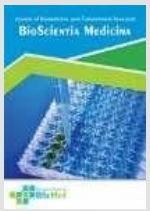
eISSN (Online): 2598-0580



Bioscientia Medicina: Journal of Biomedicine & Translational Research

Journal Homepage: www.bioscmed.com

Intensive Management of Spontaneous Basal Ganglia Hemorrhage: A Case Report

Liliriawati Ananta Kahar[1*], Rico Ikhsani[2], Wiwi Monika Sari[3]

[1]Department of Anesthesiology and Intensive Care, Faculty of Medicine, Universitas Andalas, Padang, Indonesia

[2]Department of General Surgery, Faculty of Medicine, Universitas Andalas, Padang, Indonesia

[3]Department of Intensive Care, Dr. M. Djamil General Hospital, Padang, Indonesia

ARTICLE INFO

Keywords:
Basal ganglia
Hemorrhage
Intensive management

***Corresponding author:**
Liliriawati Ananta Kahar

E-mail address:
lili_ananta@ymail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/bsm.v7i9.862

A B S T R A C T

Background: Simultaneous non-traumatic bilateral basal ganglia haemorrhage is an infrequent entity. ICH accounts for 10 to 20 % of all cerebrovascular events in the US, with 30-day mortality up to 40%. The predisposing factors and pathophysiological processes leading to simultaneous bilateral basal ganglia development are not well known. **Case presentation:** A 43-year-old female patient was diagnosed with spontaneous intracerebral haemorrhage in the left basal ganglia and intraventricular haemorrhage. The patient underwent an urgent ICH evacuation craniotomy. After the procedure, the patient was treated in the intensive care unit. The patient was intubated for 4 days. Then, on the fifth day of stay in the ICU, a tracheostomy was performed on the patient. **Conclusion:** Spontaneous ICH has high mortality and morbidity. ICH management is mainly on timely targeted blood pressure management, effective and rapid reversal of coagulopathy to prevent hematoma expansion, and uncompromising intracranial pressure management.

1. Introduction

Simultaneous non-traumatic bilateral basal ganglia haemorrhage is rare. Intracerebral haemorrhage accounts for 10 to 20% of all cerebrovascular events in the United States, with a 30-day mortality rate of up to 40% and severe disability in most surviving patients. Spontaneous intracranial haemorrhage is the most frequent type of stroke case and has a high mortality rate.[1,2]

The incidence of intracerebral haemorrhage (ICH) is more common in Asians, males, and the elderly, and morbidity and mortality are increasing, especially in low- and middle-income countries. Risk factors that increase the incidence of ICH include advanced age, male, chronic kidney disease, cerebral microhemorrhage, and cerebral amyloid angiopathy. The clinical picture of ICH varies according to the location and size of the hematoma, as well as how extensive the haemorrhage is and the extension to the intraventricular. Simultaneous bilateral non-traumatic basal ganglia haemorrhage of the intracerebral haemorrhage type is extremely rare. The incidence of ICH is mainly due to uncontrolled hypertension.[3,4]

One of the leading causes of basal ganglia haemorrhage is hypertension, which is not treated correctly, especially in developing countries. Hypertension causes damage to the vascular structure and leads to the formation of microaneurysms. Bleeding in the basal ganglia is usually caused by the rupture of these microaneurysms, namely at the

location of the lenticulostriate artery and thalamic-perforated artery simultaneously.[4,5]

Patients who are often admitted to the intensive care unit are those with neurosurgical conditions. Intensive care in postoperative neurosurgical patients generally focuses on maintaining adequate brain tissue perfusion by maintaining excellent and appropriate brain perfusion pressure to ensure the brain is well-oxygenated.[6]

2. Case Presentation

A 43-year-old female patient complained of decreased consciousness for 18 hours before entering the hospital. The patient was initially lying in bed and suddenly found the family unconscious. The patient was convulsing for 3 minutes. The family previously denied headaches. There were complaints of vomiting 1 time. A family history of hypertension and mellitus was denied.

