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Original Research Paper

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ANAESTHETIC CHALLENGES IN A CASE OF INTRACRANIAL AVM EXCISION

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ABSTRACT

Background: Intracranial arteriovenous malformations (AVMs) are congenital vascular anomalies involving abnormal high-flow arteriovenous shunts. Their management, especially surgical excision, poses significant anaesthetic challenges due to the risk of haemorrhage, rupture, and perioperative cerebral ischemia. ***Case Presentation:*** A 32-year-old female presented with seizures and later developed sudden-onset left hemiparesis following a complicated embolization. Imaging revealed a right temporal AVM with a nidus measuring 23×13 mm, fed by the right posterior cerebral artery and draining into the right transverse sinus. The patient was scheduled for elective surgical excision. ***Conclusion:*** This case emphasizes the role of individualized, neurophysiologically informed anaesthetic strategies in a case of AVM excision.

KEYWORDS : Intracranial arteriovenous malformation, elective craniotomy , sevoflurane, mannitol normocapnia, controlled hypotension

INTRODUCTION

Intracranial arteriovenous malformations (AVMs) are congenital vascular anomalies characterized by a tangled network of arteries and veins directly connected without an intervening capillary bed. This abnormal communication results in high-flow, low-resistance shunting of blood, which can predispose surrounding brain tissue to ischemia and increases the risk of rupture and haemorrhage.

The incidence of intracranial AVMs is estimated to be approximately 1 in 100,000 people per year, with a prevalence of 0.1% in the general population. AVMs are often diagnosed between the ages of 20 and 40 years, and there is a slight male predominance with a male-to- female ratio of approximately 1.3:1. However, presentations in females are not uncommon, especially when associated with symptomatic events.

The aetiology of AVMs is believed to be developmental, arising from disruptions in vascular formation between the 3rd and 8th week of gestation. While most AVMs are congenital, some studies suggest they may also result from abnormal angiogenesis or failure of vascular remodelling in utero. In the present case, the AVM was likely congenital in origin, unmasked during adulthood by neurological symptoms, as commonly seen in such cases.

Clinical presentation of AVMs can vary based on size, location, and venous drainage patterns. Common symptoms include seizures, headache, neurological deficits, and intracranial haemorrhage. In our patient, the AVM initially presented with seizures, followed by sudden-onset hemiparesis and facial deviation—likely due to either micro-haemorrhage or post-embolization ischemic insult-correlating well with documented manifestations of AVM-related complications.

Case Report

A 32-year-old female presented with a history of seizure onset in October 2023 and was started on tablet levetiracetam. The patient then underwent AVM embolization, which was uneventful, following which she was advised to continue anti-convulsive medication. In December 2024, patient developed sudden onset paralysis of the left upper and lower limb with deviation of the angle of mouth. She was then immediately taken to the Cath lab from casualty for the embolization of the AVM. The embolization of the AVM was eventful, due to which the patient developed neurological deficits, with power in the left upper limb 0/5 and left lower limb 1/5, while right-sided motor strength remained intact with slight deviation of the angle of the mouth. The patient was shifted to the ICU for further management, where she was continued on anti-convulsive medication and inj. mannitol.

