A CASE STUDY: HYDROCEPHALUS AS A COMPLICATION OF SUBARACHNOID HEMORRHAGE

Putri Maria Grace Bintang, Feda Anisah Makkiyah*

Neurosurgery Department, Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, South Jakarta, Jakarta, Indonesia 12450

*Correspondence: fedaanisah@upnvj.ac.id

ABSTRACT

Acute hydrocephalus develops within 72 hours of subarachnoid hemorrhage (SAH). As many as 25% of patients die from this complication of SAH. Intracranial blood vessel rupture causes blood to accumulate in the subarachnoid, causing cerebrospinal fluid obstruction or hydrocephalus. A 65-year-old woman came with complaints of decreased consciousness since 4 hours before hospital admission, which was preceded by severe headache, neck tension, and vomiting. She had a history of uncontrolled hypertension. She had no history of head trauma or drug usage. Physical examination found her Glasgow Coma Scale score on admission was 12, hypertensive grade I, and neurologic examination revealed nuchal rigidity. CT scans showed SAH in the cisterna basal extending to bilateral fissure sylvii, parietal sinistra region, and bilateral occipital region with cerebral edema and hydrocephalus. Management was ABC control, head position 300, oxygen, mannitol, citicolin, tranexamic acid, glaucon, nimodipine, perdipine, and external ventricular drainage (EVD). After 10 days, an EVD clamp test was performed, found to be independent of the shunt, and EVD removal was performed on day 13. External ventricular drainage is the key to managing SAH with hydrocephalus.

Keywords: Cancer; Stem Cell; Epigenetic

Received: Month Year, *Accepted: Month Year,* *Published: Month Year*

INTRODUCTION

Sudden, severe headaches that one has never experienced before, accompanied by neck pain and even loss of consciousness, should be suspected as a SAH. Subarachnoid is caused by intracranial bleeding into the cerebrospinal fluid-filled area between the arachnoid membrane and the pia meter on the surface of the brain (Adrianto et al., 2016). Each year, approximately one in every 10.000 people suffers from spontaneous SAH. Women are more likely to be affected than men. Although the disease is increasingly common with age, about half of those who experience SAH are under the age of 55. About 80% of nontraumatic SAH is caused by ruptured intracranial aneurysm. Two forms of aneurysms include saccular or berry aneurysms, which range in size from a few millimeters to 20 to 30 mm, and clerotic fusiformateros aneurysms (AK et al., 2020).

Other causes of SAH include arterial malformations, cavernous malformations or angiomas, idiopathic coagulopathy, bacterial endocarditis, venous thrombosis, arterial dissection, tumors, hypertension, and medications. High blood pressure, smoking, and family history are risk factors for SAH (Chen et al., 2017). SAH is a dangerous neurological attack with sudden onset and often without warning. Hydrocephalus is a complication in patients with SAH. As many as 25% of patients die from this complication of SAH. A buildup of cerebrospinal fluid (CSF) in and around the brain and spinal cord is known as bydrocephalus. This happens because it may not be able to drain out after hemorrhage. Hydrocephalus can be seen from

a CT scan and needs to be treated with CSF drainage to prevent obstruction in a fast time.

This study aims to analyze the diagnosis and management of patients with SAH who develop hydrocephalus, providing greater insight into the neurological complication that often occur in SAH patients, hopefully helping in better treatment planning and reduction of the risk of long-term complications.

CASE

A 65-year-old woman was presented to the emergency department with decreased consciousness. Her family said that fifteen days before being taken to Cileungsi Regional General Hospital, the patient complained of headaches with neck tension. The pain was felt continuously since the last week with moderate to severe intensity. The pain interfered with the patient's activity; the patient took stall medicine to help relieve pain. The pain worsens when the patient exerts too much physical activity and subsides when the patient rests. Pain was not accompanied by blurred vision or nausea and vomiting. The patient was examined at a hospital near home; at that time his blood pressure reached 200/130 mmHg. The doctor advised hospitalized patients. After 2 days of hospitalization, the patient asked to go home because of the complaints of headache and neck tension were much reduced. Blood pressure at that time still remained high, 190/110 mmHg. Patient was given captopril 25 mg twice a day.