Physical examination found the general condition of severe illness, GCS E2M3V2, blood pressure 175/110, pulse 125x per minute, respiratory rate 22x per minute. pupil isochor 3mm/3mm, light reflex +/+, extremity found lateralization to the right. Laboratory examination revealed leukocytosis. Blood gas analysis obtained pH 7.39, PO_2 106, PCO_2 28, SO_2 98,2, HCO_3 17.9, base excess -7,2, PO_2/FiO_2 265. A brain CT scan examination showed spontaneous intracerebral haemorrhage in the left basal ganglia and intraventricular haemorrhage. The patient underwent an urgent ICH evacuation craniotomy.

After the procedure, the patient was treated in the intensive care unit. The patient was intubated and assisted by a mechanical ventilator. During the ICU stay, the patient was given light sedation for the first 24 hours, after which the sedation was slowly decreased and then stopped to assess the patient's consciousness. After the fifth day in ICU, the patient showed no improvement in consciousness; the cough reflex was not good; then, it was decided to perform an early tracheostomy on the patient.

During the ICU stay, we maintained and closely monitored hemodynamics, especially blood pressure. Systolic blood pressure during ICU was controlled with antihypertension such as nicardipine drip with a target systolic blood pressure below 140 mmHg. Labour examinations were also routinely done. Blood sugar was also examined periodically.

The patient also presented with sepsis with a leukocyte count 20,600 and procalcitonin of 86,49. The patient also began to fever and was given antipyretics routinely. The patient's temperature ranged from 37.5 - 38.5°C, the patient also used a cooling blanket to reduce the patient's body temperature, but it was still challenging to maintain in the range of 36,5 - 37,5°C.

On the fifth day of stay in the ICU, a tracheostomy was performed on the patient. The therapy given to patients was cefepime 3x1 g, levofloxacin 1x750 mg, tranexamic acid 3x1 g, Vit K 3x10 mg, Paracetamol 3x1 g, Mannitol 3x150 cc. From the labour results, haemoglobin was 7.6 g/dl and then the patient was given a packed red cell transfusion.

3. Discussion

This case reported a 43-year-old female patient diagnosed with spontaneous intracerebral haemorrhage in the left basal ganglia and intraventricular haemorrhage. Spontaneous intracranial haemorrhage is the most common type of stroke and has a 30-day mortality rate that can reach 52%. Intracerebral haemorrhage (ICH) affects >1 million people yearly, causing high morbidity and mortality rates. The most common risk factors for ICH are uncontrolled hypertension.[2,7]

Based on the cause, spontaneous ICH can be classified as primary or secondary ICH. The incidence of primary ICH is more frequent than secondary, accounting for almost 80% of cases. It is mainly caused by spontaneous rupture of small blood vessels due to uncontrolled hypertension and amyloid angiopathy. Based on its location, primary ICH is also classified into lobar or non-lobar and supratentorial or infratentorial ICH. Non-lobar ICH is the most common type of ICH that results from prolonged high blood pressure resulting in lipo hyalinosis of small

perforating arteries in the basal ganglia, thalamus, pons and cerebellum, usually causing deep bleeding and often extending to the ventricles.[7]

Management of spontaneous ICH consists of a choice of surgery or medication, depending on the patient's age, neurological status at the onset of bleeding and progression of neurological status, location of ICH, the extent and volume of hematoma, presence of ventricular system haemorrhage and presence of comorbidities.[8]

As the most common sites of spontaneous ICH are deep brain structures, such as the basal ganglia and thalamus, enormous layers of brain tissue must be passed through during surgery, possibly causing damage to healthy brain tissue. In addition, neurosurgical procedures are not free from possible risks of action and side effects. Post-surgical complications such as bleeding and infection are likely to occur, leading to increased mortality and morbidity in ICH patients.[9]

In the Intensive Care Unit focuses on treating the disease and early identification and prevention of complications that often occur during patient care in the ICU. Medical problems are usually related to immobility, swallowing disorders, infection, immune response and hemodynamic stability, neurological disorders and deficits that occur and changes in consciousness, some of which are problems that doctors and neuroscience nurses must be able to deal with during hospitalization.[10]

