JURNAL KOMPLIKASI ANESTESI
VOLUME 12 NOMOR 3, August 2025

CASE REPORT

Case Series: Lumbar Cerebrospinal Fluid Drainage in Aneurysm Clipping

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How to cite: Bayu TK, et al, Case Series: Lumbar Cerebrospinal Fluid Drainage in Aneurysm Clipping. Jurnal Komplikasi Anestesi 12(3)-2025.

Receive: July 11, 2024
Accepted: December 16, 2025

ABSTRACT

Intracranial aneurysm rupture is a medical emergency often accompanied by subarachnoid hemorrhage (SAH), which requires immediate medical intervention to reduce the risk of morbidity and mortality. This case report provides an overview of lumbar cerebrospinal fluid drainage (LCFD) as a method to reduce increased intracranial pressure and prevent or treat hydrocephalus that can occur after SAH. It is done by inserting an epidural catheter in the subarachnoid lumbar. The effectiveness of LCFD is more significant in patients with MFS 3-4. This finding supports the use of LCFD in managing SAH aneurysm to improve patients' prognosis. Management of this case involved meticulous preoperative evaluation, appropriate intraoperative intervention, and strict postoperative monitoring, including treatment of cerebral vasospasm that may arise as a complication of SAH. The results suggest that LCFD can successfully reduce complications and improve clinical outcomes in patients with ruptured aneurysms who have undergone aneurysm clipping. The success of this method emphasizes the importance of a multidisciplinary approach in the management of SAH to achieve optimal patient outcomes.

Keywords: Cerebral vasospasm, intracranial aneurysm rupture, lumbar cerebrospinal fluid drainage, SAH management, subarachnoid hemorrhage

INTRODUCTION

Intracranial aneurysm rupture is a life-threatening medical condition where prompt and accurate medical intervention is crucial to reduce morbidity and mortality risks. This condition is often associated with subarachnoid hemorrhage (SAH), which can lead to serious neurological complications and impact patient prognosis. In addressing this challenge, lumbar cerebrospinal fluid drainage (LCFD) has emerged as a promising management strategy for SAH, aiming to reduce intracranial pressure and prevent or manage hydrocephalus.[1]

This case series discusses the application of LCFD in managing patients with ruptured intracranial aneurysms undergoing clipping procedures. Through the analysis of these cases, we aim to provide valuable insights to the medical community regarding the potential of LCFD as part of post-rupture aneurysm management protocols.

All cases in this report underwent elective procedures and received consistent perioperative care. Preoperatively, comprehensive blood tests, electrocardiography (ECG), and chest X-rays were performed. Patients fasted for 8 hours and received an 18G intravenous cannula on the left dorsum of the hand, with a continuous infusion of 0.9% NaCl at a rate of 20 drops per minute. Informed consent related to the anesthesia procedure was obtained. On the day of surgery, patients were taken to the operating room, where standard ASA monitoring devices were applied. Endotracheal intubation was performed using an endotracheal tube (ETT) no. 7.0 at a depth of 20 cm.

Arterial lines were placed on the left hand, and a Central Venous Catheter (CVC) was inserted via the left subclavian vein. Anesthesia maintenance was achieved using Total Intra Venous Anesthesia (TIVA) with continuous fentanyl, propofol, and rocuronium. The LCFD catheter was inserted after intubation, with the patient in a left lateral decubitus position at the interspinal area of lumbar vertebrae 3-4 or 4-5, following aseptic procedures. Postoperatively, patients were transferred to the Surgical Intensive Care Unit (SICU) while intubated and under sedation for close monitoring. On the first day after surgery, consciousness was evaluated.