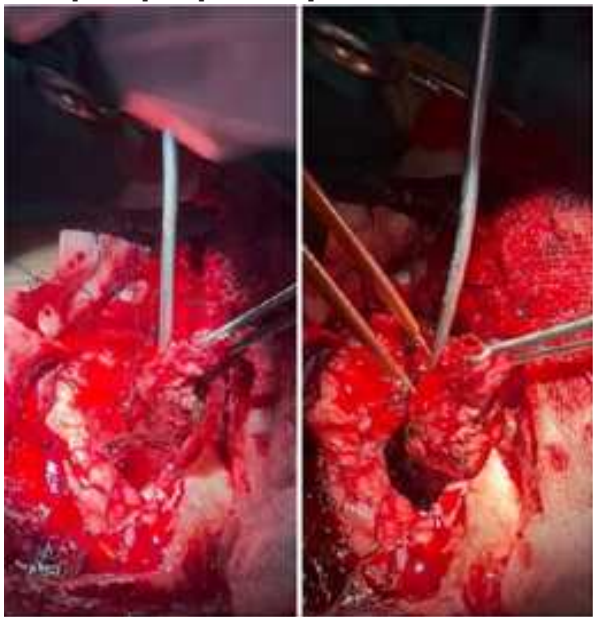
A preoperative brain angiogram revealed a right temporal AVM with post-coiling status, nidus measuring 23x13 mm, predominantly fed by the right posterior cerebral artery (PCA), and draining via an enlarged deep parenchymal vein into the right transverse sinus. An ill-defined CSF attenuation adjacent to the AVM with possible encephalomalacia in the right occipital region was noted. The patient was scheduled to undergo an elective intra-cranial AVM excision surgery after considering risk benefit ratio.

A detailed preoperative assessment was done. General examination and systemic examinations were unremarkable except presence of neurological deficits – power in the left upper limb was 1/5 and that of left lower limb was 2/5, with deviation of the angle of the mouth. Preoperative investigations were within normal range with Hb 12 gm/dL; coagulation profile and renal profile was within normal range. ECG and chest x-ray were done which were normal. An echocardiography was done which reveled LVEF-60% with Trivial TR and no other abnormality. It is important to assess baseline cardiac function and to rule out any cardiac anomalies which is crucial for anticipating hemodynamic fluctuations during AVM excision, particularly in cases with high-flow shunts or potential right to left circulations. Informed anaesthesia consent, and consent for high-risk, and consent for blood transfusion and postoperative ICU admission were taken after explaining the procedure and potential risks to the patient and the relatives.

On the day of the surgery the patient was continued on anti-convulsive therapy and inj. mannitol 20g iv. The patient was taken inside the operation theatre and all standard ASA monitors attached including ecg, pulse oximetry, temperature probe and NIBP. Patient was pre oxygenated and premedication with - inj. glycopyrrolate 0.2mg iv, inj. midazolam 0.03mg/kg iv, inj. fentanyl 2mcg/kg iv were given. Anaesthesia was induced with inj. propofol 2 mg/kg iv and inj.

atracurium 0.5mg/kg iv. A smooth atraumatic endotracheal intubation was facilitated with a 7.0 mm cuffed endotracheal tube which further reduced the risk of intracranial pressure spikes. Potential hypertensive surge during laryngoscopy, which was prevented by using preservative free lignocaine 1-1.5mg/kg iv. In high-flow AVMs, this may raise the risk of rupture or intraoperative bleeding. Shortly after administering iv atracurium, the patient developed a generalized erythematous reaction, not associated with bronchospasm or any haemodynamic instability. In the light of this cutaneous response, inj. atracurium was discontinued and inj. vecuronium was subsequently used for maintaining neuromuscular blockade. Anaesthesia was maintained using sevoflurane in air – oxygen mixture and vecuronium infusion. Ventilation was maintained using Pressure-Regulated Volume Control (PRVC) mode, with adjustments to maintain normocapnia ($EtCO_2$ between 35–40 mmHg). Controlled arterial hypotension (CAH) was selectively applied to reduce bleeding from high-flow AVM channels. A radial arterial line was inserted to monitor strict controlled hypotension. The surgery lasted for 10 hours. Intraoperatively total 3L of Ringers lactate were given as iv fluids, total urine output was 2700mL and blood loss was 800mL, which was managed with one unit of PRBC transfusion. An arterial blood gas (ABG) was sent at the end of surgery to identify any hidden acid-base or metabolic disturbances, especially after prolonged anaesthesia which did not show any respiratory or metabolic disturbances.

