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Perioperative Anaesthetic Challenges in Endovascular Coiling of a Ruptured Distal Anterior Cerebral Artery Aneurysm with Intracerebral and Intraventricular Hemorrhage - A Case Report

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Abstract

BACKGROUND AND AIMS: Distal anterior cerebral artery (ACA) aneurysms are uncommon intracranial lesions that frequently present with frontal lobe intracerebral hemorrhage (ICH) and intraventricular hemorrhage (IVH), posing significant perioperative anaesthetic challenges. We report the anaesthetic management of a ruptured proximal A2 segment ACA aneurysm treated with endovascular coiling. ***CASE DESCRIPTION:*** A 54-year-old hypertensive female presented with sudden loss of consciousness. CT brain and cerebral angiography revealed right frontal ICH with pan-ventricular IVH extension and a saccular aneurysm (3.5 x 2.7 mm) at the right proximal A2 ACA segment, demonstrating interval growth to 5 x 3.24 mm on repeat angiography. Urgent endovascular coiling was performed under general anaesthesia, achieving complete obliteration. Postoperatively, the patient demonstrated progressive neurological improvement. ***CONCLUSION:*** Meticulous perioperative anaesthetic management with haemodynamic optimisation, neuroprotective strategies, and smooth induction and emergence is pivotal for favourable outcomes in ruptured distal ACA aneurysms undergoing endovascular treatment.

Keywords: Anterior Cerebral Artery Aneurysm, Distal ACA, Endovascular Coiling, Neuroanesthesia, Intracerebral Hemorrhage, Intraventricular Hemorrhage, Haemodynamic Management, Neuroprotection.

INTRODUCTION

Intracranial aneurysms affect approximately 3.2% of the general population, with the anterior cerebral artery (ACA) accounting for 30–40% of all aneurysms.[1] However, those arising from the distal ACA - particularly the A2 segment and beyond - represent a rare and clinically distinct subset, comprising only 1–9% of all intracranial aneurysms.[2] Their unique anatomical location and propensity to present with frontal lobe ICH and IVH, rather than the classic subarachnoid pattern, pose distinctive diagnostic and perioperative anaesthetic challenges.[2,3]

Unlike aneurysms of the internal carotid or middle cerebral arteries, distal ACA aneurysms are often small (<7 mm) and may be difficult to visualize on initial non-invasive imaging. Their rupture frequently results in ICH due to proximity to the corpus callosum and cingulate gyrus, leading to combined SAH, ICH, and IVH presentations.[2] This complex haemorrhage pattern is associated with higher intracranial pressure (ICP), impaired cerebral autoregulation, and greater risk of secondary brain injury.[1,4]

The anaesthetic management of ruptured intracranial aneurysms requires careful balancing of competing priorities: maintaining adequate cerebral perfusion pressure (CPP) while preventing dangerous spikes in systemic arterial pressure that may precipitate re-rupture.[5,6] In patients with concurrent ICH and IVH, where ICP may be significantly elevated and autoregulatory capacity compromised, these challenges are amplified.[1,4] We present the perioperative anaesthetic management of this patient and discuss evidence-based strategies for optimising outcomes.

CASE PRESENTATION

Patient History and Presenting Features

A 54-year-old female presented to the emergency department following sudden loss of consciousness with an accidental fall. The ictus was heralded by a brief prodrome of profuse diaphoresis, dizziness, palpitations, and generalised weakness - consistent with a sentinel symptomatic event.[1] Past medical history was notable for a 10-year history of chronic headaches and systemic hypertension diagnosed 4 months prior, managed with oral propranolol 40 mg once daily. On examination, she had altered sensorium and was classified as WFNS Grade III / Hunt-Hess Grade III at presentation.[1,2] Pupils were equal and reactive bilaterally. No focal neurological deficit was documented on initial assessment.

Investigations and Imaging

A structured investigative workup was undertaken. Key findings are summarised in Table 1 below, with corresponding neuroimaging presented in Figures 1–7.