CASE 1

A 59-year-old female patient presented with a decreased level of consciousness. She was referred from a private hospital with a diagnosis of subarachnoid hematoma confirmed by computed tomography (CT scan). The patient had received intravenous therapy including Citicoline 1000 mg, Mecobalamin 500 mcg, Tranexamic Acid 1000 mg, Mannitol 40 mg, and Pantoprazole 40 mg. Consciousness was regained 4 hours later, with symptoms of left-sided limb weakness, nausea, and vomiting. Upon examination, the patient was conscious with a Glasgow Coma Scale (GCS) score of E4V5M6, experiencing neck pain with a Visual Analogue Scale (VAS) score of 1-2, and vertigo. The patient had uncontrolled hypertension.

Neurological examination revealed pupils of 3mm/3mm with a positive light reaction, no cranial nerve paralysis, and positive meningeal signs. Thoracic and abdominal examinations were normal, with good motor and sensory function (score 5/5). Laboratory findings showed an increase in leukocytes (14.6 x 10^3/µL) and D-dimer (3421 ng/mL FEU). Chest X-ray examination revealed bilateral pneumonia, cardiomegaly (CTR 0.57), and aortic elongation. The patient was diagnosed with a ruptured aneurysm at the bifurcation of the left middle cerebral artery causing SAH.

The patient underwent LCFD and craniotomy aneurysm clipping. The patient was classified as American Society of Anesthesiologists (ASA) Physical Status IV due to a recent cerebrovascular accident (CVA) (less than 3 months ago), uncontrolled hypertension, and elevated D-dimer levels. Hemodynamic stability was maintained during the 10-hour operation with systolic blood pressure (SBP) ranging from 80–180 mmHg, diastolic blood pressure (DBP) from 46-120 mmHg, heart rate (HR) of 50-70 beats per minute, respiratory rate (RR) of 16-24 breaths per minute, and end-tidal CO_2 ($EtCO_2$) of 34-36 mmHg. The total fluid intake was 3550 ml (2000 ml crystalloids, 1050 ml packed red cells (PRC), and 500 ml medication). The total fluid output was 3960 ml (2160 ml

redistribution and evaporative losses {REL}, 800 ml blood loss, and 1000 ml urine). The patient had a positive intraoperative fluid balance of 410 ml with a diuresis of 1.2 cc/kg/hour.

Postoperative evaluation after aneurysm clipping showed significant clinical improvement. The patient was sedated in the Surgical Intensive Care Unit (SICU) until Post Operation Day (POD) 1 with a GCS score of E4VtM6. Extubation was performed on POD 3, followed by transfer to the Surgical High Care Unit (SHCU), then to the Stroke Unit on POD 5, and discharge on POD 11. One month postoperatively, the patient was readmitted with complaints of recurring dizziness, with no other complaints. Digital Subtraction Angiography (DSA) evaluation showed improvement, and the patient was discharged after two days of ward care. The patient was discharged with a condition of frequent anxiety, without neurological deficits.

CASE 2

A 52-year-old female patient presented with a severe headache radiating to the neck, persisting for two days with moderate to severe intensity, unrelieved by rest. The symptoms were accompanied by a single episode of projectile vomiting. The patient appeared lethargic without neurological deficits. Upon examination, the patient reported a reduction in headache severity. Neurological examination revealed 3mm/3mm pupils with a positive light response, no cranial nerve paralysis, and positive meningeal signs. Thoracic and abdominal examinations were normal, with good motor and sensory function (score 5/5).

Laboratory findings indicated leukocytosis. Chest radiography showed normal lungs without abnormalities, but there was an enlargement of the heart with a cardiothoracic ratio (CTR) of 0.61. The patient was diagnosed with a right anterior cerebral artery aneurysm causing SAH and grade 2 hypertension. The patient underwent LCFD and attempted craniotomy aneurysm clipping, which failed, leading to craniotomy aneurysm wrapping. The patient was categorized as ASA Physical Status IV due to a recent CVA (less than 3 months ago) and grade 2 hypertension.

