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Perioperative Management of Craniotomy for Cerebral Clipping Aneurysm with Intraoperative Neurophysiological Monitoring: A Case Report

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Abstract

Introduction: The incidence of subarachnoid hemorrhage (SAH) is approximately 9 per 100,000 population per year. SAH caused by ruptured brain aneurysm accounts for around 80% of non-traumatic events, followed by 10% perimesencephalic hemorrhage and another 10% due to arteriovenous malformation (AVM).
Case: A 50-year-old female patient weighing 50 kg who presented on post-ictus day 14 with aneurysmal subarachnoid hemorrhage secondary to a ruptured middle cerebral artery (MCA) aneurysm with GCS 14 (E4V4M6) without any neurological deficit with WFNS grade II.
Discussion: The patient underwent craniotomy and aneurysm clipping performed by a vascular neurosurgeon. A lumbar drain was inserted preoperatively to facilitate controlled cerebrospinal fluid (CSF) drainage and promote intraoperative brain relaxation. During aneurysm clipping, intraoperative neurophysiological monitoring (IONM) was performed by a neurophysiologist to continuously assess motor evoked potentials (MEPs) and somatosensory evoked potentials (SSEPs), ensuring the preservation of neural pathway integrity throughout the procedure.
Conclusion: The primary goal of anesthetic management was to prevent elevations in intracranial pressure (ICP) and carefully control mean arterial pressure (MAP) before vessel occlusion and throughout aneurysm clipping to maintain an optimal balance between cerebral perfusion pressure (CPP) and transmural pressure (TMP). Neuroanesthesia plays an important role in the perioperative management of patients from the initial stabilization in the emergency department, intraoperative neuroprotective strategies during surgical intervention, and postoperative care in the neurologic intensive care unit.

Keywords: SAH, craniotomy clipping aneurysma, intraoperative neurophysiological monitoring, brain protection, neurologic intensive care unit

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Introduction

Subarachnoid hemorrhage (SAH) remains a serious global health problem because it is associated with high mortality and long term disability. Patient outcomes are influenced by several factors including the amount of bleeding at the onset the occurrence of rebleeding and the extent of cerebral ischemia. Clinical complications including cardiac disturbances and neurogenic pulmonary edema often reflect the severity of the condition. The worldwide incidence of SAH is estimated at about 9 cases per 100000 population each year although the rate differs across regions. Lower incidence rates are reported in Central and South America at around 4.2 per 100000 people per year while higher rates are observed in countries such as Japan and Finland which reach 22.7 and 19.7 per 100000 people per year. The higher incidence in these countries is not caused by a greater number of intracranial aneurysms but is more related to an increased likelihood of

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aneurysm rupture. In contrast China reports a lower incidence of approximately 2.0 per 100000 people per year. Although SAH represents only about five percent of all stroke cases it contributes disproportionately to death and long -term disability. Data from a large multinational observational study conducted by the World Health Organization across several populations in Europe and China show that the case fatality rate within 30 days reaches 42 percent which highlights the severity of this condition. SAH caused by ruptured cerebral aneurysms accounts for approximately 80% of non-traumatic cases, followed by 10% due to perimesencephalic hemorrhage and the remaining 10% caused by arteriovenous malformations (AVMs). The majority of these cases involve bleeding in the anterior carotid circulation, while the remaining 10–20% occur in the posterior vertebrobasilar circulation. Studies on surgical risk and prognosis of such cases began with Botterell and colleagues in 1956, who introduced a grading system based on specific criteria. This was later followed by the grading scales developed by Hunt and Hess, Fisher, and most recently, the World Federation of Neurosurgical Surgeons (WFNS).[3]

Neuroanesthesia plays a vital role in the management of such cases across several stages, starting from prehospital first aid, transportation, initial diagnosis, early emergency management, interventional neuroradiology or surgical procedures, and intensive care following definitive treatment. Definitive therapies include endovascular coiling, surgical clipping, and prevention of ischemic events. A thorough understanding of the pathophysiology in these cases is essential for proper anesthetic management during surgery. This includes resuscitation and stabilization, intensive care therapy management, and providing perioperative anesthesia for occlusion procedures. Certain conditions require specialized skills from neuroanesthesiologists, as the comprehensive management of SAH cases significantly influences therapeutic outcomes.[4]

Case

Anamnesis

Mrs. S, a 50-year-old female weighing 50 kg, presented with a decreased level of consciousness prior to hospital admission, based on information obtained from her family. The decreased consciousness occurred in the morning while she was shopping at the market. Initially, the patient experienced weakness in her left limbs. There were no seizures or vomiting, and no headache was reported prior to the loss of consciousness. Her past medical history revealed no known allergies, seizures, asthma, diabetes mellitus, or hypertension; however, she is an active smoker. The patient was first brought to RSI Jemursari Surabaya, the nearest hospital from the scene. A neurologist examined her and found her Glasgow Coma Scale (GCS) score to be E2V1M1. A head CT scan was performed, which revealed intracranial hemorrhage, and hospitalization was recommended. During the early phase of treatment, her GCS improved to E3V4M5. However, after three days of hospitalization, she became increasingly agitated, experienced shortness of breath, and developed a fever. As a result, she was referred to Dr. Soetomo General Hospital in Surabaya for further management.

Physical Examination

On the 14th day of hospitalization, physical examination revealed the following findings: Breathing showed spontaneous breathing with a respiratory rate of 18 breaths per minute, symmetrical bilateral vesicular breath sounds, no wheezing, and minimal rhonchi on both sides. Oxygen saturation was 98% with nasal cannula. Blood and circulation findings included blood pressure of 130/80 (96) mmHg, heart rate of 87 BPM, regular rhythm, with no murmurs or gallop. Brain and disability indicated a Glasgow Coma Scale (GCS) score of 446, with no signs of lateralization. Bladder revealed urine output via catheter (UOP) of 0.8 cc/kgBW/hour. Bowel showed a soft abdomen with normal bowel sounds. On bone examination, there was no edema, and body temperature was 36.3 C.