Investigation	Findings
CT Brain (15/07/2025)	Acute hyperdense haemorrhage in the right frontal lobe adjacent to the falx cerebri with surrounding vasogenic oedema and intraventricular extension into all four ventricles. Mild hydrocephalus noted. (See Figures 1–2)
CT Cerebral Angiography (15/07/2025)	Small saccular aneurysm (3.5 x 2.7 mm) with narrow neck arising from the right medial orbitofrontal artery at the proximal A2 segment of the ACA. No other aneurysms identified. 3D reconstruction demonstrated ACA, MCA and ICA anatomy. (See Figure 3)
MRI Brain (pre-op)	Confirmed acute ICH with intraventricular extension on multiple sequences (T2, FLAIR, T1, IR, DWI). Haemosiderin deposition on GRE/SWI consistent with acute-on-chronic changes. No underlying cavernous malformation or tumour. (See Figures 4–7)
DSA (24/07/2025)	Interval enlargement of aneurysm to 3.6 x 3.5 mm. Saccular morphology with dome-to-neck ratio suitable for primary coil embolisation.
Laboratory / ECG	Haemoglobin, renal function, coagulation profile, serum electrolytes, and cardiac enzymes within normal limits. ECG: sinus tachycardia without ischaemic changes.

Table 1. Summary of key investigations and findings

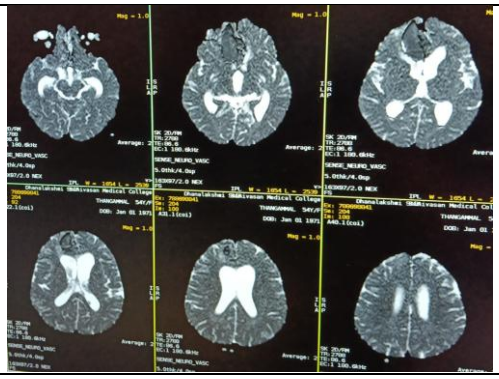

Figure 1. CT Brain (15/07/2025) — Axial (upper) and Coronal (lower) views demonstrating acute hyperdense haemorrhage in the right frontal lobe adjacent to the falx cerebri with surrounding oedema and intraventricular extension into all four ventricles, consistent with subarachnoid haemorrhage and IVH

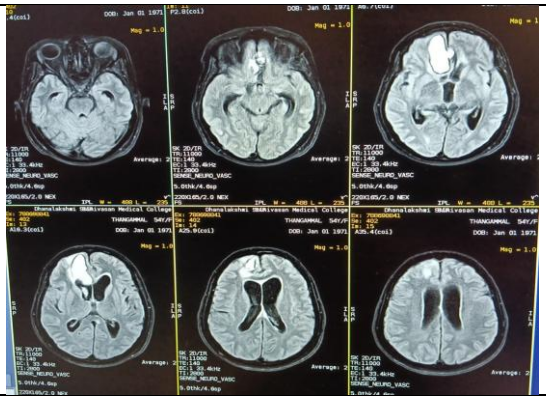

Figure 2. CT Brain (15/07/2025) — Coronal (upper) and Sagittal (lower) reconstructions further delineating the extent of right frontal ICH and IVH, with evidence of mild hydrocephalus

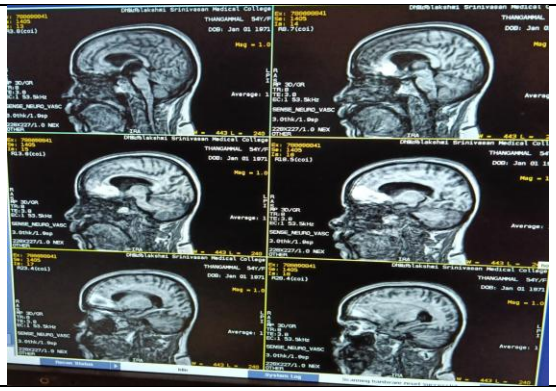

Figure 3. CT Cerebral Angiography (15/07/2025) - 3D volume-rendered reconstruction (AP and PA views, upper panels) and MIP images (lower panels) demonstrating the cerebrovascular anatomy. Arrows indicate the small saccular aneurysm (3.5 x 2.7 mm) arising from the right medial orbitofrontal artery at the proximal A2 segment of the anterior cerebral artery

Preoperative MRI - Multi-sequence Characterisation of Haemorrhage

MRI Brain provided multi-sequence characterisation of the haemorrhage, delineating extent, acuity, and relationship to adjacent structures.[1] Axial T2/FLAIR sequences confirmed the right frontal ICH with IVH. Coronal T2 and IR sequences demonstrated the vertical haemorrhage extent. Sagittal T1-weighted sequences showed the relationship of the frontal haematoma to midline structures. DWI sequences excluded acute territorial infarction.