Hemodynamic parameters during the 9 hours and 45 minutes of surgery were as follows: SBP 110-130 mmHg, DBP 50-80 mmHg, HR 60-80 beats per minute, RR 18-20 breaths per minute, and EtCO2 29-32 mmHg. The total fluid intake was 4950 ml (3900 ml crystalloids, 500 ml PRC, and 550 ml medication). The total fluid output was 4820 ml (1920 ml REL, 500 ml blood loss, and 2400 ml urine). The patient had a positive intraoperative fluid balance of 130 ml with a diuresis of 4.1 cc/kg/hour.

Postoperative evaluation after aneurysm clipping showed significant clinical improvement. The patient was sedated in the SICU until POD 1 with a GCS score of E4VtM6. Extubation was performed on POD 1, followed by transfer to the SHCU on POD 2, and discharge on POD 11. One week postoperatively, the patient attended the clinic with complaints of heaviness in both legs and was able to walk with assistance indoors. The patient also experienced right cranial nerve VII palsy of the lower motor neuron (LMN) type, without neck stiffness.

CASE 3

A 67-year-old male patient presented with a sudden onset of severe headache four days before admission. The condition deteriorated, leading to loss of consciousness. Head CT scan revealed intracranial hemorrhage. During intensive care unit treatment, the patient experienced a temporary improvement in consciousness. However, he often appeared drowsy, only opening his eyes upon auditory stimulation.

Neurological examination showed isochoric pupils at 3mm/3mm with positive light reflex. Left-sided cranial nerve VII palsy was observed, without meningeal signs. Chest examination was normal, the abdomen was soft with good peristalsis, and no edema was present, but there was evidence of left-sided hemiparesis. Laboratory findings indicated several abnormal values. Leukocyte count increased to 12.0 x 10^3/µL. D-dimer levels rose sharply to 1223 ng/mL FEU. Blood urea nitrogen (BUN) increased to 26 mg/dL. Calcium levels decreased to 2.07 mmol/L, and magnesium levels increased to 2.5 mg/dL. HBsAg testing returned reactive. Chest

radiography showed normal lungs without abnormalities, but cardiac enlargement was noted with a cardiothoracic ratio (CTR) of 0.61. The patient was diagnosed with a ruptured anterior communicating artery aneurysm causing right frontal lobe intracerebral hematoma (SICH) and obstructive hydrocephalus due to intraventricular hemorrhage (IVH). The patient underwent LCFD, decompressive craniotomy for hematoma evacuation, and craniotomy aneurysm clipping. The patient was classified as ASA Physical Status IV due to a recent CVA (less than 3 months ago) with left-sided hemiparesis, elevated D-dimer levels, and reactive HBsAg.

Hemodynamic parameters during the 8-hour surgery were as follows: SBP 98–120 mmHg, DBP 58–75 mmHg, HR 49–65 beats per minute, RR 12-16 breaths per minute, and EtCO2 22–30 mmHg. The total fluid intake was 3300 ml (1550 ml crystalloids, 500 ml PRC, and 750 ml medication). The total fluid output was 4575 ml (975 ml REL, 1200 ml blood loss, and 2400 ml urine). The patient had a negative intraoperative fluid balance of 1275 ml with a diuresis of 4.9 cc/kg/hour.

Postoperative evaluation after aneurysm clipping did not show significant clinical improvement. The patient was sedated in the SICU until POD 1 with a maximum GCS score of E3VtM5. A decline in GCS led to the decision to perform Percutaneous Dilatational Tracheostomy (PDT) on POD 5, and he was transferred to palliative care on POD 7 with a GCS of E1VtM1. The patient was declared deceased on POD 10.