The results of the laboratory examination (Table 1) showed that most values were within normal limits, except for hypernatremia at 148 mEq/L. The head CT scan (Figure 1) performed at RSI

Laboratory and Radiology Examination

Table 1. Laboratory Results Before Surgery

Examination	Explain	Examination	Explain
Hemoglobin	12,8 g/dl	PT	12,8 g/dl
Trombosit	432.000 / mm3	APTT	24,3 (Control 25) Second
Leukosit	8,20/mm3	Albumin	3,49 u/L
Hematrokit	40,7%	GDS	127 u/L
Natrium	148 mEq/L	pH	7,49
Kalium	3,60 mEq/L	PCO_2	44 (mmHg)
Clorida	98,0 mEq/L	PO_2	69 (mmHg)
Ureum	14 mg/dl	HCO_3	34,3 (mmol)
Creatinine	0,43 mg/dl	BE	11,0 (mmol)
SGOT	42 u/L	TCO_2	35,7 (mmol)
SGPT	59 u/L	SO_2C	95%

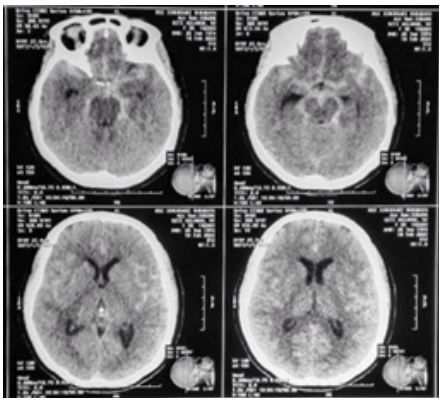

Figure 1. Head CT Scan Shows SAH In The Basal Cistern And Sulci Of The Right And Left Frontotemporoparietal Lobes

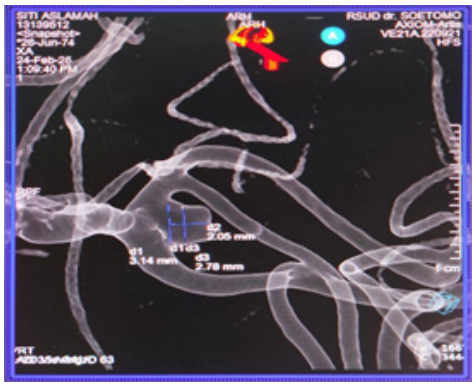

Figure 2. Digital Subtraction Angiography Reveals A Wide-Neck Saccular Aneurysm At The Left MCA Bifurcation Pointing Posterolaterally

Jemursari revealed a subarachnoid hemorrhage (SAH) in the basal cistern and sulci of the right and left frontotemporoparietal lobes. At Dr. Soetomo General Hospital, Digital Subtraction Angiography (Figure 2) was performed, which showed a wide-neck saccular aneurysm at the left MCA bifurcation with a neck size of 3.14 mm, a dome width of 2.78 mm, and a height of 2.05 mm, pointing posterolaterally. Due to a neck to dome ratio of 0.6, coiling would be technically challenging. A chest X-ray showed worsening pneumonia compared to the previous image, a prominent cardiac silhouette, and an elevated right hemidiaphragm. Cardiac evaluation revealed an ECG with sinus rhythm at 76 bpm, frontal axis showing LAD, horizontal axis with clockwise rotation (CWR), and slow R wave progression from V1–V3. Echocardiography showed concentric left ventricular remodeling with normal systolic function (Biplane EF 72%). The patient received the following treatments in the ward: NaCl 0.9% infusion at 1000 cc/24 hours, metamizole 1 gram IV three times a day, furosemide 20 mg IV once daily (if MAP > 65), omeprazole 40 mg IV twice daily, cefoperazone-sulbactam 2 grams IV twice daily (based on culture result showing Acinetobacter baumannii sensitive to cefoperazone-sulbactam), nimodipine 60 mg orally six times a day, vitamin B1B6B12 once daily, curcuma one tablet three

times a day, N-acetylcysteine 200 mg three times a day, salbutamol nebulizer 2.5 mg three times a day, and spironolactone 50 mg once daily. The patient was classified as ASA physical status 4. From the central nervous system, SAH was noted with a 14-day onset and GCS of 14 (E4V6M4). Respiratory assessment revealed neurogenic pulmonary edema and Acinetobacter baumannii pneumonia (PF ratio 328, SpO_2 97% on room air). Cardiovascular assessment showed a history of acute decompensated heart failure (ADHF) that had improved (NT-proBNP 1818, normal <125), with concentric left ventricular hypertrophy and normal systolic function (biplane EF 72%). Electrolyte analysis showed hypernatremia (Na 148 mEq/L).

General Anesthesia

The patient underwent induction using brain protection principles and measures to prevent increased intracranial pressure, with a combination of intravenous administration of lidocaine 80 mg, thiopental 75 mg, fentanyl 75 mcg, and rocuronium 50 mg. Preoxygenation was performed for 5 minutes without hyperventilation. Intubation was carried out under sleep apnea conditions using a Macintosh size 3 laryngoscope, with a 7.0 mm endotracheal tube (ETT) inserted to a depth of 19 cm at the lip. Post-intubation, hemodynamics remained stable with a heart rate of 72 bpm, blood pressure of 121/74 mmHg, and pupils equal in size (3 mm) and reactive to light.