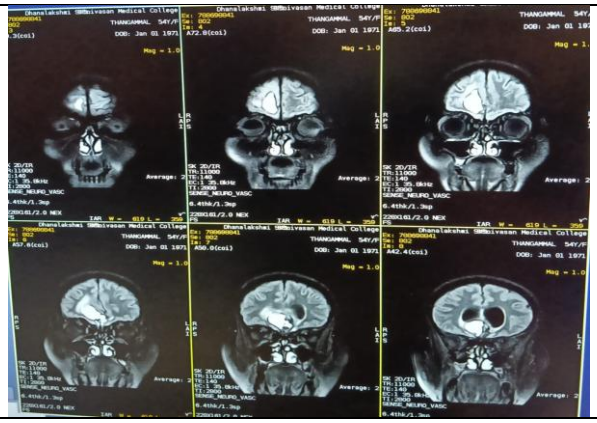

Figure 4. MRI Brain — Axial T2/FLAIR sequences demonstrating acute right frontal intracerebral haemorrhage with intraventricular extension. The bright T2 signal within the ventricular system confirms IVH with layering of blood products in the posterior horns bilaterally

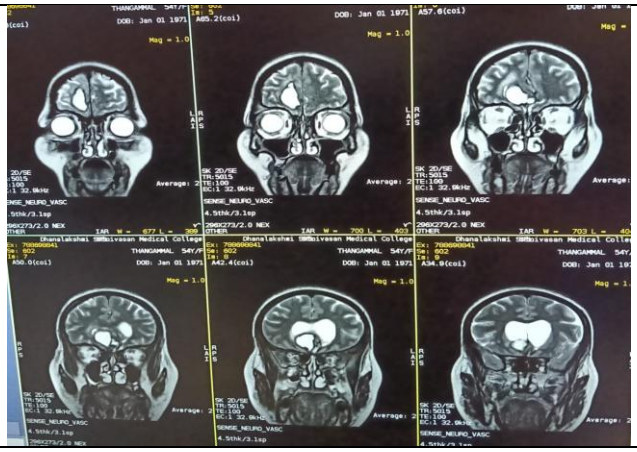

Figure 5. MRI Brain - Coronal T2-weighted sequences showing the vertical extent of the right frontal ICH and confirming bilateral intraventricular haemorrhage with dependent haemosiderin layering

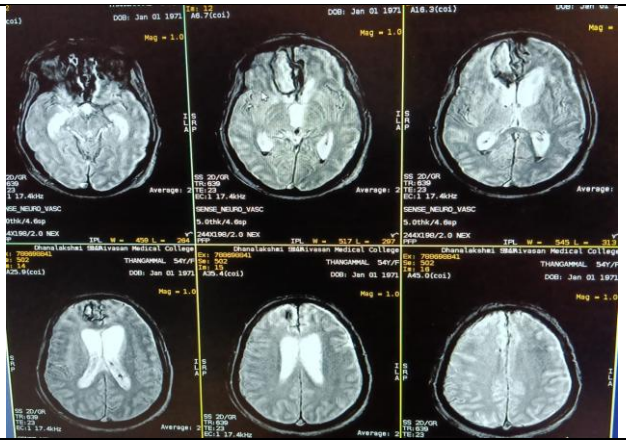

Figure 6. MRI Brain - Coronal FLAIR/IR sequences providing additional characterisation of the perihaematomal oedema zone and intraventricular blood products, confirming pan-ventricular extension of the haemorrhage

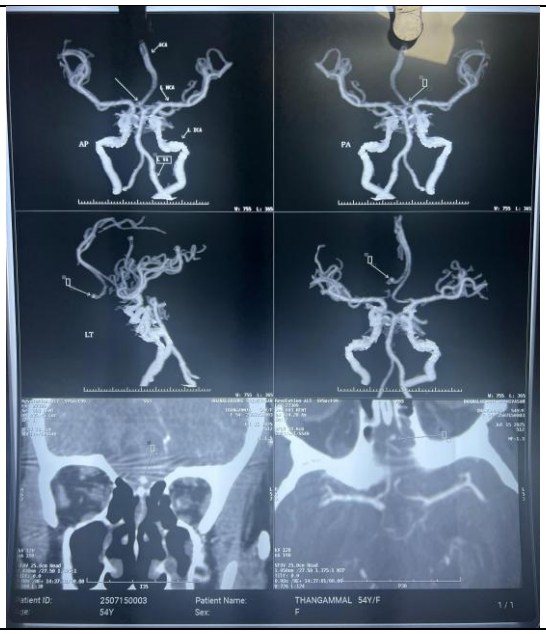

Figure 7. MRI Brain - Sagittal T1-weighted (MP-RAGE) sequences demonstrating the right frontal haematoma in relation to the falx cerebri and corpus callosum, confirming the parasagittal location of the haemorrhage consistent with a proximal A2 segment ACA aneurysm origin

