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Case Report

The Use Dexmedetomidine as a Total Intravenous Anesthesia–Propofol Adjuvant for Aneurysm Clipping

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Abstract

Intracranial aneurysm is a cerebrovascular disease with a high mortality rate, particularly in cases of rupture. Aneurysm clipping surgery is one of the definitive management methods; however, it involves significant hemodynamic fluctuations that may lead to intraoperative complications and worsen prognosis. Hemodynamic stability and rapid anesthetic recovery are crucial aspects for the success of this procedure. We report a case of a 57-year-old female with a saccular aneurysm in the right M1 segment of the middle cerebral artery, scheduled for aneurysm clipping surgery. The patient had previously undergone decompressive craniectomy and hematoma evacuation due to non-traumatic intracranial hemorrhage, which was not initially diagnosed as an aneurysm, and showed no significant improvement postoperatively. In anesthetic management, dexmedetomidine was used as an adjuvant to maintain hemodynamic stability and support rapid recovery. Throughout the procedure with TIVA-Propofol, dexmedetomidine effectively maintained stable blood pressure without episodes of hypertension, hypotension, or bradycardia. The patient did not experience significant intraoperative complications, and postoperative recovery was optimal. This emphasizes the critical role of dexmedetomidine within modern anesthetic approaches to the management of intracranial aneurysm cases.

Keywords: Aneurysm clipping, dexmedetomidine, intracranial aneurysm

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Introduction

Intracranial aneurysms is a common cerebrovascular disease and a primary cause of subarachnoid hemorrhage. Its true prevalence is difficult to assess due to the large number of asymptomatic aneurysmal lesions. Generally, the prevalence in the adult population ranges from 0.2% to 9.9%.[1] In individuals without risk factors, the incidence is only about 2%. If an aneurysm ruptures, approximately one-third of patients do not survive, and only half of the survivors can live independently.[2] Approximately 85% of intracranial aneurysms occur in the anterior circulation and are classified by their location. The most common locations are the anterior communicating artery, the bifurcation of the middle cerebral artery, the ophthalmic artery, and the bifurcations of the internal carotid artery.[2] The management of intracranial aneurysms generally involves surgical clipping and interventional embolization procedures. However, intense stimuli such as anesthetic and surgical interventions can trigger fluctuations in the circulatory system, potentially leading to aneurysm rupture and hemorrhage. This condition directly affects the success of the procedure and the patient's prognosis. Therefore, the role of the anesthesiologist is crucial in establishing good coordination with the surgeon during the

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procedure, maintaining hemodynamic stability, minimizing the stress response to painful stimulation, and enhancing patient safety.[1] Despite advances in anesthetic techniques, optimal strategies to achieve stable hemodynamics during aneurysm surgery remain a clinical challenge and are still under investigation.

Although rare, the incidence of aneurysm rupture during anesthetic induction is reported to be around 2%, and even less than 1% with modern anesthetic techniques. It is usually triggered by a sudden increase in blood pressure during tracheal intubation and is associated with high mortality.[3] Thus, the goal of anesthetic induction in cerebral aneurysm surgery is to reduce the risk of aneurysm rupture by minimizing transmural pressure while maintaining adequate cerebral perfusion pressure (CPP). Ideally, CPP should be maintained at the same level as the preoperative period during induction, but this is not always possible. As a general principle, blood pressure should be reduced by 20–25% below the baseline value. To reduce the incidence of aneurysm rupture or ischemia, changes in transmural pressure or cerebral perfusion pressure must occur gradually, not abruptly.[3]

Dexmedetomidine is a selective α-2 agonist that has sympatholytic, sedative, amnestic, and analgesic effects.4 Dexmedetomidine can help maintain hemodynamic stability to reduce the risk of complications caused by blood pressure fluctuations.[5] Additionally, dexmedetomidine is known to have neuroprotective effects, reduce anesthetic and opioid requirements, decrease the need for hypertensive medications, and allow for early emergence.6 However, clinical evidence regarding its perioperative use in intracranial aneurysm surgery, particularly in terms of real-world anesthetic management and outcomes, remains limited. In this case report, the author discusses the use of dexmedetomidine in the anesthetic management of a patient undergoing intracranial aneurysm clipping surgery.