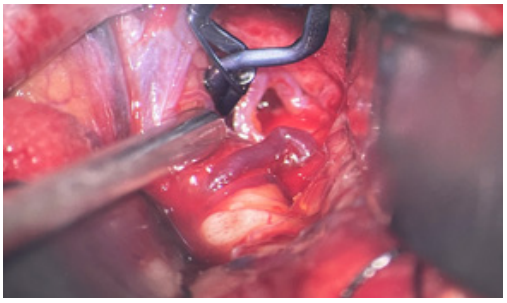

Figure 3. The Process of Clipping an Aneurysm in The Middle Cerebral Artery

An arterial line was placed using a 20 G Surflo catheter in the right radial artery for real-time blood pressure monitoring. Anesthesia maintenance was provided using Ringer fundin solution and Gelofusin during estimated blood loss (EBL >10%), and propofol was administered using target-controlled infusion (TCI) with the Schneider model targeting 3–4 mcg/mL effect-site concentration. Dexmedetomidine was infused at 0.4–0.6 mcg/kgBW/hour adjusted according to the patient's hemodynamics, along with continuous infusion of fentanyl at 20 mcg/hour, and norepinephrine at 0.05–0.1 mcg/kgBW/min at the initiation of the clipping procedure. Intravenous paracetamol 1 gram was also given to maintain the patient in controlled hypothermia (35°C). Mechanical ventilation was managed using an anesthesia machine in controlled mode, targeting a $PaCO_2$ of 35 mmHg, adjusted based on arterial blood gas analysis and end-tidal CO_2 readings from the anesthesia monitor. Intraoperative monitoring included continuous evaluation of systolic and diastolic blood pressure, mean arterial pressure, oxygen saturation, ECG waveform, precordial stethoscope auscultation,

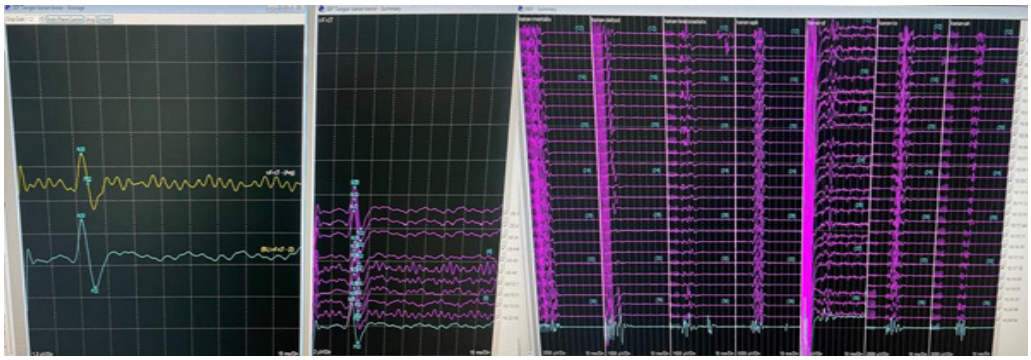

Figure 4. Intraoperative Neurophysiological Monitoring to Assess Somatosensory Evoked Potential Function & Motor Evoked Potential Function.

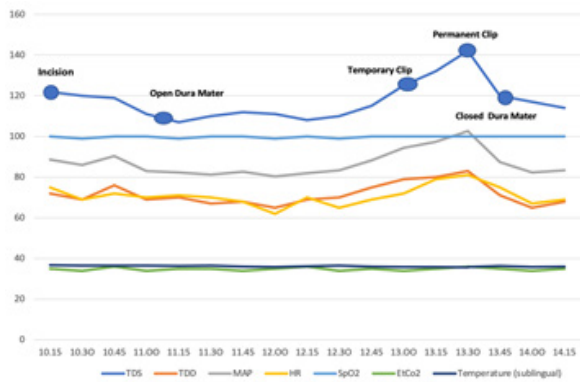

Figure 5. Intraoperative Hemodynamics

and urine output via urinary catheter. The patient was positioned supine throughout the surgical procedure.

Surgical Procedure

The vascular neurosurgeon began by placing a lumbar drain at the L3–L4 level using a 14 G trocar to reduce cerebrospinal fluid (CSF) in the event of excessive production and to allow the brain to remain relaxed. A craniotomy was then performed, followed by dissection and dural opening. The brain was already relaxed and not edematous, making the administration of mannitol at a dose of 0.5 g/kgBW unnecessary. The surgeon proceeded to locate the middle cerebral artery (MCA) with the aneurysm. Before occlusion, a technique was used to control the mean arterial pressure (MAP) under basal conditions. Just prior to performing occlusion at the proximal segment, a norepinephrine syringe infusion at 0.05–0.1 mcg/kgBW/min was initiated to achieve a MAP target 10–20% above the baseline level. The surgeon then proceeded with the clipping of the aneurysm neck (Figure 3), which took approximately 10 minutes. During the clipping process, the role of the neurophysiologist was essential in utilizing intraoperative neurophysiological monitoring to assess the function of motor evoked potentials (Figure 4) and somatosensory evoked potentials (Figure 5), ensuring they remained within normal limits. After the clip was placed, bleeding was explored and confirmed to have stopped, and the size of the aneurysmal sac had reduced. At this point, blood pressure was returned to baseline or normal levels. Before closing the dura mater, the surgeon ensured that the brain remained relaxed. The surgery lasted approximately 4 hours with an estimated blood loss of 400 mL, which was replaced with 350 mL of colloid fluid. Hemodynamic parameters remained relatively stable throughout the procedure. Upon completion of the surgery, the patient, still intubated, was transferred to the Neurologic Intensive Care Unit for close monitoring and postoperative care.

Neurologic Intensive Care Unit

On the first day in the Neurologic Intensive Care Unit, respiratory system examination showed that the patient was on a ventilator with SIMV mode, Pinsp 15 cmH_2O, PEEP 5 cmH_2O, FiO_2 50%, VTE 390 mL, and MV 4900 mL. Bilateral vesicular breath sounds were present with no rhonchi or wheezing. Cardiovascular assessment revealed warm extremities, heart rate of 66 beats per minute, blood pressure of 110/65 mmHg, mean arterial pressure (MAP) of 79 mmHg, capillary refill time (CRT) less than 2 seconds, and single S1 and S2 heart sounds with no murmur or gallop. Neurologically, the patient was sedated, pupils were isochoric (3 mm) and reactive to light, with no signs of lateralization, and lumbar drain output was 150 cc. In the urogenital system, urination was via catheter with a urine output of 300 mL over 4 hours (1.2 mL/kg/hour). Gastrointestinal examination found a soft abdomen with normal bowel sounds.