ANAESTHETIC MANAGEMENT

Preoperative Assessment and Preparation

Comprehensive pre-procedural assessment was performed. Notable anaesthetic risk factors included: (1) WFNS Grade III subarachnoid haemorrhage with ICH/IVH and suspected elevated ICP;[1,7] (2) systemic hypertension on propranolol, which may blunt tachycardia as a surrogate of inadequate anaesthetic depth;[5] (3) risk of cerebral vasospasm in the delayed postoperative period[1,4]; and (4) a narrow haemodynamic window given the conflicting requirements of adequate CPP and aneurysm re-rupture prevention.[5,6] Anxiolytic premedication was withheld given the reduced GCS and aspiration risk. Written informed consent was obtained from the next of kin.

Intraoperative Monitoring and Access

Standard ASA monitoring was established including continuous ECG, pulse oximetry, and non-invasive blood pressure. Invasive arterial blood pressure monitoring via right radial artery cannulation was instituted prior to induction, enabling beat-to-beat assessment and facilitating serial arterial blood gas sampling.[5] Large-bore peripheral venous access was secured bilaterally. Bilateral near-infrared spectroscopy (NIRS) cerebral oximetry was applied to monitor regional cerebral oxygen saturation as a non-invasive surrogate of cerebral perfusion.[3] Urinary catheterisation was performed for fluid balance monitoring.

Induction of Anaesthesia

The induction of anaesthesia in ruptured aneurysm patients represents the highest-risk phase of the perioperative period, associated with haemodynamic lability and increased risk of aneurysmal re-rupture.[5,6,8] Pre-oxygenation was performed for 3 minutes with 100% oxygen. Anaesthesia was induced with propofol (1.5–2 mg/kg titrated to effect) and fentanyl (2 mcg/kg IV, administered 3 minutes prior to laryngoscopy) to attenuate the sympathetic pressor response to laryngoscopy.[5,8] Rocuronium (1.2 mg/kg) facilitated neuromuscular blockade. Intravenous lidocaine (1.5 mg/kg) was administered 90 seconds before laryngoscopy as an adjunct to attenuate airway reactivity.[6] Video laryngoscopy facilitated smooth, rapid intubation. Blood pressure remained within the pre-agreed MAP target of 70–90 mmHg throughout induction without vasoactive rescue.[1,7]

Maintenance and Intraoperative Conduct

Anaesthesia was maintained with isoflurane (0.8–1.0 MAC) in an oxygen-air mixture (FiO2 0.4), titrated to BIS 40–60, ensuring adequate depth while limiting haemodynamic suppression. Intermittent rocuronium maintained neuromuscular blockade throughout. Volume-controlled ventilation targeted normocapnia (ETCO2 35–40 mmHg), confirmed by 30-minute ABG correlation.[5,8] Hypercapnia was strictly avoided, as it may exacerbate intracranial hypertension, while excessive hypocapnia risks cerebral ischaemia through vasoconstriction.[5] Tidal volumes were set at 6–8 ml/kg ideal body weight with PEEP 5 cmH2O.

Core temperature was maintained within 36.5–37.5°C using an active warming blanket, as hyperthermia exacerbates ischaemic brain injury.[4,5] Goal-directed isotonic crystalloid (normal saline 1–2 ml/kg/hour) was used for fluid maintenance; glucose-containing solutions were avoided given the risk of hyperglycaemia-mediated secondary brain injury.[4] Blood glucose was monitored hourly, targeting 7.8–10 mmol/L as per neurocritical care protocol.[4] Systemic heparin (70 IU/kg, ACT >250 s) was administered after femoral sheath placement per neurointerventional protocol, with ACT monitored at 30-minute intervals.