Case

Anamnesis/ disease history

A 57-year-old female patient, weighing 60 kg with a height of 160 cm (Body Mass Index/ BMI): 23.4, presented with a diagnosis of post-decompressive craniectomy and evacuation for intracranial hemorrhage and subarachnoid hemorrhage, suspicious for a ruptured saccular aneurysm of the right middle cerebral artery. The patient had a history of hypertension for 7 years prior to hospital admission, with a highest recorded systolic blood pressure of 200 mmHg, which was uncontrolled. The patient also had a history of smoking for the past 20 years. From a third-party history (heteroanamnesis), it was found that the patient presented with a decreased level of consciousness 3 hours before hospital admission; the family stated the patient appeared drowsy and was difficult to arouse. The complaint began with a sudden, worsening headache accompanied by three episodes of vomiting, particularly since the day before admission. A decompressive craniectomy and evacuation surgery were performed under balanced anesthesia, using oxygen + air, sevoflurane 0.8-1 vol%, and intermittent rocuronium. A total of 10 cc of blood clot and 10 cc of lysed blood were evacuated. A tracheostomy was performed immediately post-surgery, and the patient was subsequently cared for in the ICU. The cause of the non-traumatic hemorrhage had not yet been confirmed.

Physical Examination

On physical examination, the patient's GCS was E2M4Vt. Vitals were: blood pressure 127/73 mmHg, heart rate 96 beats/min, respiratory rate 22 breaths/min, temperature 36.7°C, and oxygen saturation 99% with oxygen delivery at 3 lpm via a T-piece. The neurological examination revealed isocoric pupils measuring 3mm/3mm with a positive light reflex; there was an impression of left hemiparesis with pathological reflexes present in the left extremities. After 4 days of treatment without significant improvement, a decision was made to perform a follow-up examination with digital subtraction angiography (DSA) to confirm the cause of the bleeding. The DSA results revealed a saccular aneurysm in the middle cerebral artery, and a decision was made to perform an aneurysm clipping operation.

Supporting Examination

Preoperative laboratory and arterial blood gas analysis results showed in the table below (Table 1). Patient had normal hemostasis profile. A chest X-ray revealed bronchopneumonia, left pleural effusion, and cardiomegaly (Figure 1). The DSA showed a saccular aneurysm on the M1 segment of the right middle cerebral artery with a neck size of 0.23 cm, a dome size of 0.41x0.7 cm, and a dome-to-neck ratio of 3 with an anterosuperior projection (Figure 2). The patient was assessed as ASA physical status IV, post-decompressive craniectomy and evacuation for intracranial hemorrhage and subarachnoid hemorrhage, and scheduled to undergo an aneurysm clipping procedure.

Table 1. Laboratory Examination and Arterial Blood Gas Analysis Result

Variable	Value
Hb	9.7 g/dL
Ht	29.1%
Sodium	133 mmol/L
Arterial blood gas analysis	
pH	7.448
pCO_2	30
pO_2	125.2
HCO_3	20.4
BE	-2.4
Oxygen saturation	99.5%.

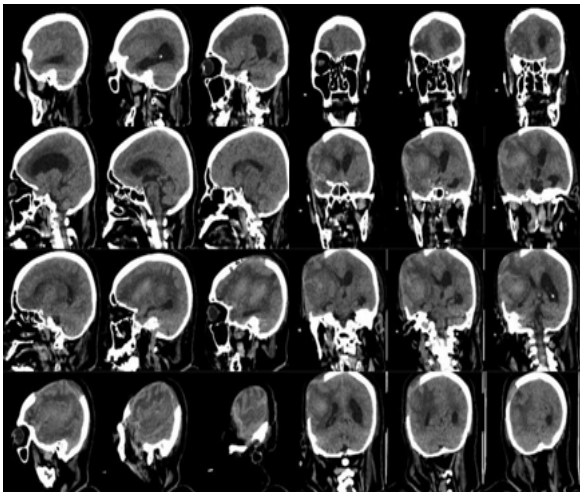

Figure 1. Patient's CT Image

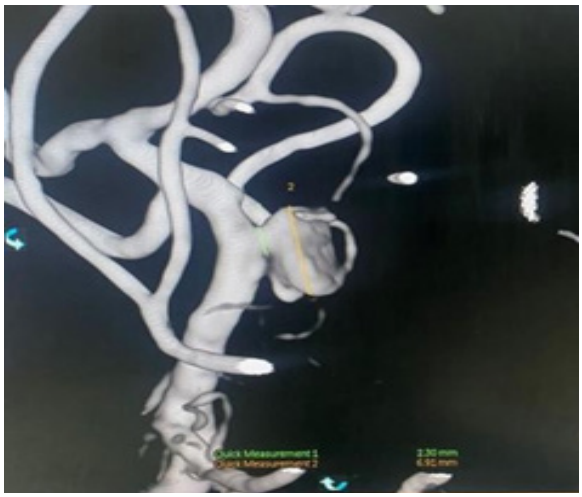

Figure 2. Results of digital subtraction angiography showing the presence of a saccular aneurysm.