Nerve dysfunction is one of the problems that must require a mechanical ventilator. The goal of using a ventilator in a neurosurgical patient is to maintain a patent airway in a patient with a low level of consciousness. Patients with low consciousness have a risk of aspiration, which can cause hypoxemia and hypercapnia. Neurologic patients on ventilators usually find it difficult to reduce their breathing assistance, so they require a more extended ICU stay and have a higher tracheotomy rate. Traditionally, PEEP levels (≤5 cmH_2O) were kept low to avoid increased intracranial pressure (ICP), but recent studies have shown that it is safe to use higher PEEP levels because these higher levels improve brain oxygenation. On the other hand, it is not recommended to maintain prophylactic hyperventilation for long periods to reduce pCO_2. However, it may temporarily reduced an intracranial hypertension (ICH).[6,10]

The goal of management of mechanical ventilation in neurosurgical patients is to prevent secondary cerebral ischemia and improve neurological outcomes. The hyperoxia and hypocapnia that may occur as complications after ICH surgery will increase mortality and poor neurological outcome.[11]

The provision of deep sedation has been shown to increase the length of stay in the ICU and the need for mechanical ventilators. Clinical guidelines on managing sedation in the ICU recommend, if there are no contraindications, use of a light sedation strategy and prioritize the use of analgesia and non-benzodiazepine drugs In post-operative neurosurgery patients, it is essential to carry out frequent and adequate neurological examinations, so in this case, it is useful to has a light sedation to open the neurological window so that optimal neurological monitoring can be optimal.[6]

One of the problems that can occur after neurosurgical surgery is seizures. The postoperative stress response and surgical injury may cause seizures. Still, if seizures occur late in the postoperative period, they may have epilepsy and require long-term prophylaxis.[12]

Postoperatively, electrolyte abnormalities may also occur, and the most common are hypernatremia and hyponatremia. The two leading causes of hyponatremia are inappropriate antidiuretic hormone secretion syndrome and brain salt depletion syndrome, which must be differentiated, as each case has specific and different management.[12]

Postoperative infection is also a common occurrence. The risk factors for infection, especially in postoperative neurosurgery patients, include pre-existing infections, altered sensorium and consciousness, multiple operations and emergency operations, long duration of surgery (>4 hours),

cerebrospinal fluid (CSF) leakage, and long mechanical ventilator.[12]

Systolic blood pressure during ICU was controlled with antihypertension, such as nicardipine drip with a target systolic blood pressure below 140 mmHg. Blood pressure should be intensively monitored and controlled in all post-neurosurgery patients. High blood pressure occurs in intracranial haemorrhage due to disruption of cerebral autoregulation and disruption of autonomic nervous system activation. Persistently high blood pressure will lead to recurrent and uncontrollable bleeding. If hypertension cannot be adequately managed, it will increase patient morbidity and mortality.[12,13]

Sustained elevation of systolic blood pressure will also expand the hematoma, leading to neurological deterioration. Therapeutic options to lower blood pressure in postoperative neurosurgical patients include intravenous calcium channel blockers (e.g., nicardipine) and β-blockers (e.g., labetalol). These drugs are preferred due to their short half-life and ease of titration. Oral antihypertensive agents should be started as soon as possible to control persistent hypertension and may also facilitate the transition of care from ICU to long-term management. Arterial line insertion is recommended to optimize blood pressure monitoring.[7]

American Heart Association guidelines recommend that "In patients with spontaneous ICH of mild to moderate severity presenting with SBP between 150 and 220 mmHg, acute lowering of SBP to a target of 140 mmHg to maintain in the range of 130 to 150 mmHg is safe and may be reasonable to improve functional outcomes".[10]

Controlling blood pressure to stay within the <140/90 range will improve functional outcomes without reducing death or severe disability. However, lowering systolic blood pressure to a more aggressive target of <120 mmHg is also not recommended, especially during acute conditions, as it can lead to other organ compromise, such as cardiac and renal insufficiency.[14]

The patient also began to fever and was given antipyretics routinely. The patient's temperature ranged from 37.5 - 38.5°C. Fever is also common in intracranial haemorrhage, especially those with intraventricular extension. Studies show that sustained fever will result in a worse prognosis. In a case-control study of patients with spontaneous ICH who had two consecutive fevers ≥38.3°C despite antipyretics, although the patients eventually developed normothermia, it was still shown to be associated with poor outcome, increased duration of mechanical ventilator use and more prolonged sedation, and also increased length of stay in the intensive care unit.[7]