Haemodynamic management was guided by a MAP-based protocol targeting 70–90 mmHg.[1,7] Transient hypertensive episodes were managed with labetalol (5–10 mg IV boluses) or propofol dose adjustment; hypotension was treated with phenylephrine (100 mcg bolus) or noradrenaline infusion as indicated.[5]

Intraoperative Angiographic Findings

Intraoperative DSA confirmed further interval aneurysm growth, with the maximal dimension increasing to 5 mm from 3.6 mm documented at the prior DSA on 24/07/2025. The orthogonal diameter measured 3.24 mm compared to 3.5 mm on the prior study; this apparent reduction reflects measurement variation inherent to differing angiographic projection angles rather than true size reduction - a recognised limitation of two-dimensional DSA measurements.[2] The overall aneurysm volume had clearly increased, evidenced by the significant growth in maximal dimension and altered dome morphology. This rapid enlargement over 18 days conferred substantially elevated imminent re-rupture risk and validated the urgency of intervention.[2,3] Primary coil embolisation was performed using GDC-type platinum coils, achieving Raymond-Roy Class I (complete occlusion) on final angiographic check.[9] The intraoperative period remained haemodynamically stable with no episodes of aneurysmal rupture, thromboembolic events, or haemodynamic escalation. Total procedure duration was 2 hours 40 minutes. Anaesthesia time was 3 hours 10 minutes.

EMERGENCE AND POSTOPERATIVE MANAGEMENT

Emergence Strategy

Emergence from anaesthesia represents a high-risk period in ruptured aneurysm patients due to haemodynamic perturbations associated with tracheal extubation.[5,6] Neuromuscular blockade was reversed with sugammadex (2 mg/kg) following confirmation of TOF ratio >0.9. A smooth, cough-free extubation was facilitated by dexmedetomidine infusion (0.4–0.7 mcg/kg/hour, commenced 30 minutes prior to extubation) for sympatholysis and cooperative sedation without respiratory depression,[5] and intravenous lignocaine (1 mg/kg) 60 seconds before extubation. The patient was extubated awake and cooperative, maintaining SpO2 >98%. Blood pressure at extubation was 138/82 mmHg (MAP 100 mmHg). No coughing, bucking, or agitation was observed.

Postoperative Course and Imaging

The patient was transferred directly to the neurocritical care unit (NCCU). GCS on NCCU admission was 13/15 (E4V4M5), representing marked improvement from presentation. Post-procedural MRI Brain (03/08/2025) demonstrated the coil mass in situ with no residual or recurrent aneurysm filling, residual right frontal ICH (48 x 22 mm), and residual bilateral posterior horn IVH - consistent with pre-existing haemorrhage without new bleeding and no acute infarction.

Serial TCD ultrasonography on days 3, 5, and 7 showed mean cerebral blood flow velocities within acceptable limits.[1,4] No pharmacological hypertensive therapy was required for vasospasm management. The patient achieved mRS 2 at hospital discharge on day 14 and was transferred to a dedicated neurorehabilitation facility.

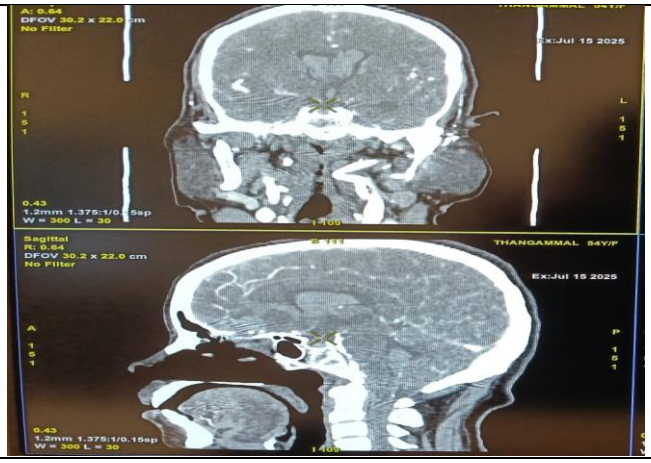

Figure 8. MRI Brain (03/08/2025) - Post-procedural axial FLAIR/IR sequences demonstrating the coil mass in situ at the right proximal A2 segment, with residual right frontal intracerebral haemorrhage (48 x 22 mm) and bilateral posterior horn intraventricular blood products. No new haemorrhage or acute infarction identified