The Hunt and Hess (HH) and World Federation of Neurological Surgeons (WFNS) classifications are used to assess clinical severity, and the Fisher scale is used to assess radiological findings, which helps in determining the patient's prognosis and management. This patient was classified as Hunt and Hess grade IV, for which the mortality rate reaches 23.6% (Table 2).While in the ICU, the patient's blood pressure was controlled with amlodipine 10 mg and candesartan 32 mg, both administered orally once daily. After consent for the procedure was obtained, preparations were made, including keeping the patient NPO (nothing by mouth) for 6 hours before surgery and preparing blood products.

Anesthesia management

The anesthetic procedure began with the administration of dexmedetomidine at 0.5 mcg/kg for 10 minutes before induction. Induction was performed using fentanyl 100 mcg, propofol 100 mg, and rocuronium 50 mg. The anesthesia machine circuit was connected to the tracheostomy cannula. Anesthesia was maintained with a propofol infusion at 5 mcg/kg/hour and a dexmedetomidine infusion starting at 0.2 mcg/kg/hour, with maintenance fluids of Ringer's Fundin at 80 cc/hour. The patient was placed in the supine position. During the surgery, monitoring included blood pressure, central venous pressure, end-tidal CO_2, oxygen saturation, and urine output. Before the incision, a scalp block was performed

Table 2. Hunt and Hess Classification

Grade (WFNS/ Hunt & Hess)	Hunt & Hess Symptoms	GCS	Hemiparesis/Aphasia (WFNS)
I	Asymptomatic, or mild headache and neck stiffness	15	Absent
II	Severe headache, neck stiffness, cranial nerve palsy (optional)	13–14	Absent
III	Drowsy, minimal neurological deficit	13–14	Absent
IV	Stupor, severe neurological deficit (hemiparesis), vegeta-tivedisturbances	7–12	Present or absent
V	Coma, signs of decerebration	3–6	Present or absent

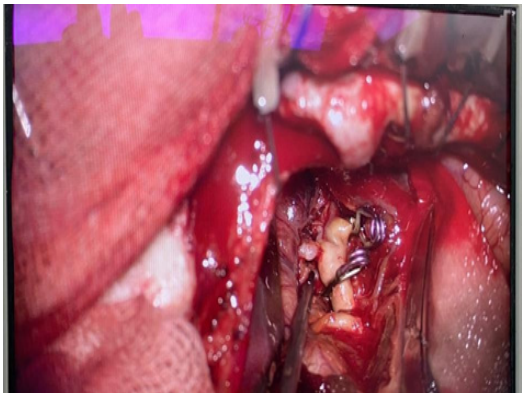

Figure 3. Clipping Aneurisma

using 0.25% levobupivacaine. The operation began with the placement of an external ventricular drain (EVD). Before direct clipping (Figure 3), the surgeon performed temporary clipping with a mini temporary clip.

Prior to temporary clipping, brain protection was provided with the administration of mannitol at 0.25 mg/kg. The temporary clipping lasted for 12 minutes, during which hemodynamic conditions remained stable. An intraluminal thrombus within the aneurysm was found, and the aneurysm was