Now, the patient lost consciousness while working in the field four hours ago, therefore she was taken to the Cileungsi Regional General Hospital. There were denials of spinning dizziness, thick or tingling sensation on one side of the face, swallowing disorders, slurred voice, disorders of defecation and urination, fever, seizures, weakness in the sides of the body. It is also denied that complaints are preceded by trauma, impact to the head, or drug usage. History of uncontrolled hypertension since 2 years ago, previous history like this before, stroke, diabetes mellitus, high cholesterol, smoking habits are denied. Her father had a stroke and hypertension.

The results of the physical examination were blood pressure 159/95 mmHg, pulse rate 104/min, respiratory rate 22/min, temperature 37.2 °C, oxygen saturation 98% on non-rebreathing mask, and Glasgow Coma Scale score on admission was 12. A general physical examination found no abnormalities. Neurological examination revealed meningeal excitatory signs of neck rigidity. The pupils were round isochor 3 mm/3 mm; direct and indirect light reflexes of the right and left eyes were positive. Examination of the cranial nerve cannot be assessed, physiological reflexes are within normal limits, and there are no pathological reflexes. Laboratory tests were performed: hemoglobin 11.2 g/dL, hematocrit 37%, MCV 97 fl, MCHC 30 g/dL, thoracic x-ray, and electrocardiography within normal limits. CT-scan without contract showed subarachnoid hemorrhage in the cisterna basalis, which extended to bilateral fissure sylvii (Figure 1), parietal sinistra region, and bilateral occipital region (Figure 2) accompanied by cerebral edema and hydrocephalus (Figure 3).

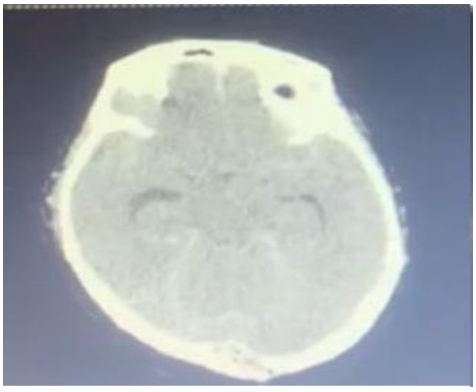

Figure 3. Bilateral fissure sylvii hemorrhage.

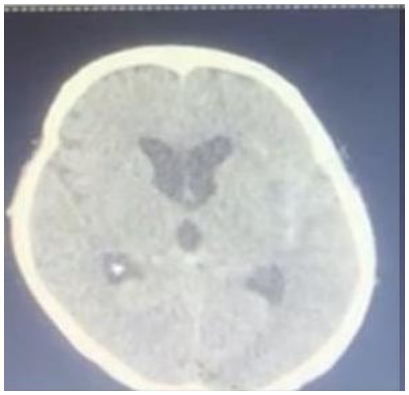

Figure 2. Bilateral parietal sinistra and occipital hemorrhage.

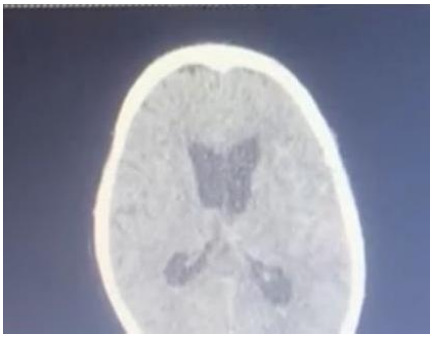

Figure 3. Hydrocephalus.

She was admitted to the intensive care unit. The initial therapy given is patients treated airway, breathing, circulation, head position 30°, NaCl 0.9%, mannitol, citicoline, tranexamic acid, glaucon, omeprazole, nimodipine, folic acid, and external ventricular drainage. External Ventricular Drainage was performed on the second day of treatment with a height of 10 cm from the tragus and a depth of 6 cm. Monitoring of External Ventricular Drainage production for 8 days with the results of clear red liquid drainage volume of 100 cc and day 9 with result clear drainage and volume of 100 cc. On the 10th day and 13th, a 24-hour clamp test was carried out (Figure 4). The result obtained is no fluid that comes out and no headache appears, no vomiting, and papilla edema. This indicates that there was no need for permanent CSF drainage. On day 13, External Ventricular Drainage was removed. The patient was discharged home after 15 days with GCS score of 15 and no focal neurologic deficit.