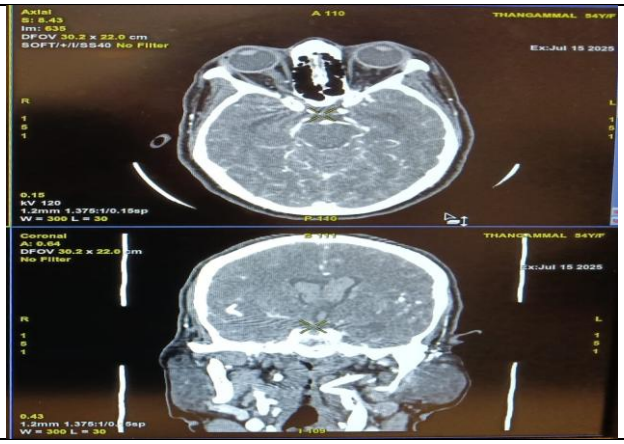

Figure 9. MRI Brain (03/08/2025) - Axial DWI and T2-weighted sequences confirming absence of acute ischaemic infarction in the ACA territory. The ventricular system demonstrates residual blood products with expected evolution of haemorrhage signal characteristics, without evidence of re-bleeding or hydrocephalus progression

Postoperative Pharmacological Management

Pharmacological management was directed at four principal targets:

- Vasospasm prophylaxis: Nimodipine 60 mg orally every 4 hours for 21 days - the only pharmacological agent proven to reduce delayed cerebral ischaemia and improve neurological outcomes after SAH.[1,4]
- Seizure prevention: Levetiracetam 500 mg twice daily, chosen for its favourable pharmacokinetic profile and absence of CYP-enzyme interactions.[1]
- Analgesia: Multimodal - regular paracetamol (1 g four times daily) with low-threshold opioid supplementation. NSAIDs avoided due to antiplatelet effects.
- Haemodynamic control: Propranolol withheld acutely; gradual antihypertensive reintroduction planned once neurologically stable, targeting SBP 120–140 mmHg.[7]

DVT prophylaxis: Sequential compression devices intraoperatively; pharmacological thromboprophylaxis commenced 48 hours post-procedure following multidisciplinary haemostatic review.

DISCUSSION

Our patient exemplifies several overlapping challenges that make anaesthetic care for ruptured distal ACA aneurysms uniquely demanding. The presence of ICH and IVH on a background of hypertension, combined with radiologically confirmed aneurysm growth, placed this case at the highest end of the perioperative risk spectrum. The following sections contextualise our management decisions within the existing literature.

Epidemiology and Clinical Features of Distal ACA Aneurysms

Aneurysms arising from the distal ACA constitute a small but clinically significant subgroup, reported across published series to account for between 1% and 9% of all intracranial aneurysms.[2] What distinguishes this subgroup from more proximal aneurysms is their tendency to bleed directly into adjacent frontal lobe parenchyma rather than exclusively into the subarachnoid space, a consequence of their proximity to cortical and pericallosal structures.[2] When SAH, ICH, and IVH occur simultaneously - as in this patient - the clinical picture becomes substantially more complex, carrying a case fatality rate estimated between 40% and 60% and a meaningfully worse functional trajectory than pure SAH alone.[1,2] The serial imaging in our case documented maximal aneurysm diameter growth from 3.5 mm at initial CTA to 5 mm at the time of intervention over just 18 days. Such accelerated enlargement has been recognised as a surrogate marker of structural wall instability and an indicator of high near-term rupture risk, providing strong justification for urgent rather than delayed treatment.[2,3] The variation in orthogonal diameter across sequential two-dimensional angiographic studies (3.5 mm to 3.24 mm) should not be misinterpreted as size regression; it represents an inherent limitation of projecting a three-dimensional structure onto a two-dimensional plane at differing acquisition angles, a well-described phenomenon in serial aneurysm surveillance.

Haemodynamic Complexity in the Setting of ICH and IVH

Perhaps the most demanding aspect of perioperative care in this case was haemodynamic management. In patients without intracranial pathology, brief surges in arterial pressure are generally well tolerated. In a patient with an unsecured ruptured aneurysm, raised ICP from ICH and IVH, and potentially disrupted cerebrovascular autoregulation, the same surges can be catastrophic.[1,4] When autoregulation is compromised, cerebral blood flow loses its buffering capacity and becomes directly pressure-dependent, meaning that both inadequate perfusion pressure and excessive systemic pressure translate almost immediately into cerebral consequences.[5,6] This narrowing of the therapeutic haemodynamic corridor - where too low risks watershed ischaemia and too high risks haematoma expansion or aneurysm re-rupture - is the defining challenge of the anaesthetic course in such patients.[5,7] Our decision to forgo prophylactic EVD insertion was deliberate. While CSF drainage is intuitive in the setting of IVH with hydrocephalus, abrupt decompression of the supratentorial compartment in the presence of a sizeable frontal haematoma carries a real risk of upward tentorial herniation. Close clinical and radiological surveillance was deemed the safer initial approach.[1,4]