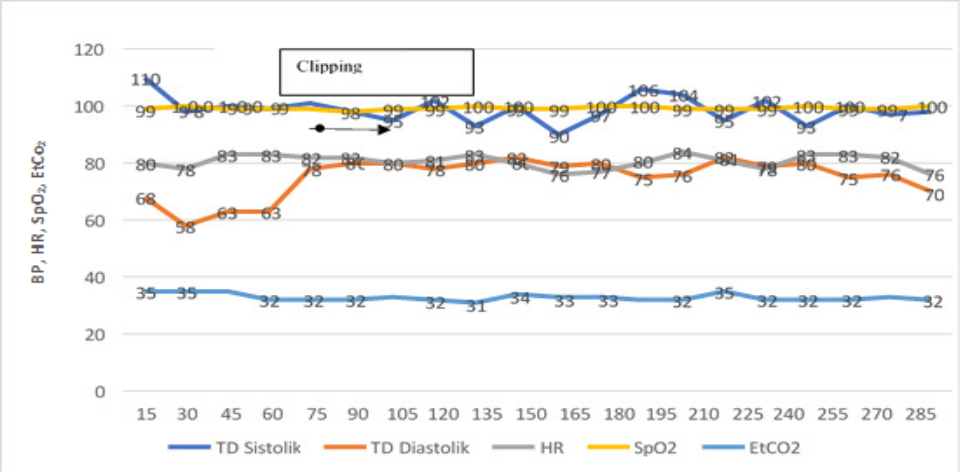

Figure 5. Patient's Monitoring day 2 – 5 Post-Operative

located at the bifurcation of the M1 segment and the lenticulostriate artery. During the operation, hemodynamics was stable with no episodes of hypotension, hypertension, or bradycardia (Figure 4). Intraoperative blood loss was 400 cc. Intraoperative fluid input consisted of 1000 cc of Ringerfundin, 500 cc of Ringer's Lactate, 400 cc of Packed Red Cells (PRC), and 300 cc of Fresh Frozen Plasma (FFP). The patient's intraoperative urine output was 600 cc over 5 hours.

Post-operative Anesthesia Management

Post-procedure, the patient remained on mechanical ventilation via the tracheostomy cannula and was managed in the Intensive Care Unit (ICU), with the head of the bed elevated 30 degrees. Analgesia was provided with a fentanyl infusion at 25 mcg/hour and a dexmedetomidine infusion at 0.2 mcg/kg/min. In the ICU, the patient's blood pressure was 104/60 mmHg and respiratory rate was 18 breaths/minute, with ventilator settings of VC-SIMV mode, VT 450, RR 14, PEEP 5, and FiO_2 40%, resulting in an oxygen saturation of 100%. The patient received nimodipine 60 mg every 4 hours to reduce the risk of delayed cerebral ischemia. A chest X-ray was performed on the second postoperative day and compared with the previous one. It showed no improvement in the left pleural effusion and suspected left lung atelectasis, while the bilateral bronchopneumonia appeared to have improved. The patient's pneumonia was managed with a ventilator strategy of low tidal volumes and high PEEP, and antibiotics were administered according to the hospital's local antimicrobial resistance patterns.

On the third postoperative day, the dexmedetomidine was discontinued, and the ventilator mode was switched to CPAP. Hypokalemia was noted (2.9 mEq/L), which was corrected, and a heparin infusion of 5000 units/24 hours was started. On day 4, hemodynamics was relatively stable, and on day 5, the tracheostomy cannula was connected to a T-piece in preparation for a planned transfer to the semi-intensive care unit. On day 6, the patient was transferred to the semi-intensive care unit. The patient's condition improved, and following decannulation (removal of the tracheostomy tube), the patient's level of consciousness also improved (Figure 5).

Discussion

An intracranial aneurysm is a severe arterial disorder characterized by the thinning and

widening of a cerebral blood vessel wall, which can be fatal. The mortality rate following aneurysm rupture is very high.[9] In this case, the patient presented with symptoms of intracranial hemorrhage, leading to a decompressive craniotomy and evacuation surgery. However, the exact cause of the non-traumatic hemorrhage was not known initially because supplementary examinations such as CT angiography and DSA were not performed when the diagnosis of SAH was established. Consequently, definitive treatment was not provided during the first operation.

There are four main principles of anesthesia for aneurysm clipping. The first is to avoid sudden increases in the aneurysm's transmural pressure (the difference between mean arterial pressure and intracranial pressure) to prevent rupture or re-bleeding. In this patient, sudden increases were prevented using modalities such as a scalp block and dexmedetomidine as an adjuvant drug. The second principle is maintaining cerebral perfusion pressure through euvolemia; it is important to maintain the body's fluid balance to prevent hypovolemia or hyperhydration. Hypovolemia can worsen brain perfusion, while hyperhydration risks causing cerebral edema. In this patient, there was no hemodynamic instability; triple-H therapy (hypervolemia, hypertension, hemodilution) is no longer recommended. Sufficient anesthetic depth and adequate fluids resulted in relatively stable hemodynamics.

The third principle is optimizing surgical access by achieving brain relaxation. This is done through adequate cerebral oxygenation, normal ventilation or temporary hyperventilation, appropriate anesthetic choice, administration of mannitol or diuretics like furosemide, and CSF drainage if necessary. In this patient, $ETCO_2$ was maintained in the 32–35 mmHg range, and mannitol 0.25 mg/kg was administered. The fourth principle is facilitating a rapid recovery from anesthesia to allow for the detection of complications that can be managed promptly. In this case, because the patient's initial GCS was E2M4Vt, the decision was made to allow for a slow emergence in the ICU.[10] The surgical clipping approach in this case could be enhanced by using advanced monitoring technology, such as intraoperative neuromonitoring (IONM) for ICP-CPP and transcranial Doppler (TCD), to ensure cerebral blood flow remains optimal during the procedure. Other research shows that using such monitoring can track the patient's cerebral blood flow in real-time during the clipping procedure. Based on this monitoring, timely interventions can improve the patient's level of consciousness in the early postoperative period and reduce the occurrence of early postoperative neurological deficits.[11]