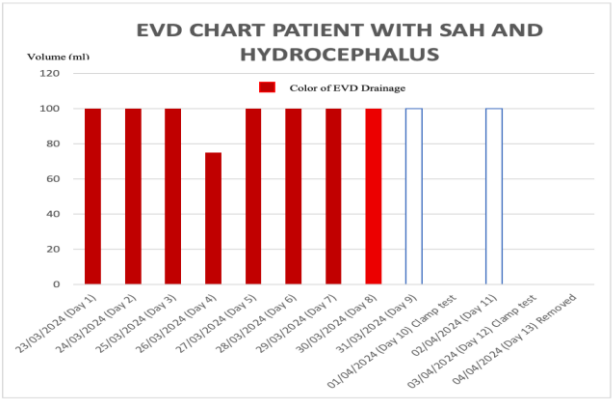

Figure 4. EVD chart patient with SAH and hydrocephalus.

DISCUSSION

The patient has had a history of uncontrolled hypertension since 2 years ago. Diffuse atheromatous degradation of the arterial wall caused by hypertension can result in aneurysms in the cerebral circulation, especially in the internal carotid artery and vertebrobasilar artery. Subarachnoid bleeding occurs when this brain aneurysm ruptures. The most common kind of aneurysm is a saccular one, sometimes referred to as a congenital or berry aneurysm. Berries or saccular aneurysms form at the intersection of blood vessels when the muscular layer is congenitally lacking. Unlike arteries elsewhere, the cerebral arteries only have an elastic layer on the interior lamina, which makes these vessels more vulnerable to the weakening effects of degeneration. The internal elastic membrane dissolves and fragments at the aneurysm formation site. Factors associated with increased blood pressure include strenuous physical activity and moderate physical activity up to 2 hours before the onset of SAH. This incidence can occur about 62% first at the age of 40-70 years, and an estimated 6.5-26.4 out of 100,000 cases of SAH occur each year (Agrawal et al., 2022).

Sudden headaches experienced by patients of moderate to severe intensity, accompanied by vomiting, loss of consciousness, and stiffness, lead to the diagnosis of SAH. Headaches in SAH usually appear suddenly and reach maximum intensity within seconds or minutes, cause a sensation of flashes or thunderclap headaches, and are also described as "the worst headache of my life." Other complaints are vomiting, photophobia, fever that usually occurs in the first 2-3 days, or seizures. More than 50% of cases of SAH are accompanied by decreased consciousness. On physical examination can be obtained stiff horn, preretinal hemorrhage, increased blood pressure, and focal neurological deficits. The scores from Hunt and Hess and the World Federation of Neurological Surgeons (WFNS) were used to determine the severity of SAH. This patient had a Hund and Hess score of degree 2, which is severe headache with signs of meningeal excitability and the possibility of cranial nerve deficits, and a WFNS score of degree 4, which is a motor deficit may or may not be present (AK et al., 2020).

Aneurysm rupture will cause transient global cerebral ischemia and other pathologies called early brain injury, then there is an increase in intracranial pressure that will press on the medulla oblongata so that there is a disturbance of alert neurons resulting in a decrease in consciousness. This increase in intracranial pressure also causes durameter stretching and then stimulates pain-sensitive structures, resulting in severe headaches. The vomiting center in the dorsolateral formation reticular responds to stimuli increased intracranial pressure, then contraction of the duodenum and antrum of the stomach occurs so that intraabdominal pressure increases. This increased pressure causes the stomach to fill, the diaphragm to rise, the intratoraction pressure to increase, the esophageal sphincter to open and vomiting occurs.

Deficits in brain function arise from either blood destroying brain tissue or blood accumulating in the subarachnoid space. Vasospasm can be brought on by blood in the subarachnoid space, especially in the basal cisterna, between the third and seventh days following onset. Sustained vasospasm may result in subsequent cerebral infarction, which would cause more widespread brain tissue injury. However, after installing EVD, no neurological abnormalities were found in the patient again, indicating that vasospasm did not occur during the patient's hospitalization. This is an assessment that the treatment given to the patient is appropriate.