Induction and Emergence: The Two High-Risk Transitions

Anaesthetic induction and tracheal extubation are the two moments during the perioperative course at which the risk of acute haemodynamic disturbance is greatest, and consequently the two moments most closely linked to the risk of aneurysmal re-rupture.[5,6,8] During induction, the mechanical stimulus of laryngoscopy provokes a well-characterised adrenergic surge that can transiently but significantly elevate transmural aneurysm wall tension. Blunting this response requires pre-emptive rather than reactive pharmacology.[6,8] In this patient, propofol was chosen as the primary induction agent for its cerebral metabolic suppression and ICP-lowering properties, while acknowledging that its vasodilatory profile demands careful dose titration, particularly in the setting of propranolol co-administration which may mask compensatory tachycardia.[5,8] Fentanyl administered several minutes ahead of laryngoscopy, combined with intravenous lidocaine, provided layered attenuation of the airway reflex arc and the associated haemodynamic response - an approach grounded in neuroanesthetic practice for high-risk cranial procedures.[5,6] At the other end of the anaesthetic - emergence - the risk is no less significant. Coughing, straining, or agitation during extubation generates sharp transient increases in intracranial and arterial pressure. Dexmedetomidine, with its unique ability to provide dose-dependent sedation and sympathetic attenuation without suppressing the respiratory drive, allowed our patient to emerge cooperatively and calmly, permitting immediate neurological assessment without haemodynamic cost.[5]

Blood Pressure Targets: Navigating Without a Precise Map

One of the recurring frustrations for the clinician managing ruptured aneurysms is the absence of robust, prospectively validated numerical blood pressure targets. Current major guidelines, including the 2023 AHA/ASA document, advocate for blood pressure control prior to aneurysm securing without stipulating specific thresholds, a deliberate acknowledgement that existing evidence does not support a one-size-fits-all figure.[1] In practice, most experienced neuroanaesthetic centres work to a MAP range of 70–90 mmHg during the intraoperative phase, guided by continuous invasive monitoring and contextualised against the individual patient's baseline pressures and neurological status.[5,7] Once the aneurysm is secured, the haemodynamic calculus changes. The dogma of triple-H therapy - hypertension, hypervolaemia, and haemodilution - which dominated vasospasm management for decades, has been substantially revised. Current evidence does not support deliberate hypervolaemia and identifies it as a potential source of harm through fluid overload, pulmonary complications, and electrolyte imbalance. The prevailing consensus now favours maintaining euvolaemia as the post-procedural fluid target.[4,10]

Ventilation, Glucose, and Temperature: The Neuroprotective Triad

Beyond haemodynamic management, three intraoperative physiological parameters warrant specific

attention in the neurologically vulnerable patient: carbon dioxide tension, blood glucose, and core temperature.[4,5] Carbon dioxide is a potent regulator of cerebral vessel calibre. Hypocapnia constricts cerebral vasculature - useful in some open craniotomy settings but hazardous when autoregulation is impaired and ischaemic territories are already at risk. Hypercapnia, conversely, dilates cerebral vessels, raises ICP, and increases the pressure differential across the aneurysm wall. Maintaining PaCO2 within the normal physiological range throughout the procedure is therefore not a minor detail but a clinically significant neuroprotective priority.[5,8] Hyperglycaemia compounds neuronal injury in ischaemic zones through multiple mechanisms including free radical generation and lactic acidosis, while hypoglycaemia is directly neurotoxic. Hourly glucose monitoring with a target of 7.8–10 mmol/L threads this needle without exposing the patient to either extreme.[4] Temperature dysregulation is similarly consequential. The injured brain has a heightened metabolic sensitivity to thermal changes; even modest hyperthermia accelerates neuronal death in marginal ischaemic zones, while hypothermia - though intuitively appealing - has not demonstrated clinical benefit in this setting and risks coagulation impairment. Active normothermia throughout the procedure is the pragmatic and evidence-supported approach.[4,5]