Hemodynamic stability is a crucial aspect for patients undergoing neurosurgery. An increase in blood pressure during the procedure can lead to bleeding or edema in the surgical area, while a sudden spike in blood pressure during the postoperative recovery phase can risk triggering a hematoma and prolonging the hospital stay.[12] Hypotension, on the other hand, can increase the risk of cerebral ischemia because cerebral blood flow autoregulation is often impaired. Furthermore, the cerebrovascular response can increase intracranial pressure and decrease cerebral perfusion pressure, potentially worsening ischemic damage.[13] Dexmedetomidine, used in this case, can help maintain hemodynamic stability to reduce the risk of complications caused by blood pressure fluctuations.[5] A meta-analysis[13] showed that dexmedetomidine as an adjuvant in intracranial surgery provides better hemodynamic control, lower opioid consumption, and less use of postoperative antiemetics.[13]

A rapid recovery from anesthesia is a priority in neurosurgical patients to allow for immediate postoperative neurological evaluation. In this context, dexmedetomidine offers significant benefits. As a selective α2-adrenergic agonist, dexmedetomidine has a sedative effect that does not depend on the gamma-aminobutyric acid (GABA) system, thereby producing calm sedation without significant respiratory depression. This profile makes dexmedetomidine an ideal choice to support rapid recovery while maintaining optimal conditions for early neurological evaluation in neurosurgical patients.[13,14] Postoperative blood pressure stability is vital to prevent vasospasm, cerebral ischemia, and re-bleeding.[15] In this

patient, the initial postoperative blood pressure was 104/60 mmHg and was relatively stable without episodes of severe hypertension or hypotension. Dexmedetomidine at 0.2 mcg/kg/min was maintained postoperatively for stress control and hemodynamic stabilization until the third day. Hypotension must be watched for, especially if blood pressure becomes too low and threatens cerebral perfusion. Correction can be performed with vasopressors if necessary.

Comorbid pulmonary diseases, such as bronchopneumonia, in the patient can complicate recovery. A cohort study showed that a bundled care approach in brain-injured patients undergoing mechanical ventilation, including those with aneurysmal subarachnoid hemorrhage, has been proven to accelerate readiness for extubation, reduce the duration of mechanical ventilation, and increase the number of ventilator-free and ICU-free days. Some key components of this bundled care include protective lung ventilation with low tidal volumes, the use of moderate PEEP to prevent atelectasis, early enteral nutrition to support metabolic recovery, and standardized antibiotic therapy for nosocomial pneumonia.[16] The patient was maintained on VC-SIMV ventilator mode, then weaned to CPAP before decannulation. A T-piece was used post-decannulation to facilitate weaning from the ventilator.

This patient experienced all of the above problems as a result of prolonged bed rest. Electrolyte and coagulation disorders frequently occur in patients with cerebral injury, compounded in patients with decubitus ulcers from prolonged bed rest. Therefore, hypokalemia must be corrected as was done in this case. Detecting potential electrolyte imbalances should be a focus during the intraoperative phase and can even be initiated postoperatively.[17] Heparinization using heparin 5000 U/24 hours was administered as postoperative thromboembolism prophylaxis. On the 5th day, the patient was transitioned to a T-piece on the tracheostomy, showing progress in respiratory recovery, and on the 6th day, the patient was transferred to the semi-intensive care unit in stable condition, with controlled blood pressure and an improved level of consciousness.

Conclusion

With its sympatholytic effects, dexmedetomidine helps maintain hemodynamic stability, which is a critical aspect in avoiding the risk of aneurysm rupture due to blood pressure fluctuations. Additionally, its sedative effect, which is not accompanied by respiratory depression, allows for effective pain control without disrupting other physiological functions. In the second operation, a TIVA-based approach with adjuvant dexmedetomidine proved effective in maintaining hemodynamic stability throughout the surgery. This underscores the importance of using dexmedetomidine as part of a modern anesthetic strategy in managing intracranial aneurysm cases. Early detection of an intracranial aneurysm directly influences the success of the procedure and the patient's prognosis. The role of the anesthesiologist is crucial for maintaining good coordination with the surgeon during the procedure, maintaining hemodynamic stability, minimizing the stress response to painful stimulation, and enhancing patient safety.

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