

Perioperative Management of Craniotomy for Clipping Aneurysm: A Case Report

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Abstract

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Background:

Ruptured aneurysm is the most common cause of spontaneous subarachnoid hemorrhages. The clinical manifestations of aneurysmal subarachnoid hemorrhage are rapid onset of severe headache, loss of consciousness, nausea and/or vomiting, nuchal rigidity, or photophobia. The definitive treatment of the aneurysm is craniotomy and clipping or endovascular coiling. Several pathophysiological consequences of subarachnoid hemorrhage have been reported in previous studies. The role of the anesthesiologist is to acknowledge these sequelae in order to conduct a thorough pre anesthesia evaluation and to prepare perioperative management. However, case reports that discussed perioperative management of craniotomy for clipping aneurysm are still lacking.

Case Description:

We present a case of a 66-year-old woman diagnosed with acute non-communicating hydrocephalus, intraventricular hemorrhage paraventricle and subarachnoid hemorrhage in basal cistern, right and left sylvian fissure, right and left temporal due to aneurysm rupture post VP. Craniotomy for aneurysm clipping was planned and the surgery was done under general anesthesia. Intraoperatively, the patient's hemodynamic was successfully maintained stable.

Conclusion:

Postoperatively, patient was closely monitored in the intensive care unit and no neurological deficits or other complications were found. This case report aims to further discuss the importance and concerns regarding the perioperative management of craniotomy for clipping aneurysm.

Introduction

Stroke is the first common cause of disability and the third leading cause of death in the world after heart disease and cancer, both in developed and developing countries. According to National Basic Health Research, the prevalence of stroke in Indonesia increased by 56% from 7 per 1000 population in 2013 to 10.9 per 1000 population in 2018.¹ Ruptured aneurysm is the most common cause of nontraumatic or spontaneous subarachnoid hemorrhages. The clinical manifestation of aneurysmal subarachnoid hemorrhage is rapid onset of the worst headache in the patient's life.

Some of the patients may lose consciousness and an unusual headache may be present several weeks prior. The patients also often have nausea and/or vomiting, nuchal rigidity, or photophobia. Patients may be comatose and hypertensive at presentation. Craniotomy and clipping or endovascular intervention involving coils and stents is the definitive treatment of intracranial aneurysm.^{2,3} There are several main goals of perioperative management of craniotomy in aneurysmal subarachnoid hemorrhage. A more detailed explanation on these main goals will be discussed later.

Case Description

A 66-year-old female patient with acute non-communicating hydrocephalus, intraventricular hemorrhage pan-ventricle and subarachnoid hemorrhage in basal cistern, right and left sylvian fissure, right and left temporal due to aneurysm rupture post VP was referred to our hospital complaining severe headache and projectile vomiting for three times two days prior admission. Afterward, the patient experienced decreased consciousness which improved on its own. Complaints of seizures, slurred speech and weakness in half of the body were denied. Physical examination was performed and found high blood pressure and a ruptured brain blood vessel. The patient underwent VP Shunt on December 28th 2023 and after the VP Shunt procedure was done, the patient was unable to communicate. The patient underwent DSA on December 29th 2023 and the result of anterior communicating artery aneurysm was obtained.

On physical examination, the patient's body weight was 60 kg, height was 160 cm, and measured BMI was 23,4 kg/m². Patient was apathetic (E3VaphasiaM6), with respiratory rate of 15 x/min, heart rate of 95 beats/min, blood pressure of 110/60 mmHg and oxygen saturation of 98% on room air. Other physical examination was within normal limits. A complete blood count examination found leukocytosis (20,70 x 10³/μL) and hemoglobin was within normal limit. An electrolyte examination found hyponatremia (131 mmol/L) and hypokalemia (3,10 mmol/L). Other laboratory examinations, including liver and renal function test and urinalysis were within normal limits. Chest x-ray showed a catheter placed from the right neck into the abdomen with non-visualized distal tip suggesting VP shunt. DSA examination was performed and found a small wide neck anterior communicating artery aneurysm with size of 2.3x1.3 mm. CT angiography showed no visibility in A1 segment of right anterior communicating artery and rupture was suspected with intraventricular hemorrhage in right and left lateral ventricle, third and fourth ventricle; subarachnoid

hemorrhage in the sulcus and gyrus of the right and left frontal, temporal, parietal region and right occipital region, anterior inter-hemisphere, basal cistern, right and left sylvian cistern dan sylvian fissure. Aneurysm clipping was planned. We concluded the patient with ASA physical status IV. Surgery was done under general anesthesia.