The musculoskeletal system showed no edema, and the body temperature was 36.4°C.Therapy in the Neurologic Intensive Care Unit included ventilator support with SIMV mode (Pinsp 15 cmH_2O, PEEP 5 cmH_2O, FiO_2 50%, VTE 390 mL, MV 4900 mL), Ringer's fundin infusion at 1000 cc per 24 hours, dexmedetomidine infusion at 0.2 mcg/kgBW/hour, fentanyl at 10 mcg/hour, paracetamol 1 gram IV three times daily, metoclopramide 10 mg IV three times daily, furosemide 20 mg IV (administered if MAP > 65 mmHg), omeprazole 40 mg IV twice daily, cefoperazone-sulbactam 2 grams IV twice daily (based on culture results showing Acinetobacter baumannii sensitive to this antibiotic), and nimodipine 60 mg via nasogastric tube six times a

Table 2. Postoperative Laboratory Results

Examination	Explain	Examination	Explain
Hemoglobin	11,8 g/dl	PT	15,1 (control 11) second
Trombosit	432.000 / mm3	APTT	24,3 (control 25) second
Leukosit	12,50/mm3	Albumin	3,47 u/L
Hematrokit	36,3%	GDS	117 u/L
Natrium	145 mEq/L	pH	7,41
Kalium	4,30 mEq/L	PCO_2	32 (mmHg)
Clorida	104,0 mEq/L	PO_2	110 (mmHg)
Ureum	14 mg/dl	HCO_3	20,3 (mmol)
Creatinine	0,43 mg/dl	BE	-4,3 (mmol)
SGOT	42 u/L	TCO_2	21,3 (mmol)
SGPT	59 u/L	SO_2c	98%

day. On the second day in the Neurologic Intensive Care Unit, the patient underwent laboratory examinations, with the results summarized in Table 2.

After evaluating the patient's clinical condition on the second day in the Neurologic Intensive Care Unit and reviewing the laboratory results, sedation medications were gradually de-escalated in preparation for extubating. Respiratory system assessment showed a clear airway, spontaneous breathing at 20 breaths per minute, SpO_2 98% with a nasal cannula at 2 L/min, bilateral vesicular breath sounds, and no rhonchi or wheezing. Cardiovascular examination revealed warm extremities, heart rate of 81 beats per minute, blood pressure of 114/75 mmHg, MAP 84 mmHg, capillary refill time (CRT) < 2 seconds, single S1 and S2 heart sounds without murmur or gallop. Neurological status showed a Glasgow Coma Scale (GCS) score of 15 (E4V5M6), pupils were isochoric (3 mm), reactive to light, with no lateralization signs, and lumbar drain output was 100 cc over 24 hours. In the urogenital system, urination was via catheter with a urine output of 150 mL over 3 hours (1 mL/kg/hour). Gastrointestinal examination revealed a soft abdomen with normal bowel sounds. Musculoskeletal examination showed no edema, and the body temperature was 36.5°C.

On the third day in the Neurologic Intensive Care Unit, a head CT angiography with contrast was performed, revealing a postoperative defect in the right temporoparietal calvaria, scalp hematoma measuring approximately 1 cm in thickness in the left frontotemporal region with a drain in place, and a medical device (aneurysm clip) positioned at the left M1 segment. Based on the patient's clinical improvementand the results of laboratory and radiological examinations, the patient was deemed stable for transfer to the High Care Unit.

Discussion

Several factors are known to increase the likelihood of developing cerebral aneurysms, including advancing age beyond 50 years, female sex which shows a higher occurrence compared to males, elevated blood pressure, and smoking habits. The case described involves a 50-year-old woman who smokes and is suspected to have hypertension, although she has not undergone a formal medical evaluation. In addition, autosomal dominant polycystic kidney disease represents a hereditary systemic condition that is closely linked to the presence of intracranial aneurysms, with reported prevalence rates that

are two to four times greater than those found in the general population.[1] Certain disorders such as Marfan syndrome, neurofibromatosis type 1, and fibromuscular dysplasia are also linked to intracranial aneurysms, although their contribution to the risk is relatively limited compared to other major factors.

On CT imaging, patients with subarachnoid hemorrhage often show typical findings, and the distribution of blood within the basal cisterns can help suggest the origin of the bleeding. When the initial CT scan does not reveal abnormalities, confirmation of the diagnosis can be obtained through a lumbar puncture performed several hours after symptom onset, generally within a window of 6 to 12 hours, although some sources extend this period up to 4 days. Digital subtraction angiography is still regarded as the most reliable method for identifying intracranial aneurysms. In this case, a CT scan was carried out shortly after the patient arrived and demonstrated acute bleeding involving the basal cisterns, cortical sulci, and both cerebral gyri. Further evaluation with CT angiography identified a saccular aneurysm at the left middle cerebral artery bifurcation with a neck measuring 3.14 mm, a dome width of 2.78 mm, and a height of 2.05 mm, oriented toward the posterolateral direction. Due to a neck to dome ratio of 0.6, coiling would be technically challenging; thus, clipping was selected as the primary treatment. This aligns with most literature, which indicates that aneurysms often occur at arterial bifurcations due to turbulent blood flow. Other causes can include trauma or infection.

The aneurysm is located in the middle cerebral artery and its branches, which is consistent with findings in the literature that report this site as a common location with a prevalence of around 80%. Outcomes after subarachnoid hemorrhage are mainly influenced by three key factors, namely the level of consciousness after the event, the patient's age, and the amount of bleeding seen on the initial CT scan. In this case, the patient arrived with a Glasgow Coma Scale score of 14 and showed no neurological deficits, while the bleeding volume was relatively small despite the older age, which indicates a more favorable outlook and a lower likelihood of death or long-term disability.

