



VP shunt in Non-Communicating Hydrocephalus due to Intracerebral Hemorrhage – A Case Report

Firman Muharam¹, Rully Hanafi Dahlan², Seyline Estethia Ompusunggu³

¹Department of Neurosurgery, Faculty of Medicine, Universitas Padjadjaran / Dr. Hasan Sadikin General Hospital, Bandung, West Java, Indonesia

² Division of Spine and Peripheral Nerves, Department of Neurosurgery, Faculty of Medicine,

Universitas Padjadjaran / Dr. Hasan Sadikin General Hospital, Bandung, West Java, Indonesia

³ Division of Spine and Peripheral Nerves, Department of Neurosurgery, Faculty of Medicine, Universitas Padjadjaran / Dr. Hasan Sadikin General Hospital, Bandung, West Java, Indonesia

Abstract. Stroke is one of the top three cause of death and disability globally. Approximately, only 10% to 15% of first-ever stroke are intracerebral hemorrhages (ICHs), but the rates of disability and death are significantly higher. Hydrocephalus may occur in more than 50% of patients with intraventricular hemorrhage (IVH), which is secondary to ICH. Intraventricular hemorrhage (IVH), an accumulation of blood to ventricles that may be cause by extension of ICH, occurs in up to 50% of patients with primary ICH. Hydrocephalus itself may serves as a predictor of poor outcome after ICH.1,2,3 A 71 years old male came to the hospital with the complaint of loss of consciousness, difficulty in communicating, and weakness of extremities on the left side of the body since 12 hours ago. The complaint was preceded by headache that did not alleviate with medication since 1 day before admission and there was history of slurred speech. A head CT scan without contrast was done and the result showed an intracerebral hemorrhage and a suggestive obstructive hydrocephalus. The patient was diagnosed noncommunicating hydrocephalus due to spontaneous intracerebral haemorrhage (ICH) at the right thalamus with intraventricular haemorrhage due to suspect hypertension with differential diagnosis of cerebral amyloid angiopathy with emergency hypertension. The patient was advised to underwent an emergency VP shunt placement. Implantation of a ventriculoperitoneal (VP) shunt is the most widely used treatment of hydrocephalus. Considered to be a major privonce of neurosurgery, it accounts for 70.000 hospital admission in the US. Even though, VP shunting of CSF reduces the morbidity and mortality of post-hemorrhagic. hydrocephalus, it is associated with potential complications requiring multiple surgical procedures, as well as shunt revision due to its failure during the patient's lifetime.4

Keyword: Intracerebral Hemorrhage, Hydrocephalus, Stroke, VP Shunt

Received date month year. | Revised date month year | Accepted date month year

^{*}Corresponding author at: [address of author's affiliation, city and country]

E-mail address: [author's email address]

1. Introduction

Intracerebral hemorrhage (ICH) is a disease with high mortality and morbidity rates. About 10% to 23% of strokes are caused by the rupture of cerebral blood vessels. Approximately, only 10% to 15% of first-ever stroke are intracerebral hemorrhages (ICHs). The overall incidence of ICH worldwide is 24.6 per 100.000 person per years. Recent review reported ICH had higher incidence in low-income countries compared to higher-income countries. Furthermore, ethnicity has also been reported to ICH risk. It was reported Asians had a two-fold higher rate of stroke compared to other ethnic groups.^{1,5}

Hypertension is one of the most important modifiable risk factor for ICH. Regardless of ethnicity, age, or gender, about 75% of ICH patients have pre-existing hypertension. Studies showed that high blood pressure have two to six times risk of ICH. Other common risk factor is cerebral amyloid angiopathy (CAA) which was the second most common risk factor for ICH, particularly lobar ICH. CAA itself was a condition that increases with age, thus more common to happen in older ages. Other leading causes include amyloid angopathy, tumors, hemorrhagic transformation of an ischemic stroke, cerebral venous thrombosis, vasculitis, and vascular malformations, e.g. cavernous malformation, arteriovenous malformation, and ruptured saccular aneurysms.^{1,6}

Hydrocephalus may occur in more than 50% of patients with intraventricular hemorrhage (IVH), which is secondary to ICH. IVH, which was characterized by an influx of blood into the ventricles, occurs in up to 50% of patients with primary ICH. IVH-induced hydrocephalus may occur as a result of blockage in the CSF drainage pathway due to mass effect of blood clot or as a result of absorption disturbances. Some studies showed that hydrocephalus is a predictor of poor outcome after ICH.^{7,8}