DISCUSSION

Intracranial aneurysm is a critical brain vascular disorder, classified based on morphology and dimensions. Saccular aneurysms have a diameter of less than 2.5 cm, while giant aneurysms can exceed 10 cm. Other types of aneurysms include fusiform, mycotic, and traumatic. Cerebral aneurysms typically occur at arterial bifurcations, especially in the anterior circle of Willis, with 10% to 30% of patients having multiple aneurysms. Although the reported incidence of saccular aneurysms is 5%, only a small proportion will experience complications. Ruptured saccular aneurysms are the leading cause of SAH, with acute mortality around 10%, and up to 25% of patients die within 3 months due to delayed complications. Additionally, up to 50% of survivors experience neurological deficits.[1]

The incidence of intracranial aneurysms is approximately 1 per 10,000 individuals, more commonly identified in the sixth decade of life. The prevalence of these aneurysms is higher in males up to the age of 50, after which the prevalence in females becomes higher. One in six patients will experience death due to SAH immediately following rupture. Of those hospitalized, 25% will die, and another 50% can expect full recovery. Risk factors for aneurysms and SAH include connective tissue disorders such as polycystic kidney disease and Ehlers-Danlos syndrome, cerebral trauma, infections, tumors, hypertension, smoking, substance abuse like cocaine, and a family history of aneurysms. Therefore, clinical management focuses on the prevention of rupture, although many patients only seek care after a rupture has occurred.[1,2]

Treatment of intracranial aneurysms can be conducted through surgical or neuroradiological intervention. These interventions may be performed electively for prevention or as an emergency response following hemorrhage. Meanwhile, other non-traumatic hemorrhages are typically managed with a medical approach. Surgical intervention for intracranial aneurysms can be undertaken using either an early or late approach. Early intervention, occurring within the first 24–48 hours after SAH), involves aneurysm clipping techniques and offers the advantage of preventing rebleeding. Furthermore, early intervention also contributes to a reduction in the incidence of vasospasm, a decrease in medical complications, and hospitalization costs. On the other hand, late interventions are usually performed around 2 weeks after the occurrence of SAH.[1,2]

Even when surgical treatment is successful, delayed ischemic neurological deficits that result in permanent neurological injury or death can occur in patients who experience vasospasm complications. Because the incidence and

Table 1. Fisher Grades for Computed Tomography Findings in Subarachnoid Hemorrhage

Grade	CT Findings
1	No blood detected
2	Diffuse thin layer of subarachnoid blood (vertical layers or clots <1mm thick)
3	Localized clot and/or vertical layer of subarachnoid blood (vertical layers or clots ≥1mm thick)
4	Intracerebral or intraventricular blood with diffuse or no subarachnoid blood

severity of vasospasm are associated with the amount of subarachnoid blood present, CT scan findings are often categorized according to the Fisher grading system. Despite criticisms of the Fisher grading and proposed modifications, it remains the principal method for describing CT findings related to clot burden and the risk of vasospasm in aneurysmal SAH.[3]

Comprehensive preoperative evaluation is essential before surgical procedures, involving a review of medical history, physical examination, laboratory, and radiology findings. ECG examinations often detect stress-induced myocardial injury or cardiomyopathy. Elevated cardiac troponin levels can serve as an indicator of poor prognosis. Patients with normal intracranial pressure (ICP) are typically sedated to prevent rebleeding, while those with high ICP should avoid premedication that may lead to hypercapnia. Neurogenic pulmonary edema and cardiac involvement can occur in response to SAH. Chronic medical conditions such as hypertension, diabetes mellitus, and chronic kidney insufficiency must be considered in anesthesia planning. The risk of rebleeding and vasospasm is a critical complication after SAH, with a higher risk in women with poor medical conditions and high blood pressure. Surgical decision-making should account for these potential complications as well as the possibility of electrolyte disturbances and myocardial infarction.[2,4]