The patient was not given any pre-medications. Allen test was performed and continued with arterial line insertion and administration of 2% Lidocaine as local anesthetic. Induction was done with target-controlled infusion (TCI) Propofol for Ce target of 3.5-5 mcg/ml. The patient was given Fentanyl 200 mcg (3-4 mcg/kg) intravenously for analgesia and Rocuronium 50 mg (0,6-1,2 mg/kg) intravenously to facilitate intubation. Intubation was performed with a 7.5 endotracheal tube. Anesthesia was maintained using O2 compressed air, TCI Propofol Ce target effect of 3-4 mcg/ml, Fentanyl 0,5 mcg/kg every 45 minutes IV, and Rocuronium 0,15 mg/kg every 30 minutes IV, dexmedetomidine 0.2 – 0.7 mcg/kg/hour.

After the surgery, the patient was admitted to the intensive care unit and mechanically ventilated. The patient was given Fentanyl 500 mcg in 50 mL NaCl 0,9% with rate of 2,1 mL/hour and Paracetamol 1000 mg every 8 hours intravenously for pain control.

During the maintenance phase, the depth of anesthesia was maintained at BIS 40-50. A urinary catheter was placed to monitor urine output. She was then placed in a supine position and blood pressure baseline values at 118/65 mmHg (83 mmHg), pulse rate 63 beats/min without intervention. Before incision, the patient was given mannitol 1 g/kg. The blood pressure change was found 30 minutes later when the surgeon had dissected the multifidus muscle, obtaining an arterial monitoring of 163/81 mmHg (111 mmHg) with no change in pulse rate (64 beats/min). This condition persisted even after craniotomy of the suboccipital bone and opening of the dura via Y-incision (figure 2) with a duration of 30 minutes after the onset of hypertension. We concluded that she had intraoperative neurogenic hypertension after confidently

excluding other causes (hypercarbia, desaturation, pain, light anesthesia, and drug interactions). We decided to give her intravenous nicardipine titrated at a dose of 1-3 mcg/kg/min with a target mean arterial pressure return to baseline of 80-90 mmHg. Intraoperative arrhythmia was not found.

There was another blood pressure fluctuation during the Valsava maneuver for the evaluation of cerebrospinal fluid leakage after the duraplasty process. However, it was maintained stable until the neurosurgeon completed suboccipital decompression and cranioplasty with an upside down-inside out approach to the suboccipital bone. The need for antihypertensive agents was significantly reduced and discontinued before extubation (pre-discontinuation dose 0.4 mcg/kg/min). The surgery lasted 3 hours and 30 minutes.

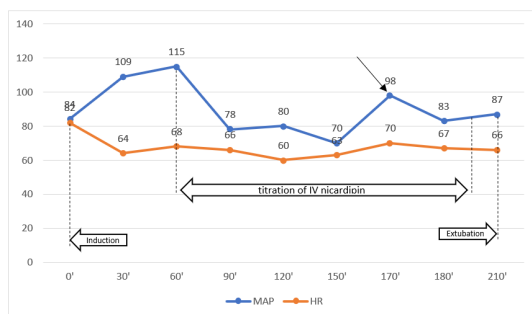


Figure 1. Intraoperative hemodynamic fluctuation: the blue graph represents mean arterial pressure; the red graph represents heart rate. Asterisk (*) represents time and hemodynamics during suboccipital craniotomy. Arrow (→) represents time and hemodynamics during breath-holding (Valsava) maneuver.

We gave 1700 mL ringer fundin intraoperatively with total bleeding of 350 mL and urine production of 500 mL. Rocuronium was stopped 1 hour before the end of the surgery. We reversed the paralyzed muscle with sugammadex. Fentanyl 25 mcg was given 5 minutes before extubation and dexmedetomidine infusion was maintained at 0.3 mcg/kg/min to prevent coughing. After the anesthetic drugs were stopped, we extubated her when she was easily arousable in the operating room.