Shunting of CSF remains one of the most commonly done neurosurgical procedures for management of hydrocephalus patients. VP shunt is commonly used as a management of post-hemorrhagic hydrocephalus conditions, including subarachnoid hemorrhage (SAH), subdural hemorrhage (SDH), ICH, and IVH. Despite significant advancement of shunt technology and treatment approaches, the ideal management of hydrocephalus is yet to be established. Thus, VP shunting still becomes the preferred and commonly-used approaches in managing hydrocephalus, including post-hemorrhagic hydrocephalus in adults.^{4,9}

Case Report

A 71 years old male came to the hospital with the complaint of loss of consciousness, difficulty in communicating, and weakness of extremities on the left side of the body since 12

hours ago. The complaint was preceded by headache that did not alleviate with medication since 1 day before admission. There was history of slurred speech but no seizure, fever, or any prior trauma. Due to the patient's complaint, the patient was taken to Advent Hospital Emergency Room (ER) and was where head CT without contrast was done (08/09/2021). The patient was diagnosed with an intracerebral haemorrhage and was referred to Neurosurgery ER of Hasan Sadikin General Hospital. The patient had another head CT without contrast (15/09/2021) and was advised to underwent an emergency VP shunt placement.

There were no walking or gait disturbance, history of bedwetting, and forgetfulness. The patient had hypertension for 10 years with the highest systole measurement of 200 mmHg. There no prior history of stroke or diabetes. The patient had a history of smoking but no history of consuming any blood thinner nor alcohol.

From physical examination, the patient's vital sign were as follow: blood pressure 110/70 mmHg, PR 90 x/minute, RR 20 x/minute, and body temperature was afebrile (36.9 °C). From thorax examination, we found an equal vesicular breathing sound without rhonchi nor wheezing.

From neurological examination, the patient had GCS of 13 (E4M5V4) without nuchal rigidity. Pupil was round and isochoric with diameter 3 mm each and positive light reflex. Visual acuity of both eyes was difficult to assess. Funduscopic examination was not conducted. Eyeball movement and other cranial nerve examination were difficult to assess. Left hemiparesis was found on motoric examination and from sensory examination it was found that the patient still responded to pain stimulation. Physiological reflex was reduce on the right side and normal on the left side of body. Babinski's reflex was negative on the right side and positive on the left side of body. NIH Stroke Scale/NIHSS was 16 indication moderate to severe neurological deficit.

Laboratory examination in Advent Hospital (08/09/2021) result was Hb 15.0 g/dl, Ht 43.1, leukocyte 13.200, thrombocyte 253.000, and PCR COVID-19 was negative. Laboratory examination in Hasan Sadikin General Hospital (15/09/2021) result was Hb 12.9 g/dl, Ht 36.7, leukocyte 9/520, thrombocyte 251.000, and PCR COVID-19 was negative. Random blood glucose of patient was 131 mg/dl, Na/K was 137/2.0, ureum level 45.0, creatinine 0.90, OT/PT was 35 and 48, and albumin was 2.91. Thorax x-ray conducted in Advent Hospital (08/09/2021) was within normal limit.

Advent Hospital head CT without contrast (08/09/2021) result was sulcus, gyrus, and Sylvian fissure , and Cisterna was not compressed. TH> 2 mm, FH/ID 36%, and Evans ratio was 0.38. Hyperdense lesion appeared to fill the lateral ventricle and was found on right thalamus with volume of 12 cc. Calcification was found bilaterally on basal ganglia and cerebellum. There was no midline shift. ICH score was 1 and mGraeb score was 2. The result is showed in Figure 1 below.



Figure 1. Head CT Scan without contrast result from Advent Hospital (08/09/2021)

Advent Hospital head CT without contrast (15/09/2021) result was sulcus, gyrus, and Sylvian fissure, and Cisterna was not compressed. TH> 2 mm, FH/ID 43%, and Evans ratio was 0.33. Hyperdense lesion appeared to fill the lateral ventricle and was found on right thalamus with volume of 8 cc. Periventricular edema was found. Calcification was found bilaterally on basal ganglia and cerebellum. There was no midline shift. ICH score was 1 and mGraeb score was 2. The result is showed in Figure 2 below.



Figure 2. Head CT scan without contrast result from Advent Hospital (15/09/2021)

We diagnosed the patient with spontaneous intracerebral haemorrhage (ICH) at the right thalamus with intraventricular haemorrhage due to suspect hypertension with differential diagnosis of cerebral amyloid angiopathy with emergency hypertension. The patient was planned to undergo emergency VP shunt placement and watchful observation in semi-intensive care unit. The advice from Internal Medicine Department was to regulate the patient's blood pressure and to treat patient's general condition.