In the intraoperative management of intracranial aneurysms, the primary focus is on preventing rupture or rebleeding and avoiding factors that may trigger cerebral ischemia or vasospasm. Rigorous intraarterial pressure monitoring and the avoidance of sudden blood pressure increases during intubation or surgical stimulation are key steps. Prudent intravascular volume loading is necessary to maintain surgical anesthesia levels without excessive blood pressure reduction. General anesthesia with opioid infusion and volatile anesthetics is standard practice, with special consideration given to the use of heparin and radiological contrast for endovascular procedures. Effective communication with the surgical team is crucial, particularly regarding activated clotting time and the need for protamine reversal. Anesthesiologists must be prepared to manipulate and monitor blood pressure accurately.[2,4] In craniotomy procedures, the post-dural opening use of mannitol is essential to facilitate surgical exposure and reduce the need for retraction. Controlled hypotension can be leveraged to decrease transmural tension on the aneurysm, facilitating the clipping process. Neurophysiological monitoring is implemented for early detection of cerebral ischemia during surgery.[2]

The primary objective of general anesthesia induction is to prevent an increase in transmural pressure that could potentially cause aneurysm

Table 2. Modified Fisher Grades for Computed Tomography Findings in Subarachnoid Hemorrhage

Grade	No SAH	Focal or Diffuse Thin SAH	Focal or Diffuse Thick SAH	IVH	
0	+	-	-	-	No subarachnoid hemorrhage (SAH); no intraventricular blood
1	-	+	-	-	Thin diffuse or focal subarachnoid blood but no intraventricular blood
2	-	+	-	+	Thin diffuse or local subarachnoid blood with intraventricular blood

rupture. Intravenous administration of lidocaine aims to inhibit the sympathetic response during the intubation process. Anesthesia maintenance is achieved through a balanced approach, and postoperative neurological monitoring becomes crucial for the early detection of complications such as ICH. Mild hyperventilation performed after dural opening can enhance brain relaxation, with the administration of mannitol or diuretics as needed to create optimal operative conditions.[2,4]

The timing of patient extubation following intracranial aneurysm surgery is contingent upon a multitude of factors, including preoperative medical conditions, neurological status, the severity of SAH and vasospasm, surgical procedures, and the volume of blood loss. Extubation is typically performed promptly post-surgery to facilitate rapid neurological evaluation. However, the intraoperative use of barbiturates and antiepileptics may delay this process. Patients with asymptomatic intracranial aneurysms undergoing elective surgery are often extubated in the operating room to diminish sympathetic release and cough reflex. If immediate extubation is not feasible, a reduction in anesthesia level is necessary to allow for neurological examination.

Delays in recovery may necessitate a CT scan by the surgical team to rule out complications such as intracranial hemorrhage or pneumocephalus. Patients are usually admitted to the intensive care unit with stringent monitoring, where neurological examinations are conducted periodically, and transportation is carefully managed to enable continuous monitoring and resuscitative measures if required.[4]

The mechanism of cerebral vasospasm occurring after SAH is not fully understood, but it is suspected to be related to the accumulation of vasoactive substances in the basal cisterns, inducing vasospasm in the arteries, reducing blood flow, and causing ischemic changes. Clinical vasospasm typically appears between 3–4 days after SAH and peaks between 7–10 days. Clinical manifestations include decreased consciousness and the emergence of focal neurological deficits after the 4th day of SAH, often accompanied by headaches, meningismus, and fever. The onset of vasospasm can occur suddenly or gradually.[1,4]

To prevent rebleeding after SAH, the following measures are recommended:[1]

1. Blood pressure control: Maintaining blood pressure within a target range to prevent stress on cerebral vessels.
2. Maintenance of cerebral perfusion pressure: Ensuring adequate blood flow to the brain to prevent ischemia.
3. Adequate control of pain and anxiety: Managing discomfort and stress which can contribute to increased blood pressure and potential rebleeding.
4. Adequate seizure control: Preventing seizures which can increase intracranial pressure and the risk of rebleeding.
5. Prophylaxis against hypovolemia and vasospasm: Maintaining proper fluid balance and preventing the narrowing of blood vessels which can lead to ischemia.
6. Early surgical or endovascular obliteration of the aneurysm: Addressing the source of bleeding to prevent further hemorrhage.
7. Treatment with calcium channel blockers to prevent vasospasm: Using medication to relax blood vessels and improve blood flow.
8. Treatment of vasospasm with Triple-H therapy (hypertension, hypervolemia, and hemodilution): A recognized treatment to prevent the progression from mild vasospasm-induced ischemia to infarction.

