CASE REPORT

Bali Journal of Anesthesiology (*BJOA*) 2017, Volume 1, Number 3: 48-50
E-ISSN: 2549-2276

Published by DiscoverSys

Spontaneous rupture of arteriovenous malformation: ICU based brain resuscitation

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ABSTRACT

Arteriovenous malformations (AVM) is a relatively rare intracranial abnormality. Generally, it caused by congenital abnormalities that recognized after the bleeding started. Spontaneous intracranial bleeding after AVM rupture is an emergency condition and require immediate treatment to reduce mortality rate. After stabilization of intracranial bleeding due to AVM rupture, secondary injury may occur hours or even days after the inciting traumatic event. The injury may result from impairment or local declines in cerebral blood flow (CBF) after brain injury. The decrease in CBF is the result of local edema, hemorrhage, or increased intracranial pressure (ICP). An adequate brain resuscitation is needed to decrease brain edema and intracranial pressure by achieving several targets and avoid things that can interfere with CBF. A recovery phase should be given to the patient with rupture of AVM before going to definitive therapy.

Keywords: *Arteriovenous malformation, secondary injury, brain resuscitation*
Cite This Article: Panji, P.A.S., Aryabiantara, I.W., Suranadi, I.W., Parami, P., Kurniajaya, I.G.A.M.W. 2017. Spontaneous rupture of arteriovenous malformation: ICU based brain resuscitation. *Bali Journal of Anesthesiology* 1(3): 48-50. DOI:10.15562/bjoa.v1i3.25

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INTRODUCTION

Arteriovenous malformations (AVMs) is an abnormal tangle of blood vessels in the brain or spine and the most common intracranial vascular malformation, with an estimated occurrence of 1:5000-1:2000 persons.[1-3] Some AVMs have no specific symptom and low risk to one life, while others cause severe and devastating effects when ruptures occur. The rupture rate of AVM is between 2-4% per year.[4,5] After the initial hemorrhage, the risk of recurrent hemorrhage is 6% in the first year before it returns to baseline in the following years. The risk of serious morbidity or mortality after hemorrhage is 30-35%, and the mortality rate after the first, second, and third hemorrhagic events are 10, 15, and 20% respectively.[6]

Patients with bleeding from the AVM should be stabilized in accordance with acute management for intracranial hemorrhage (ICH) guidelines. The resting phase for 2 to 6 weeks after the bleeding is recommended prior to definitive therapy to avoid hematoma.[7] An adequate brain resuscitation is needed to avoid secondary brain injury.

We will report a case of a 35 years old man with spontaneous intracranial bleeding suspected ruptured cerebral AVM, treated by clot evacuation and decompression by craniotomy, followed by brain resuscitation in intensive care unit (ICU).

CASE REPORT

A 35-year-old man was admitted to hospital with a sudden severe headache associated with nausea or vomiting. There is no history of hypertension and the use of anticoagulant drugs. Physical exam finding was marked for left hemiparesis. Non-contrasted head CT scan revealed an ICH in the right parietal, volume 56.6 cm^3, urging the right ventricle causing midline shift to the left as far as 0.4 cm, and the presence of brain edema.

The patient was scheduled for craniotomy for decompression and clot evacuation under general anesthesia. At the time of surgery, about 50 ml of the intracerebral clot was evacuated. After the procedure, he was transferred to the ICU with mechanical ventilation. Brain resuscitation was conducted in ICU to minimize cerebral edema, intracranial pressure (ICP), and to optimize cerebral perfusion pressure (CPP).

The head was elevated by 30 degrees and body temperature was maintained to normal. A mechanical ventilator is used to control PaCO2 from 35 to 40 mmHg, with initial setting pressure inspiration 16 cmH2O, FiO2 60%, PEEP 4, with RR 14 times per minute. Blood gas analysis results pH 7.42, PaCO2 35.8 mmHg, PaO2 173 mmHg, BEecf 1.0 mmol/L and HCO3- 23,0 mmol/L. We also maintain blood pressure with mean arterial pressure (MAP) more than 80 mmHg and systolic pressure about 90 mmHg to make sure cerebral perfusion pressure (CPP) about 50 mmHg to keep cerebral autoregulation intact.

Random blood sugar also checked regularly to avoid hyperglycemia (>180 mg/dL). Sedation and analgetics were given to avoid any agitation. In this patient, we gave fentanyl 0.25 mcg/kgBW/h

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for analgetic and midazolam with Richmond Agitation-Sedation Score (RASS) target -3. We also gave the patient phenytoin 100 mg intravenous every 8 hours for seizure prophylaxis and mannitol was titrated down to decrease brain edema. The patient was extubated at day two after surgery. He recovered well with no neurologic deficit and was discharged home on day six post-surgery. Elective angiography was scheduled later.