This was reflected in the patient's clinical progress which showed improvement and a stable level of consciousness during hospitalization with ongoing medical therapy. The severity of subarachnoid hemorrhage is assessed using clinical grading systems such as the Hunt and Hess scale and the World Federation of Neurological Surgeons classification. In clinical settings the WFNS scale is more commonly applied because it incorporates the Glasgow Coma Scale score together with the presence of focal neurological deficits. Higher grades on these scales are associated with less favorable clinical outcomes.

In this patient, the SAH grading was classified as WFNS grade II, Fisher grade 3, and Hunt & Hess grade 1 (Tables 3, 4, and 5). According to the literature, patients with these grades generally have intracranial pressure (ICP) values within normal limits and preserved cerebral

Table 3. SAH Grading according to WFNS, Hunt and Hess, Fisher. [4]

World Federation of Neurological Surgeons Scale[43]			Hunt and Hess Scale[44]		Modified Fisher Scale[45]		
rade	Glasgow Coma Scale	Neurologic Examination	Grade	Neurologic Examination	Scale	Subarachnoid Hemorrhage	Intraventricular Hemorrhage
	15	No motor deficit	1	Awake, alert, no cranial nerve or motor deficits, mild headache, minimal or no nuchal rigidity	0	Absent	Absent
	13-14	No motor deficit	2	Awake, alert, moderate to severe headache, nuchal rigidity, no motor deficits, may have cranial nerve deficit	1	Thin	Absent
	13-14	Motor deficit	3	Confusion or lethargy, with or without mild focal neurologic deficits	2	Thin	Present
	7-12	With or without motor deficit	4	Stuporous, more severe focal neurologic deficit	3	Thick[b]	Absent
	3-6	With or without motor deficit	5	Comatose, motor posturing or no motor response	4	Thick[b]	Present

autoregulation. Death in cases of SAH is mainly related to brain injury that occurs at the onset of bleeding, the possibility of repeated bleeding, and the development of delayed cerebral ischemia. The risk of mortality is closely influenced by how extensive the initial hemorrhage is and the patient's neurological condition at presentation. Older individuals and patients with existing medical problems face a greater risk of death. Management is directed primarily at reducing the chance of rebleeding.

Individuals with subarachnoid hemorrhage often show a range of electrocardiographic changes such as alterations in ST segments and T waves that resemble myocardial ischemia along with prolongation of the QT interval and the appearance of U waves. Cardiac involvement may also include both supraventricular and ventricular arrhythmias increased troponin levels and impairment of myocardial function even in the absence of coronary vasospasm. These changes are thought to arise from excessive sympathetic stimulation together with reduced parasympathetic control which can trigger inflammatory responses in cardiac muscle cells. In this patient, cardiovascular abnormalities included a history of improved acute decompensated heart failure (ADHF) with NT-proBNP at 1818 pg/mL (normal <125), concentric left ventricular hypertrophy, and preserved systolic function (biplane ejection fraction of 72%). Elevated troponin levels are associated with a higher risk of cardiovascular complications, vasospasm-induced injury, and poor neurological outcome.

In this case troponin levels were not measured so the cardiac findings are interpreted mainly as a reflection of the underlying neurological injury and these changes are usually temporary and tend to improve over time. When myocardial impairment becomes severe patients may need inotropic therapy and in some situations support with an intra aortic balloon pump. Elevated blood pressure after SAH is linked with a higher risk of death although its management requires careful consideration since increased blood pressure often plays a role in preserving adequate cerebral perfusion. Neurogenic pulmonary edema may occur in patients with SAH and it is believed to arise from a sudden and intense sympathetic response following brain injury. This reaction can impair left ventricular systolic function both globally and in specific segments. In this case, the patient developed lung complications in the form of neurogenic pulmonary edema along with pneumonia caused by Acinetobacter baumannii. The presence of neurogenic pulmonary edema reflects a more severe degree of subarachnoid hemorrhage and is often linked with unfavorable clinical outcomes.[7]

The timing of occlusion procedures depends on several considerations. Various studies offer differing opinions regarding early intervention (< 3 days) versus late intervention (> 10 days). Each timing approach has its own advantages and disadvantages. Early intervention has the benefit of reducing the risk of rebleeding, but it often occurs under suboptimal preoperative conditions. On the other hand, late intervention allows for a more stable and optimal preoperative condition; however, many patients do not survive while waiting for surgery. The advantage of late intervention includes a more relaxed brain condition and more stable metabolic status.[8] The surgical procedure in this case was performed on the 14th day, taking into account several factors. First, the patient's condition upon arrival was relatively good (Hunt and Hess grade I and WFNS grade II). Second, the patient's response to medication was positive, with reduced symptoms and a relatively stable level of consciousness.

Surgical clipping is commonly selected for large or giant aneurysms, lesions with a broad neck indicated by a neck to dome ratio above 0.5, fusiform shapes, and aneurysms located at arterial bifurcations. Lesions within the vertebrobasilar system are often more accessible through an endovascular coiling approach rather than open surgery. Distal basilar aneurysms in particular are difficult to reach with clipping because of their deep position and the challenge of securing proximal vascular control, which makes coiling a safer option in these cases. Aneurysms of the cavernous segment of the internal carotid artery present additional difficulty due to their close

relationship with venous structures and cranial nerves. Aneurysms in the middle cerebral artery are generally less suitable for coiling and are more effectively treated with clipping. In this patient, clipping was considered the most appropriate option since the aneurysm was located in the MCA and had a wide neck with a ratio above 0.5. For ruptured aneurysms, surgical clipping is often preferred because it allows direct removal of the hematoma, which may help reduce the likelihood of vasospasm. In younger individuals, this approach is also favored as it offers more durable protection against future episodes of subarachnoid hemorrhage.[9] Coiling is considered more suitable for older patients and those with multiple comorbid conditions. This minimally invasive approach carries a lower surgical risk, making it a safer option for patients who may not tolerate open surgery well due to age or underlying health issues.