The management of cerebral vasospasm during the perioperative period is a critical aspect that relies on blood pressure regulation to maintain adequate cerebral perfusion pressure (CPP). Triple-H therapy, which includes hemodilution, controlled hypertension, and hypervolemia, is often applied although there is still debate regarding its effectiveness. Pharmacological agents such as nimodipine, magnesium, and statins are used to reduce mortality and morbidity post-vasospasm. Radiological interventions, including angioplasty and intra-arterial vasodilator injections, are also implemented as efforts to overcome vasospasm. Complications that may arise from Triple-H therapy include rebleeding and cerebral ischemia.[1,4]

The use of temporary clips during surgery aims to isolate the aneurysm but may cause focal ischemia. Neuroprotective interventions, such as mild hypothermia or pharmacological therapy, have not consistently proven to improve neurological outcomes. After the aneurysm clipping process, an increase in mean arterial pressure (MAP) is necessary to enhance collateral perfusion and prevent ischemia.[1,4] The predictive factors for post-SAH mortality include poor neurological status at hospital admission, advanced age, comorbidities, hypertension, and findings on CT scans indicating extensive bleeding within the brain parenchyma or ventricles. These conditions indicate an increased risk for unfavorable prognosis and require prompt and accurate medical interventions to reduce the potential for serious complications or death. Rebleeding and basilar aneurysms are also significant indicators of high risk.[1]

Lumbar puncture (LP), or spinal tap, is a minimally invasive procedure used for diagnostic and therapeutic purposes. The primary goal of this procedure is to obtain CSF samples, which play a crucial role in diagnosing infections, confirming subarachnoid hemorrhage, and diagnosing various neurological conditions such as normal pressure hydrocephalus and idiopathic intracranial hypertension. Additionally, the placement of LCFD aims to divert CSF, which is beneficial during intracranial procedures and in managing postoperative complications, including CSF leaks. LP is typically performed at the patient's bedside using anatomical landmarks or with the aid of ultrasonography for precise identification in patients with morbid obesity, or in a radiological setting using contrast agents to assist in diagnosing certain conditions, making this technique a critical skill in intensive care.[5]

Before performing a lumbar puncture, it is crucial to evaluate the patient's coagulation profile, including platelet count and relevant medical history such as renal failure, cancer, and the use of oral anticoagulants. Brain imaging is also necessary to exclude brain lesions that may cause changes in mental status, such as subdural hematoma, epidural hematoma, tumors, and meningitis, which are often accompanied by cerebral edema. Conditions such as non-communicating hydrocephalus, ruptured cerebral aneurysm, and Chiari malformation are relative contraindications, and the procedure must be conducted with caution. Patients with elevated PT, PTT, and INR values should not undergo a lumbar puncture until these values are corrected. Lumbar puncture can be performed on patients taking oral aspirin, but platelet transfusion may be required if CSF sampling is imperative. Generally, a platelet count of 50,000 per cubic millimeter or higher is considered safe. Any infectious process or disease located in the lumbar area where the lumbar puncture will be performed is also a contraindication. Suspected acute spinal column trauma is also a contraindication for performing a lumbar puncture.[5]

Informed consent is an essential prerequisite before carrying out a medical procedure, encompassing a comprehensive explanation of all associated risks and potential dangers. The administration of sedation is often necessary to ensure the patient remains calm and in the correct position during the procedure, while local anesthesia is applied to eliminate the sensation of pain. The equipment required for this procedure includes sterile gloves, a surgical cap, a surgical mask, a lumbar puncture tray, sedative analgesic drugs, and a LCFD kit with a drainage bag. The lateral recumbent position is considered to reflect the actual opening pressure needed for accurate diagnosis, whereas the sitting position with the head down may be technically easier to perform but does not provide a reliable opening pressure reading.[5]