Oxygen saturation is also crucial in the monitoring of all neurosurgical patients. End-tidal carbon dioxide ($EtCO_2$) values should be measured in patients on mechanical ventilators. In addition, the partial pressure CO_2 level ($PaCO_2$) should be checked periodically, especially during neurological damage due to hypercapnia or hypocapnia. The expected $PaCO_2$ level is around 30 - 35 mmHg.[12]

In this patient blood sugar was also examined an controlled periodically. Either hyperglycemia or hypoglycemia is also associated with poor outcomes in neurological neurosurgical postoperative patients. Expected glucose levels are in the range of 140-180 mg/dL.[12]

Hyperglycemia is associated with a poor prognosis in patients with intracranial haemorrhage, but low glucose values are also associated with expanding hematomas. Careful and controlled glucose control can improve neurological outcomes. The American Heart Association guidelines also advise "to avoid hyperglycemia and hypoglycemia although no specific target blood glucose level is provided".[1,8]

Monitoring intracranial pressure is critical in managing patients with brain injuries. Increased intracranial pressure will also increase the mortality rate in neurosurgery patients. The average intracranial pressure level is between 5 and 15 mmHg. Usually, a level of >20---25 mm Hg is considered an indication for intensive intracranial monitoring.[6]

Simple tests such as monitoring and evaluation of consciousness or GCS, motor and sensory reflex examination and pupil examination can be performed to assess and detect any neurological deficits or disorders that may have recently occurred.[12]

4. Conclusion

Spontaneous ICH has high mortality and morbidity. ICH management is mainly on timely targeted blood pressure management, effective and rapid reversal of coagulopathy to prevent hematoma expansion and uncompromising intracranial pressure management.

5. References

1. Morotti A, Goldstein JN. Diagnosis and management of acute intracerebral hemorrhage. Emerg Med Clin North Am. 2016; 34(4): 883–99.
2. Dekker SE, Hoffer SA, Selman W, Bambakidis NC. Principles of neurological surgery (4th ed) 22 - Spontaneous Intracerebral Hemorrhage. 2018; (3): 22–3.
3. An SJ, Kim TJ, Yoon BW. Epidemiology, risk factors, and clinical features of intracerebral hemorrhage: An update. J Stroke. 2017; 19(1): 3–10.
4. Sheikh Hassan M, Mohamed Ali A, Farah Osman M, Ahmed A. Spontaneous bilateral basal Ganglia hemorrhage due to severe hypertension. Vasc Health Risk Manag. 2022; 473–7.
5. Kayastha J, Rajbhandari P, Gurung P, Shrestha B, Dabadi S, Pant B. Unusual case of spontaneous bilateral basal ganglia bleed. Clin Case Reports. 2022; 10(2): e05437.
6. Colomina MS, Abelló FA, Corral AS, Roca RF. Optimization of the neurosurgical patient in Intensive. 2019; 43(8).
7. Dastur CK, Yu W. Current management of spontaneous intracerebral haemorrhage. Stroke Vasc Neurol. 2017; 21–9.
8. Luzzi S, Elia A, Maestro MD, Morotti A, Elbabaa SK, Cavallini A, et al. Indication, Timing, and surgical treatment of spontaneous intracerebral hemorrhage: Systematic review and proposal of a management algorithm. World Neurosurg. 2019.
9. de Oliveira Manoel AL. Surgery for spontaneous intracerebral hemorrhage. Crit Care. 2020; 24(1): 45.
10. HemphillIII JC, Greenberg SM, Cushman M, Fung GL, Mitchell PH, Scott PA. Guidelines for the management of spontaneous intracerebral hemorrhage - A guideline for healthcare professionals from the American Heart Association/American Stroke Association. AHA Journal. 2015; 2032–60.
11. Badenes R, Bilotta F. Neurocritical care for intracranial haemorrhage : A systematic review of recent studies. BJA. 2015; 115(Suppl_2): II68–74.
12. Chauhan R, Bloria SD, Luthra A. Management of postoperative neurosurgical patients. Indian Journal of Neurosurgery. 2019; 8: 179–84.
13. Parry-jones AR, Moullaali TJ, Ziai WC. Treatment of intracerebral hemorrhage : From specific interventions to bundles of care. Int J Stroke. 2020; 15(9): 945–53.
14. Al-mufti F, Walz W. Cerebrovascular disorders Series Editor.