The severity of subarachnoid hemorrhage is influenced by several factors, including the ability of cerebral vessels to respond to changes in blood flow and the effectiveness of autoregulation. When autoregulation is impaired, the likelihood of delayed cerebral infarction becomes higher. In this case, cerebral perfusion relies largely on mean arterial pressure. During anesthesia, the main objective is to maintain a stable transmural pressure across the aneurysm wall while ensuring adequate cerebral perfusion pressure. A rapid rise in blood pressure together with a sudden drop in intracranial pressure can sharply alter this balance and increase the chance of aneurysm rupture. Rupture during anesthetic induction is uncommon and occurs in a small proportion of cases, with lower rates reported using modern techniques, although it is usually related to a sudden increase in blood pressure during tracheal intubation.

The underlying mechanism involves a rapid elevation of intracranial pressure due to bleeding into the subarachnoid space which may extend into the ventricular system. The presence of blood breakdown products and clot formation can further trigger cerebral vasospasm.[3] Several hypotheses underlying the neuropathophysiological mechanism of cerebral vasospasm include calcium ion-dependent vasoconstriction (Ca2+) and non Ca2+, The release of blood breakdown products, free radical reactions, inflammatory responses, and apoptosis contribute to this condition, leading to increased morbidity and mortality in subarachnoid hemorrhage (SAH).[3] Excessive vasospasm that is not promptly treated can lead to delayed cerebral ischemia (DCI).

The underlying process develops through a gradual decline in brain function that begins with cerebral edema and impairment of autoregulation, then progresses to cortical spreading depression and the formation of microthrombi. Cortical spreading depression is described as a wave of neuronal depolarization that moves across brain tissue at a speed of about 2 to 5 mm per minute and suppresses normal EEG activity. This slow moving depolarization disrupts ionic balance in the brain, induces marked vasoconstriction, and can ultimately lead to cerebral ischemia.[13]

Microthrombosis is the result of coagulation cascade activation following the initial hemorrhage.[12] Based on the underlying neuropathophysiological mechanisms, several therapies for cerebral vasospasm have been introduced, including nimodipine, magnesium sulfate, fasudil hydrochloride, tirilazad mesylate, and erythropoietin. These treatments aim to reduce the mortality and morbidity rates of SAH-related stroke due to cerebral vasospasm. In this patient, nimodipine was administered from the time of admission at a dose of 60 mg six times a day to prevent the onset of vasospasm. The mechanism of action of nimodipine is to inhibit the entry of Ca+ ions into cells by blocking calcium ion channels, thereby preventing depolarization of smooth muscle cells. Administration can be done either orally or continuously; according to the literature, there is no difference in outcomes between the two methods.

The systemic vasodilatory effect is only about 5% in patients receiving nimodipine. Mild hypotension can be beneficial during intubation or when controlled hypotension techniques are required before clipping, and it may also reduce the need for additional medications.[3] In this patient, a

Table 4. FAST HUGS BID[19]

For Medical Patient	For Surgical Patient
F Feeding	Feeding (NBM , enteral, TPN)
A Analgesia	Analgesia (Vas Score)
S Sedation	Sensorium (GCS, Ramsay Sedation Score)
T Thromboprophylaxis	Thromboprophylaxis, temperature, tubes
H Head up	Head-up/Hemodynamics
U Ulcer prophylaxis	Ulcer prophylaxis, urine outpus
G Glicemyc control	Glicemyc control
S Spontaneous breathing trial	Supplement O2 (Mask, NIV, high flow O_2
B Bowel Movement	Bowel (ileus/gastroparesis/distension/bowel movement)
I Indwelling catheter	Indwelling catheter (CVC, A-line, epidural, Foleys), imbalance (electrolyte, cumulative fluid)
D Drug de-escalation	Drugs (de-escalation, number of days), delirium

TPN: Total Parenteral nutrition; VAS: Visual analog scale; GCS: Glasgow coma scale; NIV: noninvasive ventilation; CVC: central venous Catheter; NBM: Nil by mouth

lumbar drain was placed, with the optimal time for releasing drainage being after the dura is opened. This approach helps prevent a sudden decrease in intracranial pressure (ICP) and abrupt changes in transmural pressure (TMP). Anesthetic induction and maintenance were managed to ensure relatively stable hemodynamics. Hypotension or hypertension during induction, intubation, surgical pin placement, incision, and dural opening must be avoided. Arterial access is typically established before induction along with placement of large-bore venous access; however, in this patient, the arterial line was inserted after induction. Elevation in ICP must be properly managed. Simple measures such as head-of-bed elevation and avoiding jugular vein compression are highly beneficial.

Keeping arterial carbon dioxide at a low-normal level around 35 mmHg and maintaining sufficient anesthetic depth are important during management. Lowering intracranial pressure can be achieved with agents such as mannitol or hypertonic saline along with gradual and carefully controlled drainage of cerebrospinal fluid through a lumbar drain. Rapid removal of CSF should be avoided because a sudden fall in intracranial pressure can sharply alter transmural pressure and raise the risk of aneurysm rupture. The main objective during anesthetic induction in aneurysm surgery is to limit this rupture risk by stabilizing transmural pressure while preserving adequate cerebral perfusion pressure. Ideally, both parameters are kept close to their preoperative values throughout the induction phase.