She was observed in the recovery room with an Aldrete score of 9/10. Blood

pressure was 115/74 (90) mmHg with a pulse rate of 72 beats/minute, axillary temperature was 35.9°C. Urine output was 0.7 mL/kg/hour. She was then admitted to the intensive care unit for monitoring. Neurological examination showed the patient remained with aphasia without other neurological deficits. Recurrent headache symptoms were absent and there were no episodes of hemodynamic instability. The patient was discharged from ICU on postoperative day 3. We continued to follow up on her condition after 1 month postoperatively. She had no complaints and showed resolution of headache and ataxia.

Discussion

Various studies have reported that eighty percent of spontaneous SAH are caused by aneurysm rupture. There are several risk factors that contribute the formation and rupture of intracranial aneurysms and one of them is poorly controlled hypertension. Hypertension causes increased transmural pressure gradient in the aneurysm wall and changes the thickness of the vessel wall.⁴ SAH predominantly occurred at the age of 50-60 years old and the incidence was found 1.6 times higher in females than males.⁵ Middle cerebral artery (MCA) and internal carotid artery (ICA) are the arteries that most often form aneurysms, whereas aneurysm in the anterior and posterior communicating arteries have a high risk of rupture.⁶ Craniotomy and clipping or endovascular coiling are the definitive treatment of intracranial aneurysm. A previous study compared the outcome of craniotomy and endovascular intervention and found that craniotomy has poorer outcome due to the surgery process itself rather than rebleeding.⁷ In this case, a 66-year-old woman was referred to our institution complaining severe headache and projectile vomiting for three times two days prior admission. The patient also experienced decreased consciousness which improved on its own. The patient has no history of diagnosed hypertension, but high blood pressure was found on physical examination. DSA examination was performed and found a small wide neck anterior communicating artery aneurysm

with size of 2.3x1.3 mm. Therefore, craniotomy for aneurysm clipping with general anesthesia was planned.

Pre-operative evaluation should be more focused on pathophysiological sequelae of intracranial aneurysm that needs to be anticipated. Neurologic complication including increased intracranial pressure, decreased cerebral blood flow, cerebral autoregulation disturbance, and delayed cerebral ischemia.⁸ Ruptured aneurysm, inflammation, and direct irritation of brainstem stimulate the sympathetic system which leads to neurogenic pulmonary edema. Sympathetic hyperactivity also leads to myocardial dysfunction. Cardiac arrhythmia and electrocardiography changes such as sinus bradycardia, sinus tachycardia, ST segment depression, T-wave inversion, U-waves, and prolonged QT interval are commonly found.⁹ Intracranial aneurysm is also associated with fluid and electrolyte imbalance such as hypokalemia, hyperglycemia that may require insulin administration, and hyponatremia caused by cerebral salt wasting due to raised secretion of brain natriuretic peptide followed by suppressed aldosterone synthesis.¹⁰ Prompt correction of these fluid, glucose and electrolyte imbalance need to be done by the anesthesiologist prior surgery. Monitoring electrolytes and osmolality of blood and urine serially are also required when mannitol or hypertonic saline is administered. In this case, the electrolyte examination showed hyponatremia (131 mmol/L) and hypokalemia (3,10 mmol/L). The patient was not given any mannitol or hypertonic saline by the neurosurgeon. One of the clinical grading that can be used in pre-operative evaluation is Hunt and Hess grading system. Patients with worse neurologic status are associated with increased intracranial pressure, intraoperative brain edema, cerebral autoregulation disturbance, and impaired cerebrovascular reactivity to carbon dioxide.¹⁰

The main goal for induction of anesthesia is preventing hypertension induced by laryngoscope and tracheal intubation. A study has reported that 8 out of 404 patients

had ruptured aneurysm during anesthetic induction or induced by coughing and bucking during tracheal intubation.[12] Airway manipulation stimulates the sympathetic system which results in hypertension. Hypertension could cause rebleeding and increased aneurysmal transmural pressure which leads to sudden rupture. Therefore, blood pressure should be monitored after induction of anesthesia, before laryngoscopy and immediately after tracheal intubation. In this case, the patient's blood pressure after induction of anesthesia is 130/63 mmHg, before laryngoscopy is 103/55 mmHg and after tracheal intubation is 110/58 mmHg.

