitive, rapid, noninvasive alternative in the acute setting. Studies have demonstrated that CTA has high sensitivity and specificity for detecting AVMs larger than 2 cm, particularly when arterial feeders and early venous drainage are clearly visualised [11].

In the present case, CTA effectively identified a compact left parietal nidus supplied by dilated M2–M4 branches of the middle cerebral artery with early venous drainage into the superior sagittal sinus. The absence of an aneurysm or arterial stenosis was clearly documented. These findings highlight the diagnostic utility of CTA as an initial screening modality in emergency settings, especially when rapid decision-making is required. Nevertheless, DSA remains indispensable for detailed angioarchitectural assessment and therapeutic planning.

The AVM in this patient was classified as Spetzler-Martin Grade I, based on its small size (<3 cm), superficial venous drainage, and location in a non-eloquent cortical region [12]. The Spetzler-Martin grading system remains a cornerstone in surgical decision-making, as it correlates strongly with operative risk and outcomes. Low-grade AVMs (Grades I and II) are generally associated with favourable surgical outcomes and high rates of complete resection with minimal morbidity [13]. The clinical significance of identifying a low-grade AVM lies in its therapeutic implications. Early definitive treatment significantly reduces the lifetime risk of rebleeding, which is particularly relevant in younger patients or those with hemorrhagic presentation. Hemorrhage at presentation is itself considered a risk factor for subsequent rebleeding, further justifying timely intervention [14].

Pathophysiological Insights- The mechanism underlying isolated IVH in AVM rupture likely relates to venous hypertension and rupture of fragile periventricular draining veins. Increased intranidal pressure, particularly in the presence of outflow restriction or high-flow shunting, can predispose to vessel wall failure [15]. The presence of early venous drainage on CTA further underscores the lesion's high-flow nature. Additionally, communication between draining veins and the ventricular ependyma may facilitate direct intraventricular bleeding without substantial parenchymal disruption. This case therefore reinforces the concept that the angioarchitectural characteristics of an AVM—rather than size alone—may influence hemorrhage pattern.

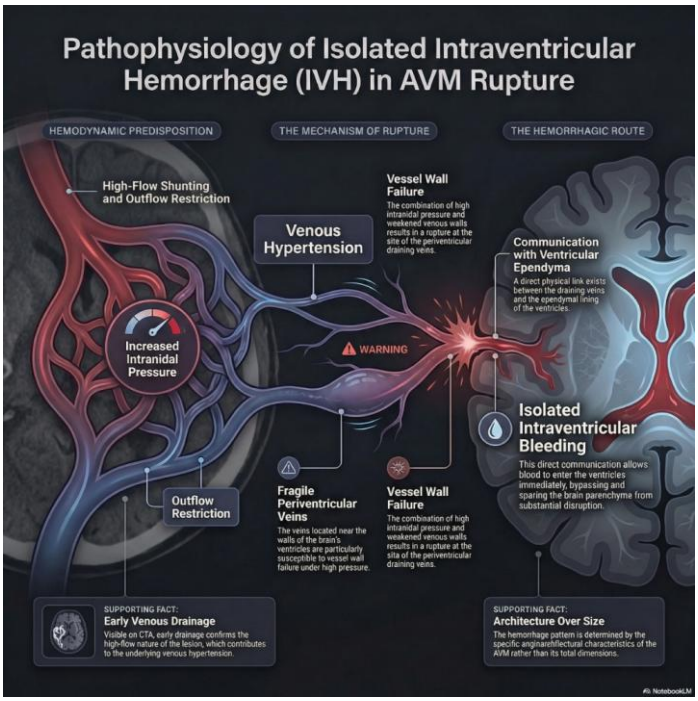

Fig. 7: Schematic Illustration of Pathophysiology of Isolated Intraventricular Hemorrhage in AVM Rupture

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Conceptual diagram depicting the hemodynamic and structural mechanisms underlying isolated intraventricular hemorrhage secondary to arteriovenous malformation rupture. High-flow arteriovenous shunting with outflow restriction leads to elevated intranidal pressure and venous hypertension, predisposing to rupture of fragile periventricular draining veins. Direct communication between these veins and the ventricular ependyma facilitates preferential bleeding into the ventricular system, resulting in isolated intraventricular hemorrhage with relative sparing of the adjacent brain parenchyma. Early venous drainage and angioarchitectural factors are highlighted as key contributors to this hemorrhagic pattern.