A non-contrast CT scan is the first stage of diagnosis, with a sensitivity of almost 100% in the first 3 days after onset. In this patient, a non-contrast CT scan is performed at 4 hours after onset. CT-scans showed SAH cisterna basal extending to bilateral fissure sylvii, parietal region sinistra, and bilateral occipital region accompanied by cerebral edema and hydrocephalus. Cerebrospinal fluid produced by the choroidal plexus in the third and fourth ventricles flows in the subarachnoid spaces and is reabsorbed into the venous circulation through specialized structures known as arachnoid villi or granulation. Cerebral aneurysms are usually located within the subarachnoid cisterna. Rupture of an aneurysm at this location causes transient communicant hydrocephalus due to blood blockage on absorption in the arachnoid villi (Marcolini and Hine, 2019).

Patients were given NaCl 0.9% for hemodynamic stabilization, head up 30°, and mannitol to lower intracranial pressure, given to lower blood pressure to be at 140-160 mmHg, given tranexamic acid to prevent rebleeding, and perdipin to prevent vasopasm and global cerebral ischemic. Intracranial pressure should also be lowered immediately to prevent complications such as herniation. In these patients, extracranial ventricular drainage is recommended to lower intracranial pressure by drainage of blood in the basal cistern so as to reduce bleeding and hydrocephalus due to SAH. EVD for the management of acute hydrocephalus in patients with high levels of

SAH may have a positive impact on outcomes if performed within 13 hours after the onset of clinical stroke, while the delay of the remaining 72 hours is unjustified (Ohbuchi et al., 2021). In these patients, a drip chamber of EVD was 16 cm above the tragus. The EVD needs to be kept open with a drip chamber 15-20 cm above the tragus (Wulandari et al., 2021).

The amount of drainage of EVD in this patient is blood fluid amounting to 100 mL/day (Figure 4). The results of this drainage are appropriate because the usual amount of drainage is 75 mL every 8 hours (Greenberg, 2020). EVDs are well checked every 2-4 hours, and serial ICP measurements, neurological examinations, or CSF outputs (for systems open for drainage). Evaluate EVD installation by checking for the presence of good waveforms with variations in breathing and transmitted pulse pressure and checking for patency (Brooks et al., 2020). However, it is not done due to limited equipment in the hospital, so what is observed in only clinical and CSF drops from EVD, which are as much as 2-3 drops of CSF.

In these patients after the EVD clamp test. This EVD clamp test is to assess whether this ventricular drainage needs to be intermittent or permanent. When EVD is weaned quickly and CSF drainage occurs sometimes, it is safer, lowers the risk of infection and complications, and shortens hospital and ICU stays than when it happens gradually and continuously. Closure of the EVD and comparatively elevated CSF compartment pressure can promote CSF resorption via arachnoid granulation, thereby diminishing the necessity for VP shun (Chung et al., 2018). The results obtained after weaning tests on days 10 and 12 found that it was not shunt dependent, so it did not require the installation of a VP shunt. This can happen because SAH that occurs in patients does not experience complications until intraventricular hemorrhage because if the bleeding has reached intraventricular hemorrhage, it is necessary to install permanent drainage. On day 13, after installation, the EVD is removed. However, these patients still need observation because it does not rule out the possibility that after EVD removal recurrence of symptomatic hydrocephalus requires VP shun because according to a study of 91 patients who passed the brace trial and had their EVD removed, 12 (13%) required a delay in VP shunt installation with a median of 54 (interquartile range: 15-75) days after EVD release (Akinduro et al., 2020).

Coiling or clipping surgery is a very important option to get optimal results after a SAH. To determine the location of the rupture of blood vessels, CT angiography must still be done, but the hospital has limited equipment, so it is not done. The longer the operation is delayed, the greater the risk of re-bleeding in the aneurysm. In general, surgery is performed as soon as possible after a cerebral angiogram. In the past, surgery was avoided when patients experienced clinically or angiographically severe vasospasm, but it is now known that it is best to bypass the aneurysm despite clinical or radiological vasospasm because the aneurysm is removed from circulation (Wulandari et al., 2021).