Why Endovascular Coiling Was the Right Choice Here

The choice between microsurgical clipping and endovascular coiling for ruptured aneurysms has been substantially informed by the ISAT trial, which demonstrated superior short-term neurological outcomes with coiling in aneurysms amenable to both techniques.[9] For distal ACA aneurysms, anatomy itself tilts the balance toward endovascular treatment. Surgical access to the proximal A2 segment requires an interhemispheric approach through tight corridors, carries risk of retractor injury to the cingulate gyrus and perforating vessels, and is further complicated by the distorted anatomy created by the associated frontal haematoma.[2] The endovascular route bypasses these anatomical constraints but introduces its own anaesthetic considerations: the procedure takes place in an interventional suite rather than an operating theatre, often with restricted access to the patient; systemic heparinisation is required and must be carefully timed relative to the anaesthetic course; prolonged table times demand vigilant attention to patient positioning and pressure areas; and the possibility of intraoperative thromboembolic events requiring intraarterial thrombolysis must be anticipated in the anaesthetic plan.[5]

Vasospasm and Delayed Cerebral Ischaemia: The Post-Procedural Threat

Securing the aneurysm does not conclude the patient's neurological vulnerability. Delayed cerebral ischaemia, a syndrome of neurological deterioration typically manifesting between the fourth and fourteenth post-haemorrhage days, accounts for a substantial proportion of adverse outcomes in patients who survive the initial bleed and early intervention.[1,4] Its pathogenesis extends well beyond large-vessel vasospasm - the mechanism traditionally emphasised - and now encompasses cortical spreading depolarisation, microthrombus formation within small perforating vessels, and inflammatory disruption of the blood-brain barrier. This mechanistic complexity partly explains why therapies targeting macrovascular spasm alone have not consistently translated into improved patient outcomes.[4] Nimodipine occupies a unique position in post-SAH pharmacotherapy: it is the sole agent supported by robust clinical trial evidence for reducing DCI-related morbidity, and its mechanism is now thought to operate at least partly through neuroprotective pathways independent of vasodilation.[1,4] In a patient with concurrent frontal ICH and IVH, as in our case, the risk of delayed neurological deterioration is compounded by haematoma-driven mass effect on adjacent microvasculature and the pro-inflammatory milieu created by intraventricular blood. This warrants a heightened index of suspicion for vasospasm, a structured monitoring protocol using serial TCD and clinical assessment, and a low threshold for advanced perfusion imaging should neurological status change unexpectedly.[1,4]

CONCLUSION

This case reinforces that the anaesthetic management of a ruptured distal ACA aneurysm with ICH and IVH is far more than a technical exercise in airway management and drug selection - it is a continuous process of physiological optimisation across a narrow therapeutic corridor, from the moment of first contact through to neurocritical care discharge. Several specific lessons from this case are worth distilling:

- Pre-induction invasive arterial monitoring is not optional - it is the clinical foundation upon which every haemodynamic decision in this patient population rests.
- Documented aneurysm growth on serial imaging, regardless of whether absolute dimensions appear dramatic, should be treated as a signal of structural instability demanding urgent rather than elective intervention.
- The induction sequence should be designed around blunting the adrenergic response to laryngoscopy, using layered pharmacology - opioid pre-treatment, propofol titration, and adjunctive lidocaine - rather than relying on any single agent.
- Maintaining the four physiological targets of normocapnia, normoglycaemia, normothermia, and normovolaemia simultaneously throughout the procedure is the practical definition of intraoperative neuroprotection in this setting.
- A planned, pharmacologically facilitated emergence using dexmedetomidine is as important

as induction - the extubation moment carries equal potential for haemodynamic harm if not deliberately managed.

- Nimodipine initiated early and continued for the full 21-day window remains the pharmacological cornerstone of post-procedural care, with its benefit now understood to extend beyond simple vasodilatory action.
- Multidisciplinary team integration - spanning neuroanesthesia, neurointerventional radiology, neurocritical care, and neurorehabilitation - is not a courtesy; it is a structural requirement for achieving good outcomes in this patient group.

The favourable neurological recovery achieved in this patient, from WFNS Grade III presentation to mRS 2 at discharge, reflects what coordinated perioperative care can deliver when each phase of management is approached with equal rigour. As endovascular techniques continue to evolve and extend to increasingly complex aneurysm anatomies, the neuroanesthetic skillset required to support these procedures must evolve in parallel.

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