The preparation for LCFD is carried out in a manner similar to that of a LP, where the patient is placed in the lateral recumbent position. The LCFD kit is prepared with a 14–16 gauge Tuohy needle, which facilitates the insertion of the catheter into the intrathecal space. Before the procedure, the tubing is flushed with a sterile saline solution. After gaining spinal access between the fourth and fifth lumbar vertebrae (L4-L5) and ensuring adequate CSF flow, the catheter is inserted through the needle into the thecal sac. Once in place, the catheter is secured

with stitches to the skin and connected to the LCFD system that has been filled with sterile saline solution. If there is an obstruction to the flow, the needle and tubing must be removed, and the procedure carefully repeated.[5]

Lumbar puncture is a procedure that is generally safe, and serious complications are very rare. The use of atraumatic needles such as the Quincke needle can reduce the risk of complications. Post-LP headache is the most frequently reported complaint, usually due to intracranial hypotension caused by a decrease in CSF pressure. Lying flat, administering analgesics, and caffeine can help alleviate the headache. If the headache persists, an epidural blood patch may be administered as treatment. Nerve root irritation can occur if the needle directly touches a nerve, causing pain to radiate to the leg or groin. In this case, the needle stylet is replaced, and the needle is withdrawn slowly to avoid further damage. Infection or meningitis are rare complications, and the use of broad-spectrum antibiotics is recommended until specific culture results are obtained. Cerebral herniation is the most serious but very rare complication that can occur due to a sudden decrease in ICP. This risk is increased in patients with already high ICP, such as in cases of intracranial mass or bleeding. Therefore, a CT scan examination before LP is crucial to identify any mass or bleeding that may increase the risk of herniation.[5]

The multicenter study conducted in 20236 was a pragmatic randomized clinical trial that involved 287 patients across 19 hospitals in Germany, Switzerland, and Canada. Patients with aneurysmal SAH were given standard care or additional LCFD. LCFD was initiated within 72 hours after the subarachnoid hemorrhage. The rate of unfavorable neurological outcomes was 32.6% in the LCFD group and 44.8% in the standard care group. Patients treated with LCFD had fewer secondary infarcts at hospital discharge. Prophylactic LCFD after aneurysmal SAH reduced the burden of secondary infarcts and lowered the rate of unfavorable outcomes at 6 months. The findings support the use of LCFD following aneurysmal subarachnoid hemorrhage.[6]

The retrospective study conducted in 20237 involved 438 patients with aneurysmal SAH from January 2014 to December 2020. Indications for immediate LCFD were aneurysmal SAH with a modified Fisher grade III or higher without dense intraventricular hemorrhage with obstructive hydrocephalus requiring extraventricular drainage or large intracranial hemorrhage necessitating immediate decompression. The in-hospital mortality for patients with immediate LCFD was 7.4% compared to 14.4% for patients without lumbar cerebrospinal drainage, and this difference was significant. Vasospasm occurred in 10% of patients with immediate LCFD and 16.7% in controls. Hydrocephalus requiring extraventricular drainage occurred in 10.9% of the LCFD group and 28.7% of the control group. Immediate lumbar cerebrospinal drainage is a viable option for treating selected patients with aneurysmal SAH.[7]

The study conducted in 20198 used a retrospective approach to evaluate the effectiveness of LCFD in patients with aneurysmal SAH undergoing aneurysm clipping. LCFD was found to reduce the risk of hydrocephalus and improve Glasgow Outcome Scale (GOS) scores at hospital discharge and during the 3-month follow-up. In the group with high Modified Fisher Scale (mFS) scores (3-4), LCFD demonstrated significant benefits in reducing the risk of cerebral vasospasm, delayed cerebral infarction, and hydrocephalus. Lumbar cerebrospinal fluid drainage is effective in reducing postoperative complications and enhancing clinical outcomes in patients with high mFS (3 and 4). However, there was no significant benefit of LCFD in terms of complications and outcomes for patients with low mFS (0-2). LCFD is recommended as an additional treatment but not mandatory for aneurysm clipping in SAH patients with low mFS.[8]

Lumbar cerebrospinal fluid drainage after aneurysmal SAH offers significant clinical benefits, including the reduction of secondary infarcts and improved neurological outcomes. Immediate LCFD can be considered a viable treatment option for selected patients. For patients at lower risk, it can be used as an additional treatment. These findings suggest

the integration of LCFD into aneurysmal SAH treatment protocols to enhance patient outcomes.