From the intraoperative findings of VP shunt placement, on the left kocher, we did proximal shunt insertion (depth of 5 cm) and the opening pressure was 3 cmH20 with clear yellowish cerebrospinal fluid (CSF). On the right hypochondrium, we did distal shunt insertion (depth of 25 cm) and the shunt pump test had a positive result with the shunt pump working effectively. We used VP shunt intergra Flatbottom with type medium pressure on the patient.

Discussion

Hydrocephalus is associated with ICH, especially IVH secondary to ICH. Study from Bhattathiri showed that likelihood of good outcome is decreased as much as 15.1% to 11.5% due to the presence of hydrocephalus. Up to 50% of patients with IVH secondary to ICH may develop hydrocephalus. Hypertension, followed by CAA, was the most common modifiable risk factor of ICH. In the case of spontaneous ICH on patient with preexisting hypertension, Charcot-Bouchard aneurysms may ruptured as a from lipohyalinosis of small arterioles, causing ICH involving the cerebellum, pons, thalamus, and basal ganglia.^{2,10}

Approximately, more than 85% of ICHs occur as a primary event that associated to small penetrating arteries and arterioles ruptured which was damaged by chronic arterial hypertension. Besides occurring secondary to ICH, IVH in adults can also be a result of aneurysmal SAH, vascular malformation, and trauma. As an addition, ICH with intraventricular extension is more likely to occur if the hemorrhage happened in periventricular location such as the thalamus and caudate. Increased volume of ICH and significant hypertension are also associated with ICH-associated IVH in older age adults.^{2,11}

The mortality of ICH is about 40% within the first 30 days after ICH, making ICH one of the most deadly acute medical event. Death in the first month after ICH, but beyond the first few days after onset, may occurs as a result of ICH complication. At least 1 medical complication was found in 29% of all ICH admission. ICH complication that usually occurs are cerebral hernia, pulmonary infection, respiratory failure, multiple organ failure, rebleeding, intracranial infection, or extension of bleeding to ventricles, leading to IVH and expansion of ventricles causing mass effect and hydrocephalus as of this case. Though, one study showed the highest mortality still was in brainstem ICH rather than IVH.¹

IVH-induced hydrocephalus may occur as a result of blockage in the CSF drainage pathway. Acute obstructive hydrocephalus after IVH occurs from blood blocking the cerebral aqueduct or the fourth ventricular outlets, the foramen of Luschka and foramen of Magendie. Other than that, hydrocephalus may occur due to IVH after ICH as a result of several different etiology depending the onset of hydrocephalus after ICH. IVH can lead to both immediate hydrocephalus or delayed hydrocephalus. Early onset hydrocephalus, which happens within 3 days after ICH, may be caused by hematoma expansion or its extension to ventricles causing IVH. Mass effect due to the hematoma or IVH may cause early noncommunicating hydrocephalus, which was also known as obstructive hydrocephalus. Hematoma expansion, a common cause of secondary neurologic deterioration, is one of the most common modifier of survival and functional outcomes in up to one-third of patients after ICH occurs.^{10,11}

IVH-induced hydrocephalus may occurs as a result of alteration of CSF drainage pathway, especially ones that related to the cerebral aqueduct, fourth ventricular outlets, and the arachnoid villi or granulation. Expansion of ventricles, which happen secondary to loss of brain tissue from atrophy, may also occurs as a cause for hydrocephalus. Other than that, extension of hemorrhage into the ventricles, which also may happen within 4 until 13 days or more than 14 days after ICH, may prevent normal CSF as a result of mass effects of blood clot.^{2,12,13}

Delayed-onset hydrocephalus, which happens after 14 days or more after ICH, may also be caused by decompressive craniotomy and intracranial infection. Decompressive craniotomy may effectively saves life, but a large decompressive craniotomy procedure may aggravate ventricular expansion and reduce the CSF absorption due to lower intracranial pressure. Hydrocephalus itself can cause injury to the brain as a result of varied mechanism such as inflammation, ventricular distention disrupting periventricular fibers, and increased intracranial pressure with decreased cerebral perfusion.^{2,14}

VP shunt, which is commonly used as a management of post-hemorrhagic hydrocephalus, may have some complication and failure. Shunt failure may be caused by infection, obstruction, over-drainage, and mechanical disconnection or breakage. Although, these subject is predominantly studied in pediatrics population with very little studies focused in evaluating shunt complication and failure in adult population. But similarly to pediatrics population, VP shunt failure in adult requires a neurosurgical intervention. The overall incidence of shunt revision was 51.9% in adult patients with hemorrhage-related hydrocephalus and majority occurring within first 6 months of procedures. Endoscopic ventriculostomy has been developed as an alternative to avoid invasive surgery or shunt insertion in post-hemorrhage-associated hydrocephalus though VP shunt remains the best treatment option for hydrocephalus patients due the lack of efficacy studies regarding the ventriculostomy.^{4,15}