DISCUSSION

The prevalence of cerebral AVMs is estimated at 0.01-0.5%. AVM is generally present in patients aged between 20 and 40 years and is more common in those over 30 years.[4,6] Our patient is a 35 years old man with Secondary Intracerebral Hemorrhage Score more than 2, has a high probability of underlying vascular etiology suspected rupture of AVM.[8] Patients with bleeding from the AVM should be stabilized in accordance with ICH guidelines for the acute management. The resting phase for 2 to 6 weeks after hemorrhage is recommended prior to definitive therapy to avoid disturbance of brain parenchymal and intracerebral hematoma.[7]

An adequate brain resuscitation is needed to minimize cerebral edema and intracranial pressure (ICP) while simultaneously optimizing cerebral perfusion pressure (CPP) and tissue oxygenation to reduce secondary ischemic injury.[9] All patients with brain injury should have their head elevated 30 degrees to reduce cerebral edema and augment venous drainage from the cranial vault.[9,10]

Normal body temperature should be maintained. In 2001, Clifton et al. reported the results of a large, prospective, randomized trial evaluating the use of hypothermia in brain injury patients. In that study, 392 patients were randomized within 6 hours of injury to hypothermia (33 °C) vs. normothermia, and then rewarmed after 48 hours. Mortality was 28% in the hypothermia group and 27% in the normothermia group (p=0.79). The patients in the hypothermia group demonstrated a great incidence of pneumonia as well as longer hospital length of stay than patients in the normothermia group. The authors concluded that treatment with hypothermia, with a body temperature reaching 33°C is not effective in improving outcomes in patients with severe brain injury.[9]

Hypercapnia is a potent cerebral vasodilator and should be avoided in patients with cerebral edema and elevated ICP. Hyperventilation reduces ICP by causing cerebral vasoconstriction and reducing cerebral blood flow. A PaCO2 target of 35-40 mmHg is appropriate in the initial resuscitation of the patient with severe traumatic brain injury. In patients with refractory elevations in ICP, a revised target of 30-34 mmHg may be appropriate.[9]

Hyperglycemia has been associated with increased brain lactate resulting in local brain tissue acidosis. Brain tissue acidosis worsens mitochondrial function in the penumbra (ischemic brain tissue surrounding the core injury), and increase the size of cerebral infarction.

Systolic hypotension leads to cerebral ischemia and secondary brain injury. Mean arterial pressure (MAP) should be maintained above 80 mmHg through the use of judicious isotonic intravenous fluids (without dextrose) until an ICP monitor is available. The goal should be to ensure an adequate CPP >60 mmHg at all times. As the brain is very sensitive to anoxia, this will serve to improve oxygen delivery and further avoid secondary brain injury. If the patient's MAP cannot be maintained above 80 mmHg with intravenous fluid alone (or CPP > 60 mmHg if ICP monitoring is available), low-dose norepinephrine should be initiated.[9-11]

Intravenous sedation and analgesia are first line therapy for treating intracranial hypertension. Patients with severe brain injury are often agitated and require sedation regardless of their ICP. Because of the brain injury, it may be impossible to determine whether the cause of agitation is pain or another reason. Analgesia is best obtained using continuous narcotic infusions. Fentanyl has minimal hemodynamic effects and a short half-life (10-20 minutes) allowing rapid titration on and off for neurologic examination. Propofol is a sedative-hypnotic agent that has a favorable pharmacokinetic profile and beneficial effects on both cerebral metabolic rate and ICP following brain injury. It has a short half-life (9 minutes) making it easy to titrate on and off for neurologic examination.[9]

Several studies have shown that brain injury patients with no history of seizure disorder or witnessed post-traumatic seizure activity are still at increased risk of developing post-traumatic seizures if they have one or more of the following risk factors: GCS <10, cortical contusion, depressed skull fracture, epidural hematoma, subdural hematoma, intracerebral hematoma, penetrating head wound, or seizure within 24 hours of injury. In these patients, seizure prophylaxis reduces the risk of seizures in the early period (up to 7 days after injury) but does not alter late seizure occurrence (beyond 7 days).

Hyperosmolar therapy, using either mannitol or hypertonic saline, has been demonstrated to be beneficial in reducing both cerebral edema and elevated ICP. Mannitol has the additional desirable properties of decreasing blood viscosity, increasing free radical scavenging, and inhibiting cellular apoptosis. Mannitol should not be administered in

the absence of ICP monitoring unless the patient is showing signs of transtentorial herniation.[9,10]

SUMMARY

Spontaneous intracranial bleeding after rupture of AVM is an emergency condition and require immediate treatment. Patients with bleeding from the AVM should be stabilized in accordance with ICH guidelines for the acute management. The definitive treatment is started after the stabilization of the ICH. An adequate brain resuscitation is needed in purposes to minimizing cerebral edema and intracranial pressure (ICP) while simultaneously optimizing cerebral perfusion pressure (CPP) and tissue oxygenation to reduce secondary ischemic injury.

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