CONCLUSION

This report reveals that meticulous anesthesia management, controlled postoperative recovery, and strict clinical outcome monitoring are key to improving patient prognosis. Intracranial aneurysm is a serious condition with a high risk of post-rupture complications, and effective management through surgery or neuroradiological intervention is crucial. The Fisher grading system is still considered the primary method for assessing CT findings and vasospasm risk. Careful intraoperative management, including blood pressure monitoring and anesthesia use, as well as thorough preoperative evaluation, are essential to reduce mortality and morbidity. Previous research supports the use of LCFD after SAH to improve neurological outcomes and reduce complications. An in-depth discussion of intracranial aneurysm, including incidence, risk factors, and treatment strategies, provides valuable insights into effective therapeutic approaches.

REFERENCES

1. Tripathi P. Recent Advances in Anesthesiology. In: John AD, eds. Anesthesia: A Topical Update – Thoracic, Cardiac, Neuro, ICU, and Interesting Cases.. 2nd ed. Bentham Books. 2018:104–47. Doi: 10.2174/9781681087238118020l
2. Butterworth IV JF, Mackey DC, Wasnick JD. Anesthesia for neurosurgery. In: Chestnut DA, Wong CA, Tsen LC, et al., editors. Morgan & Mikhail's Clinical Anesthesiology. 7th ed. New York, NY: McGraw-Hill Education; 2022:966-1002. doi: 10.1036/9781260461884.
3. Pong RP, Lam AM. Anesthetic management of cerebral aneurysm surgery. In: Cottrell JE, Patel PM, Soriano SG, editors. Cottrell & Patel's Neuroanesthesia. 7th ed. Philadelphia, PA: Elsevier; 2025:244–71. Doi: 10.1016/B978-0-323-93273-8.00013-4.
4. Longnecker DE, Mackey SC, Newman MF, Sandberg WS, Zapol W. Neuroanesthesia. In: Longnecker DE, Mackey SC, Newman MF, Sandberg WS, Zapol W, editors. Anesthesiology. 3rd ed. New York, NY: McGraw-Hill Education; 2017:807–47. Doi: 10.1036/9780071836511.
5. Wiercinski L, Christiansen C. Lumbar puncture and drainage. In: Wiercinski L, Christiansen C, editors. Interventional Critical Care. Cham: Springer; 2021:279–88. Doi: 10.1007/978-3-030-64661-5_27.
6. Wolf S, Mielke D, Barner C, Malinova V, Kerz T, Wostrack R, et al. Effectiveness of lumbar cerebrospinal fluid drain among patients with aneurysmal subarachnoid hemorrhage: A randomized clinical trial. JAMA Neurol. 2023;80(8):833–42. Doi: 10.1001/jamaneurol.2023.1792.
7. Kim DY, Cho YH, Kim S, Jeong JH, Choi JH, Kang M, et al. Feasibility of prompt lumbar drainage in patients with aneurysmal subarachnoid hemorrhage. World Neurosurg. 2023;175:e1032-e1040. Doi: 10.1016/j.wneu.2023.04.066.
8. Fang Y, Shao Y, Lu J, et al. The Effectiveness of Lumbar Cerebrospinal Fluid Drainage in Aneurysmal Subarachnoid Hemorrhage with Different Bleeding Amounts. Neurosurg Rev. 2020;43(2):739–47. doi: 10.1007/s10143-019-01116-1.