CONCLUSION

SAH is an emergency condition with a sudden onset that begins with severe headaches and can cause a decrease in consciousness. Subarachnoid hemorrhage may be accompanied by hydrocephalus. Management with external ventricular drainage can be done for this case. When EVD is installed for 13 days in patients who are diagnosed SAH with hydrocephalus, the prognosis is good and the appropriate course of treatment is achieved. There are no complaints or abnormalities of the nervous system after EVD removed but before removal of external ventricular drainage, a clamp test to can be to determine whether the dependent shunt exists or not. Observation 54 days after EVD removal is necessary to assess whether hydrocephalus persists or not and to consider installation of VP shunt.

Conclusions are briefly explained and cover the aspects studied; make suggestions if any.

REFERENCES

Adrianto, Y., Sani, A.F., Susilo, H. (2016). Intracranical aneurysm management. *Journal of Axena*, 1(1): 15-21.

Agrawal, A., Dwivedi, S., Singh, B.R., Banode, P. (2012). Simultaneous hemorrhage in intracranial aneurysms and in renal cyst in a case of polycystic kidney disease. *Saudi Journal Kidney Diseases and Transplantation*, 23(4): 794-798. DOI: https://doi.org/10.4103/1319-2442.98163.

AK, W., Dubey, A., Chaturvedi, S., Kumar, S. (2020). Young COVID 19 presenting as fatal subarachnoid hemorrhage: association or chance?. *Medical Science*, 24(104): 2712-2715.

Akinduro, O.O., Vivas-Buitrago, T.G., Haranhalli, N., et al. (2020). Predictors of ventriculoperitoneal shunting following subarachnoid hemorrhage treated with external ventricular drainage. *Neurocrit Care*, 32(3): 755-764. DOI: https://doi.org/10.1007/s12028-019-00802-8.

Brooks, S.T., Webster, R.K., Smith, L.E., et al. (2020). The psychological impact of quarantine and how to reduce it: rapid review of the evidence. *The Lancet*, 395(10227): P912-920. DOI: https://doi.org/10.1016/S0140-6736(20)30460-8.

Chen, S., Luo, J., Reis, C., et al. (2017). Hydrocephalus after subarachnoid hemorrhage: pathophysiology, diagnosis, and treatment. *BioMed Research International*, 2017: 8584753. DOI: https://doi.org/10.1155/2017/8584753.

Chung, D.Y., Mayer, S.A., Rordorf, G.A. (2018). External ventricular drains after subarachnoid hemorrhage: is less more?. *Neurocrit Care*, 28(2): 157-161. DOI: https://doi.org/10.1007/s12028-017-0443-2.

Hidayat, R., Harris, S., Rasyid, A., et al. (2017). Subarachnoid hemorrhage. In: Textbook of Neurology. Neurology Department UI RSCM. Pp. 527-544.

Kairys, N., Das, J.M., Garg, M. (2022). Acute subarachnoid hemorrhage. In: StatPearls. Treasure Island (FL). StatPearls Publishing. URL: https://www.ncbi.nlm.nih.gov/books/NBK518975/.

Marcolini, E., Hine, J. (2019). Approach to the diagnosis and management of subarachnoid hemorrhage. *Western Journal of Emergency Medicine*, 20(2): 203-211.

Ohbuchi, H., Hagiwara, S., Arai, N., et al. (2021). Optimal timing and safety of the external ventricular drainage in patients with high-grade aneurysmal subarachnoid hemorrhage treated with endovascular coiling. *Journal of the Neurosurgical Society of Australasia*, 88: 63-69. DOI: https://doi.org/10.1016/j.jocn.2021.03.003.

Perhimpunan Dokter Specialis Saraf Indonesia. (2011). Guidelines stroke.

Winstein, C.J., Stein, J., Arena, R., et al. (2016). Guidelines for adult stroke rehabilitation and recovery: a Guidelines for healthcare professionals from the American Heart Association/American Stroke Association. Stroke, 47(6): e98-e169. DOI: https://doi.org/10.1161/STR.0000000000000098.

Wulandari, D.A., Hunaifi, I., Sampe, E. (2021). Subarachnoid hemorrhage (SAH). *Jurnal Kedokteran*, 10(1): 338-346. DOI: https://doi.org/10.29303/jku.v10i1.437.