CONCLUSIONS

This case highlights a rare but clinically significant presentation of cerebral AVM as isolated intraventricular haemorrhage. It emphasizes the need for thorough vascular evaluation in spontaneous IVH, even in the absence of parenchymal hematoma. Early recognition using CTA, followed by confirmatory DSA, enables timely therapeutic planning. The low Spetzler-Martin grade in this case carries favourable prognostic implications, reinforcing the importance of grading systems in guiding management. Ultimately, this report contributes to the growing body of literature underscoring the diverse and sometimes atypical presentations of cerebral arteriovenous malformations.

CONTRIBUTION OF AUTHORS

Research concept– Vaibhav Krishna M
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Materials– Anne Surya Kanth, Doddala Vamsi Venkat
Data collection– Jeeva Harini, Praveen P
Data analysis and interpretation– Harini Bopaiah, Guru Yogendra M
Literature search– Vaibhav Krishna M
Writing article– Vaibhav Krishna M, Harini Bopaiah
Critical review– Guru Yogendra M
Article editing– Guru Yogendra M
Final approval– Guru Yogendra M, Vaibhav Krishna M

REFERENCES

[1] Samaniego EA, Dabus G, Meyers PM, Kan PT, Frösen J, et al. Most promising approaches to improve brain AVM management: ARISE I consensus recommendation. Stroke, 2024; 55(5): 1449–63.
[2] Stone L, Colliander R, LoPresti MA, Shaibani A, Lam S. Microsurgical resection of a ruptured intraventricular arteriovenous malformation in a neonate: considerations in management. Illustrative case. J Neurosurg Case Lessons, 2023; 6(15): CASE23323.
[3] Lee BB, Do YS, Yakes W, Kim DI, Mattassi R, Hyon WS. Management of arteriovenous malformations: a multidisciplinary approach. J Vasc Surg., 2004; 39(3): 590–600.
[4] Fukuda K, Majumdar M, Masoud H, Nguyen T, Honarmand A, et al. Multicenter assessment of morbidity associated with cerebral arteriovenous malformation hemorrhages. J Neurointerv Surg., 2017; 9(7): 664–68.
[5] Sohail R, Bashir Q, Kanwal S, Ali MI. Cerebral arteriovenous malformations during pregnancy: a management dilemma. BMJ Case Rep., 2019; 12(11): e228759.
[6] Al-Shahi R, Bhattacharya JJ, Currie DG, et al. Prospective, population-based detection of intracranial vascular malformations. Lancet, 2003; 362: 1262–67.
[7] Darby DG, Donnan GA, Saling MM, et al. Primary intraventricular hemorrhage: clinical and neuroimaging features. Stroke, 1998; 19: 1094–100.
[8] van Gijn J, Kerr RS, Rinkel GJ. Subarachnoid haemorrhage. Lancet, 2007; 369: 306–18.
[9] da Costa L, Wallace MC, ter Brugge KG, et al. The natural history and predictive features of hemorrhage from brain arteriovenous malformations. Stroke, 2009; 40: 100–05.
[10] Willinsky RA, Taylor SM, ter Brugge K, et al. Neurologic complications of cerebral angiography. Radiol., 2003; 227: 522–28.
[11] Menke J, Larsen J, Kallenberg K. Diagnosing cerebral arteriovenous malformations by CT angiography: systematic review and meta-analysis. Neuroradiol., 2010; 52: 109–16.
[12] Lawton MT, Kim H, McCulloch CE, Mikhak B, Young WL. A supplementary grading scale for selecting patients with brain arteriovenous malformations for surgery. Neurosurg., 2010; 66(4): 702–13.
[13] Lawton MT, Young WL. Brain arteriovenous malformations. N Engl J Med., 2015; 372: 2014–24.

doi: 10.21276/SSR-IIJLS.2026.12.2.32

[14]Hernesniemi JA, Dashti R, Juvela S, et al. Natural history of brain AVMs: a long-term follow-up study. Stroke, 2008; 39: 1996–2001.

[15]Hashimoto T, Meng H, Young WL. Intracranial arteriovenous malformations: pathobiology and translational research. Stroke, 2006; 37: 263–71.