Induction and intubation in this case were performed using the principle of brain protection, both in technique and in the selection of medications. The combination of drugs was chosen for their neuroprotective properties, adequacy in achieving induction targets, and minimal impact on hemodynamic stability.1 In this patient, a combination of lidocaine 80 mg IV, thiopental 75 mg IV, fentanyl 75 mcg IV, and rocuronium 50 mg IV was administered, along with preoxygenation for 5 minutes without hyperventilation. The general principle is to reduce the patient's blood pressure by approximately 10%–20% below the normal value. Patients with Hunt and Hess grade I, as in this case, typically have intracranial pressure (ICP) within the normal range and do not experience ischemia, allowing for a wider tolerance range for blood pressure reduction (30%–35% or systolic pressure at around 100 mmHg).[2] During induction and intubation in this patient, the target depth of anesthesia was achieved along with blood pressure reduction within a compensatory range. The induction agents selected in this case

were chosen for their neuroprotective effects. According to the literature, lidocaine is used as an adjuvant for brain protection. Administration of lidocaine can reduce cerebral metabolic rate of oxygen ($CMRO_2$) by 15–20%. The recommended dose is 1.5 mg/kgBW. Another purpose of using lidocaine is to attenuate the hemodynamic response during intubation.[14] In this case, 80 mg of intravenous lidocaine was used during induction with the aim of preventing hemodynamic surges that could increase mean arterial pressure, while also providing neuroprotective effects. The mechanism of lidocaine in brain protection involves reducing transmembrane ion shifts, decreasing the cerebral metabolic rate (CMR), modulating leukocyte activity, and reducing the release of excitotoxins caused by ischemia. The opioid used in this case was fentanyl.

According to the literature, recommended induction combinations include propofol (1.5–2 mg/kgBW) or thiopental (3–5 mg/kgBW)

Table 5. ABCDE FLAT HUGS[20]

A–Airway	Airway: Adequacy of airway, type of airway (e.g., endotracheal tube, tracheostomy)
B–Breathing	Respiratory rate, oxygen saturation (SaO_2), PaO_2, $PaCO_2$, acid-base status (pH, lactate, bicarbonate)
C–Circulation and cardiovascular	Heart rate, rhythm, blood pressure
D–Neurologic disability	Consciousness level (Glasgow Coma Scale/FOUR Score), pupil size, symmetry and reactivity, peripheral limb motor and sensory function
E–Exposure and other organ systems	Temperature, renal function, electrolytes, fluid balance and urine output
F–Feeding	Enteral feeding if possible (or parenteral nutrition)
L – Lines and invasive monitoring	Invasive monitoring if indicated; central venous pressure catheter, arterial line for invasive blood pressure, pulmonary artery catheter, urinary catheter
A–Analgesia, antiepileptics, antimicrobials	Analgesia: verbal or visual scales for assessment, World Health Organization (WHO) analgesic ladder Antiepileptics: appropriate seizure control Antimicrobials: monitoring and treatment of infection
T – Venous thromboembolism prophylaxis	Mechanical prophylaxis: thromboelastic stockings, pneumatic compression devices
	Pharmacologic prophylaxis (if indicated): low-molecular-weight heparin, unfractionated heparin
H – Head up and ICP stabilization	Elevate the head above heart and protocol-driven strategy for ICP control
U – Ulcer (gastric) prophylaxis and nausea/vomiting:	Gastric protection against stress ulceration (e.g., proton-pump inhibitors) Antiemetics
G – Glycemic control	Maintain glucose within normal range (4.4–8.3 mmol/L; 80–150 mg/dL)
S – Specific neurologic monitoring and neurosurgical pathology-related concerns	Neurologic monitoring: ICP, SjO_2, $PbtO_2$, TCD Special concerns depending on neurosurgical pathology (e.g., postoperative steroids and electrolytes, osmolality and fluid balance following pituitary surgery)

ABCDEF LAT HUGS = aide-mémoire; ICP = intracranial pressure; $PaCO_2$ = arterial carbon dioxide tension; PaO_2 = arterial oxygen tension; $PbtO_2$ = brain tissue oxygen tension; SaO_2 = oxygen saturation; $SjvO_2$ = jugular venous oxygen saturation; TCD = transcranial Doppler

combined with the analgesic fentanyl (2–3 µg/kgBW) or sufentanil (0.3–0.5 µg/kgBW). Administration should be titrated and adjusted based on the patient's hemodynamic response. Thiopental was chosen in this patient because its mechanism of action reduces neuronal activity, thereby lowering $CMRO_2$. This reduction may subsequently lead to a decrease in cerebral blood flow and, consequently, a decrease in intracranial pressure (ICP). The decrease in cerebral blood flow is secondary to vasoconstriction of cerebral blood vessels.

This vasoconstriction occurs only in regions of normal brain tissue, while ischemic areas remain dilated. This condition produces a beneficial effect in the form of shunting blood flow from normal to ischemic areas, a phenomenon known as the "Robin Hood" effect or inverse steal phenomenon. The muscle relaxant used in this case was rocuronium, administered at an induction dose of 1 mg/kgBW. Rocuronium has a rapid onset, making it appropriate as a neuromuscular blocking agent for intubation. With its short duration of action, rocuronium is suitable for continuous infusion during anesthetic maintenance, as was done in this patient. Other neuromuscular blocking agents, such as vecuronium, do not cause histamine release that might trigger hemodynamic instability and do not increase cerebral blood flow that could lead to cerebral edema. Vecuronium also has minimal or no effects on ICP, blood pressure, or heart rate and is effective in patients with Space Occupying Lesions (SOL) or cerebral ischemia. Rocuronium is considered one of the best alternatives due to its rapid onset and minimal impact on intracranial dynamics.[15]

In this case, anesthetic maintenance did not involve the use of inhalational anesthetic agents because they can interfere with the motor evoked potential (MEP) and somatosensory evoked potential (SEP) waveforms on intraoperative neurophysiological monitoring devices. Although inhalational agents such as sevoflurane have cerebral vasodilatory effects and cause the least increase in cerebral blood flow (CBF) among all volatile anesthetics and also possess neuroprotective anti-apoptotic properties their use is still avoided when intraoperative neurophysiological monitoring is employed. Inhalational anesthetics can be replaced with propofol using a Target-Controlled Infusion (TCI) with the Schneider model, targeting an effect-site concentration of 3–4 mcg/mL, as done in this case. The mechanism of action of propofol includes a reduction in cerebral blood flow and cerebral metabolic rate of oxygen consumption ($CMRO_2$).