The anesthesiologist should also be aware of the signs of possible re-bleeding, such as persistent hypertension, bradycardia and sudden anisocoria. Arterial line placement can also cause pain and anxiety which leads to hypertension. Preinduction arterial line placement for monitoring blood pressure is not necessary unless the patient has cardiac dysfunction including increased troponin and hemodynamic instability.¹³ In this case, arterial line placement was done before the induction of anesthesia. Allen test was performed and continued with arterial line insertion and administration of 2% Lidocaine as local anesthetic. The patient's blood pressure was within normal limits (122/61 mmHg) after the arterial line placement. Bag-mask ventilation can accidentally cause hypo- or hypercarbia. Hypercarbia causes vasodilatation resulting increased intracranial pressure and disturbing cerebral perfusion whereas hypocarbia caused by hyperventilation results in reduced intracranial pressure which leads to increased aneurysmal transmural pressure.

The goal for hemodynamic is decreasing of blood pressure by 20% from baseline or systolic blood pressure less than 160 mmHg. However, aggressive treatment in lowering blood pressure could cause secondary ischemia.¹⁴ In this case, the patient's systolic blood pressure was maintained by 120-140 mmHg. Several actions to prevent hemodynamic responses are needed, such as increasing the

anesthetic depth; analgesics agents, such as boluses of fentanyl or remifentanyl and short-acting antihypertensive agents, such as esmolol and nicardipine.[13] In this case, the patient was given [hypertensive drugs] to maintain target blood pressure and Fentanyl 150 mcg (2-3 mcg/kg of body weight) intravenously for sedation. The placement of a temporary clip before the placement of the permanent clip may decreased the brain oxygenation. Therefore, raising the blood pressure by 10-20% from preinduction baseline for a short period of time is needed to increase blood flow to the region at risk of ischemia. The blood pressure can be normalized after the procedure is done.

Herniation and rapid increased in intracranial pressure could results in dilated pupil, arrhythmias and ischemia. The surgeon may also notice increased blood flow at the incision site or sudden bulging of the brain.¹⁶ Reduction of intracranial pressure and neuroprotection are the main goals before the opening of dura mater. The use of propofol bolus, continuous infusion or bolus of short acting opioids, or thiopentone sodium is useful to maintain intracranial pressure, cerebral perfusion pressure, and mean arterial pressure.¹⁷ Intravenous anesthetics is also useful in reducing cerebral metabolic rate. In this case, anesthesia was maintained by administering TCI Propofol Ce target effect of 2-3 mcg/kg/min, Fentanyl 0,25 mcg/kg every 45 – 60 minutes IV, and Rocuronium 0,15 mg/kg every 45 – 60 minutes IV. Other strategies like short period of moderate hyperventilation and hypothermia may also reduce intracranial pressure. Several studies found that mild hypothermia (32–35°) showed benefit in decreasing intracranial pressure in good grade patients.¹⁸

Hypertension may also occur during extubation due to stimulation of sympathetic nerve.[14] In this case, the patient's blood pressure after extubation was 125/68 mmHg. Clinical manifestations of intra operative aneurysmal rupture may be shown post operatively, such as delayed return of consciousness, rapid onset of neurological deterioration, hemodynamic

instability, focal neurological deficits, or seizures.¹⁹ In this case, after the surgery the patient was admitted to the intensive care unit and mechanically ventilated. Neurological examination showed the patient remained with aphasia without other neurological deficits. Recurrent headache symptoms were absent and there were no episodes of hemodynamic instability. The patient was discharged from hospital on postoperative day 5. We continued to follow up on the patient's condition after 1 month postoperatively. The patient had no complaints and showed resolution of headache and ataxia.

Conclusion

Hemorrhagic stroke caused by ruptured intracranial aneurysm is a life-threatening condition that requires prompt definitive treatment, either by craniotomy and aneurysmal clipping or endovascular coiling. The anesthesiologists need to acknowledge the sequelae of subarachnoid hemorrhage to conduct pre anesthesia evaluation thoroughly and to prepare perioperative management. The perioperative management of craniotomy for clipping aneurysm include rebleeding prevention, maintaining systolic blood pressure according to the recent recommendation, neurophysiological monitoring, maintaining intracranial pressure and neuroprotection, and prevention of postoperative pain and complications.

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