Conclusion

Hydrocephalus may occur in more than 50% of patients with intraventricular hemorrhage (IVH), which is secondary to ICH. . Hydrocephalus itself may serves as a predictor of poor outcome after ICH. Hypertension, followed by CAA, was the most common modifiable risk factor

of ICH. IVH-induced hydrocephalus may occur as a result of blockage in the CSF drainage pathway due to mass effect of blood clot or as a result of absorption disturbance. Hematoma expansion or its extension to ventricles causing IVH may result of the associated mass effect, which usually happen as early-onset hydrocephalus. Delayed-onset hydrocephalus, which happens after 14 days or more after ICH, may also be caused by decompressive craniotomy and intracranial infection VP shunt, which is commonly used as a management of post-hemorrhagic hydrocephalus, may have some complication and failure. There is still a need for further research regarding the ideal management guidelines for post-hemorrhagic hydrocephalus. Thus, until result of further research becomes available, VP shunt remains the best treatment option for hydrocephalus patients.

References

- Hu YZ, Wang JW, Luo BY. Epidemiological and clinical characteristics of 266 cases of intracerebral hemorrhage in Hangzhou, China. J Zhejiang Univ Sci B. 2013 Jun;14(6):496-504.
- Hu R, Zhang C, Xia J, Ge H, Zhong J, Fang X, Zou Y, Lan C, Li L, Feng H. Long-term Outcomes and Risk Factors Related to Hydrocephalus After Intracerebral Hemorrhage. Transl Stroke Res. 2021 Feb;12(1):31-38.
- Garton T, Keep RF, Wilkinson DA, Strahle JM, Hua Y, Garton HJ, Xi G. Intraventricular Hemorrhage: the Role of Blood Components in Secondary Injury and Hydrocephalus. Transl Stroke Res. 2016 Dec;7(6):447-451.

- Reddy GK, Bollam P, Shi R, Guthikonda B, Nanda A. Management of adult hydrocephalus with ventriculoperitoneal shunts: long-term single-institution experience. Neurosurgery. 2011 Oct;69(4):774-80
- Crandall KM, Rost NS, Sheth KN. Prognosis in intracerebral hemorrhage. *Rev Neurol Dis*. 2011;8(1-2):23-29.
- Carhuapoma, J.R., Mayer, S.A., Hanley, D.F., 2009. In- tracerebral Hemorrhage. Cambridge University Press, New York, USA, p.1-16.
- Strahle J, Garton HJ, Maher CO, Muraszko KM, Keep RF, Xi G. Mechanisms of hydrocephalus after neonatal and adult intraventricular hemorrhage. *Transl Stroke Res.* 2012;3(Suppl 1):25-38.
- Staykov D, Bardutzky J, Huttner HB, Schwab S. Intraventricular fibrinolysis for intracerebral hemorrhage with severe ventricular involvement. Neurocrit Care. 2011;15:194–209.
- Reddy GK. Ventriculoperitoneal shunt surgery and the incidence of shunt revision in adult patients with hemorrhage-related hydrocephalus. *Clin Neurol Neurosurg*. 2012;114(9):1211-1216.
- Ziai WC, Carhuapoma JR. Intracerebral Hemorrhage. *Continuum (Minneap Minn)*. 2018;24(6):1603-1622.
- 11. Druid H, et al. The Swedish cause of death register. Eur J Epidemiol. 2017;32(9):765-73
- Hwang BY, Bruce SS, Appelboom G, Piazza MA, Carpenter AM, Gigante PR, et al. Evaluation of intraventricular hemorrhage assess- ment methods for predicting outcome following intracerebral hem- orrhage. J Neurosurg. 2012;116(1):185–92.
- Hansen BM, Nilsson OG, Anderson H, Norrving B, Säveland H, Lindgren A. Long term (13 years) prognosis after primary intrace- rebral haemorrhage: a prospective population based study of long term mortality, prognostic factors and causes of death. J Neurol Neurosurg Psychiatry. 2013;84(10):1150–5.
- Brouwers HB, Chang Y, Falcone GJ, Cai X, Ayres AM, Battey TW, et al. Predicting hematoma expansion after primary intracere- bral hemorrhage. JAMA Neurol. 2014;71(2):158–64.
- Whitelaw A, Aquilina K. Management of posthaemorrhagic ven- tricular dilatation. Arch. Dis. Child. Fetal Neonatal Ed. 2011