The decrease in cerebral blood flow is due to the reduction in cerebral metabolic activity, while cerebral autoregulation is preserved. Propofol also has a greater capacity to scavenge free radicals compared to thiopental. Currently, propofol is the most commonly used agent for induction and maintenance during neurosurgical procedures.[15] Dexmedetomidine was administered in this case for maintenance at a dose of 0.4–0.6 mcg/kgBW/hour. Dexmedetomidine has neuroprotective effects in cerebral ischemia and can increase catecholamine levels in both the cerebral circulation and extracellular space. Its mechanism of action involves providing compounds that reduce the release of norepinephrine in the brain, such as alpha-2 agonists, which can offer protection against brain injury caused by cerebral ischemia. Mannitol administration at a dose of 0.5–1 gram/kgBW can be performed after the dura mater is opened, thus avoiding a sudden drop in intracranial pressure (ICP), as the brain becomes slack and transmural pressure (TMP) does not rise abruptly, thereby reducing the risk of aneurysm re-rupture. In this case, mannitol was not administered because the brain was already in a relaxed state both before and after the dural opening, and a spinal lumbar drain had already been placed to manage cerebral edema.

Proper ventilation management is essential, with mild hypocapnia ($PaCO_2$ 30–35 mmHg) maintained before dural opening and relative normocapnia during controlled hypotension and clipping.3In this case the strategy was used on the assumption that the patient still had a preserved response to carbon dioxide. During aneurysm surgery the goals of anesthetic maintenance

focus on several key aspects. The brain is kept in a relaxed state so that retraction can be minimized. Transmural pressure and cerebral perfusion pressure are maintained at appropriate levels especially during periods of reduced blood flow. Both CPP and TMP are carefully lowered at specific stages such as during and after vascular occlusion. Intracranial pressure is kept stable by avoiding sudden fluctuations. At the end of the procedure the patient is expected to regain consciousness quickly so that neurological function can be evaluated without delay.[3] For aneurysm clipping procedures, intraoperative neuromonitoring methods such as electroencephalography, motor evoked potentials, and somatosensory evoked potentials are commonly preferred. Evidence from a study involving 691 patients who underwent clipping shows that changes in somatosensory evoked potentials can predict postoperative stroke with high specificity reaching about 95%. Monitoring with SEP is particularly helpful in surgeries involving the anterior circulation, while combining MEP and SEP provides better assessment for aneurysms located in the posterior circulation.

Transcranial cortical stimulation and MEP monitoring help identify motor cortex ischemia by comparing the amplitude before and after clipping, as well as detecting secondary pathway involvement due to occlusion of perforating arteries such as the middle cerebral artery and the basilar artery. This technique can identify improperly placed permanent clips.[17,18] The use of intraoperative neurophysiological monitoring is recommended without inhalational anesthetic agents, as they can interfere with the assessment of motor evoked potentials (MEP) and somatosensory evoked potentials (SEP). Therefore, inhalational agents were not administered in this patient. The role of intraoperative neurophysiological monitoring in improving neurological outcomes requires further study, with the aim of preventing dysfunction or damage to the nervous system and achieving optimal surgical results.

The Neurologic Intensive Care Unit (NICU) is a specialized intensive care unit dedicated to patients with severe neurological conditions and plays an integral role in managing cerebrovascular diseases during both the acute and perioperative periods. Understanding how to use intracranial pressure monitoring and implement appropriate interventions for elevated intracranial pressure to ensure adequate cerebral perfusion is a fundamental principle of neurocritical care. Careful management of the interaction between cerebral and systemic physiology especially in cases of impaired cerebral autoregulation is crucial for preventing secondary brain injury. The NICU is equipped with ventilators specifically designed for managing neurological diseases that involve respiratory compromise, such as Guillain-Barré syndrome, neuromuscular disorders, or severe neurosurgical cases. Intensive Care Units generally follow protocols and principles summarized by the mnemonic FAST HUG BID (Table 6).[19] For the management of critically ill patients, the Neurologic Intensive Care Unit follows a different set of protocols and principles, summarized by the mnemonic ABCDE FLAT HUGS (Table 7). 20 In conducting assessment and monitoring of patients in neurocritical care, this case showed that after evaluating the patient's clinical condition on the second day and reviewing the laboratory results, extubating was performed. On the third day, the patient's clinical condition remained within normal limits, and a follow-up contrast-enhanced CT angiography of the head revealed no significant abnormalities. Therefore, the patient was eligible for transfer to the High Care Unit.

Conclusion

Subarachnoid hemorrhage is a serious health problem worldwide with an estimated incidence of about 9 cases per 100,000 people each year. This condition carries a high risk of death and affects not only the brain but also other organs such as the lungs and the heart. Management is mainly directed at preventing rebleeding and this can be achieved through procedures such as endovascular coiling or surgical clipping. Patients who arrive with stable neurological status generally show more favorable outcomes. A non-contrast CT scan of the head is commonly used as an initial diagnostic tool. Coiling is now

widely used as a less invasive option although it cannot be applied in all situations. Clipping is usually considered for specific cases such as large aneurysms fusiform lesions aneurysms located at arterial bifurcations and those arising from the middle cerebral artery. During surgery modern intraoperative monitoring techniques can be applied to observe motor evoked potentials and somatosensory evoked potentials and this process is carried out by a neurophysiologist to ensure that neural function remains stable. Neuroanesthesiologists are expected to understand the causes mechanisms and possible complications of subarachnoid hemorrhage in detail. They also need to maintain physiological stability by controlling cerebral perfusion pressure and mean arterial pressure while preventing increases in intracranial pressure brain swelling and cerebral vasospasm throughout the perioperative period. These aspects have a strong impact on patient outcomes after surgery and require continuous observation and prompt management. Effective teamwork and clear communication between the neurosurgeon neurophysiologist and neuroanesthesiologist are essential in providing optimal care for patients with this condition.

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