The patient was not reversed at the end of the surgery and was shifted to the ICU postoperatively for elective mechanical ventilation, where she was gradually weaned off from ventilatory support and extubated the next day. Hemodynamic and glucose levels remained stable throughout the recovery period. SBP was maintained <120 mmHg to mitigate the risk of Normal Perfusion Pressure Breakthrough (NPPB) syndrome. The motor power was assessed on post operatively day 1with power in the left upper limb 2/5 and that of left lower limb was 3/5. She was continued on anti-convulsive therapy with physiotherapy. The patient was shifted to the ward after 4 days of ICU stay. She was advised to continue physiotherapy in the post operative period in the wards. The patient was subsequently discharged from the hospital at post operative day-15.



Intra-operative excision of AVM

DISCUSSION

Intracranial AVMs present multifaceted anaesthetic challenges due to their altered cerebrovascular dynamics, risk of rupture, and the potential for intraoperative haemorrhage or postoperative neurological deficits. This case report outlines the comprehensive anaesthetic approach for a 32-year-old female undergoing elective excision of a right temporal AVM following prior embolization and neurological deterioration.

Patients suitable for intracranial AVM excision are typically those with Spetzler-Martin grade I or II lesions, aged below 40 years, and with small, compact AVMs located in non-eloquent, superficial brain regions. Additional factors include a history of prior rupture and the absence of major comorbidities. These criteria together help identify patients who are most likely to benefit from surgical excision with minimal neurological risk.

Preoperative continuation of antiepileptic therapy (levetiracetam) was crucial to maintain seizure control, as interruption could provoke perioperative seizures, particularly under the stress of surgery. Similarly, mannitol was continued on the morning of surgery, aiding in preemptive intracranial pressure (ICP) reduction and facilitating brain relaxation even before craniotomy. These premeditations play a critical role in improving intraoperative conditions and minimizing complications.

Sevoflurane being a volatile agent has several advantages in neurosurgical cases:

o It has rapid onset and offset, allowing precise titration during critical surgical periods and faster emergence postoperatively.

o At concentrations <1 MAC, sevoflurane preserves cerebral autoregulation and exerts minimal effect on intracranial pressure, making it well-suited for patients with cerebral pathology.

o It also allows smooth neuromuscular control and stable hemodynamic, essential during long neurosurgical procedures involving AVMs.

Maintaining normocapnia is vital in AVM surgery as:

o Hypercapnia can cause cerebral vasodilation, increasing cerebral blood flow and potentially precipitating rupture of fragile AVM vessels.

o Hypocapnia, on the other hand, can lead to vasoconstriction and steal phenomenon, compromising perfusion in adjacent brain tissue already at risk of ischemia due to vascular rerouting by the AVM.

Therefore, normocapnia helps optimize cerebral perfusion and reduce perioperative complications. Controlled arterial hypotension (CAH) was selectively applied to reduce bleeding from high-flow AVM channels. Inj. Mannitol administered intraoperatively, reinforced brain relaxation and ICP control. Precise monitoring of fluid input and output was done throughout the surgical duration. It is crucial during intracranial AVM excision to avoid fluid overload, which can raise intracranial pressure and worsen brain oedema. Balanced fluid management helps maintain stable cerebral perfusion without increasing the risk of bleeding. It also assists in timely replacement of blood loss and supports overall hemodynamic control during surgery

Post operative picture of the patient

CONCLUSION

This case exemplifies a structured, evidence-based approach to anaesthetic management in AVM excision. Sevoflurane proved advantageous for maintaining cerebral stability, rapid emergence, and preserving autoregulation. Preoperative continuation of levetiracetam and mannitol enhanced intraoperative seizure prophylaxis and brain relaxation. Normocapnia was stringently maintained to avoid steal or rupture phenomena. Combined with controlled hypotension, strict fluid management, and targeted postoperative care, this approach contributed to a stable perioperative course and facilitated early postoperative recovery. This case highlights the importance of integrating neurophysiological principles into anaesthetic practice for complex cerebrovascular surgeries.

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