



Subarachnoid Hemorrhage: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

We reported a case report of a 50-year-old woman with stroke hemorrhage due to subarachnoid hemorrhage with hypertensive urgency, left ventricular hypertrophy, and dyslipidemia. Subarachnoid hemorrhage indicates the presence of blood in the subarachnoid space between the pia mater and arachnoid mater which usually results from a ruptured cerebral aneurysm or arteriovenous malformation. The patient presents with decreased consciousness preceded by severe headache and projectile vomiting. In physical examination, we found hypertensive emergencies and positive meningeal signs, neck stiffness, and positive Brudzinski. CT scan shows bleeding in the pontocerebellar cistern and ventricular system. The patient was diagnosed with subarachnoid hemorrhage, intraventricular hemorrhage, and emergency hypertensive. The patient was hospitalized in the neurology ward of Ulin Hospital for 20 days with the management of antihypertensive, neuroprotectant, other symptomatic medications, and ventriculoperitoneal shunt surgery. The patient was then discharged home in a stable condition.

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1. INTRODUCTION

Subarachnoid hemorrhage or SAH indicates by the presence of blood in the subarachnoid space, the space between the pia mater and arachnoid mater, due to several pathological processes. SAH refers to a non-traumatic type of bleeding, usually from ruptured cerebral aneurysm or arteriovenous malformation (MAV). Aneurysm formation can be caused by atherosclerosis, hypertension, degenerative process, smoking, and hemodynamic stress [1].

The annual incidence of non-traumatic SAH aneurysms is about 6-25 cases of 100,000 people. More than 27,000 Americans suffer ruptured intracranial aneurysms each year. The annual incidence increases with age and may be underestimated because the death is associated with other causes that cannot be confirmed at autopsy. Various incidents of SAH have been reported in other areas of the world in about 2-49 cases of 100,000 people [1].

Rupture of blood vessels can occur at any age, but most often affects the population in 25-50 years of age. Subarachnoid hemorrhage is rare after a head injury. MAV condition is more often found in men than women population [2].

The main symptom of subarachnoid hemorrhage is a severe headache that occurs suddenly. Headaches are often instantaneous or cataclysmic. Transient loss of consciousness and seizures are common and often occur at the onset of bleeding. In most patients with subarachnoid hemorrhage, there are no signs of focal neurologic deficit. Patients often require emergency neurosurgical and neuroradiological intervention [2].

Subarachnoid hemorrhage is an emergency that needs immediate treatment. General practitioners in the ER must be able to provide emergency management before the patient is referred to the relevant specialist doctors for further treatment. It's important to have a good grip of subarachnoid diagnosis and treatment. The case report is expected to provide an overview in the diagnosis and management of patients with subarachnoid hemorrhage.

This case report is expected to delineate the diagnosis and management of patients with subarachnoid hemorrhage.

2. CASE REPORT

Here we report a case of a 50-year-old woman who was admitted to Seruni Room (neurology ward) at Ulin Regional General Hospital in Banjarmasin. The patient was diagnosed with stroke hemorrhage due to subarachnoid hemorrhage and urgency hypertension, left ventricular hypertrophy, and dyslipidemia. The patient was brought by her husband with the chief complaint of a sudden loss of consciousness during activities. Previously, the patient had a history of severe headache, vomiting without nausea more than 3 times, heavy and stiffness of the neck after late sleeping at midnight. History of seizures was denied. The patient received an infusion at the public health center; a drugs injection, chest X-ray, and blood laboratory examination at Husada Batu Licin Hospital; then was referred to Ulin Hospital for CT Scan examination. The patient suffers from hypertension but does not take medication regularly. Diabetes mellitus, heart disease, dyslipidemia, stroke and trauma, consumption of anticoagulant drugs are denied. Patient on a high-salt, oily, coconut milk, and sweet diet. Smoking habit is approximately 6 cigarettes a day.

On arrival at the ER, the patient was stuporous (GCS E3M3V6), critical hypertension (the blood pressure was 180/100 mmHg), nuchal rigidity, and Brudzinski I was positive. Physical and other neurological examinations were within normal limits. Complete blood laboratory results showed leukocytosis ($17.3 \times 10^3/\text{ul}$), thrombocytosis ($531 \times 10^3/\text{ul}$), dyslipidemia with an LDL level was 200 mg/dl. ECG examination concluded left ventricular hypertrophy.

Based on the results of the CT scan in Fig. 1, a hyperdense lesion was seen that filled all the cisterns, the left lateral ventricle, and the third ventricle. It was concluded that the patient had SAH and IVH.

The patient was treated in the neurological ward with 20 drops/minute of NaCl fluid therapy, citicoline 250 mg every 12 hours intravenously, an ampoule of ranitidine every 12 hours intravenously, a dose of metamizole every 8 hours if necessary, nimodipine 2.1 cc/hour in a syringe pump intravenously, nimodipine 60 mg every 4 hours orally, amitriptyline 0-0.5 tablet orally, acetazolamide 250 mg every 12 hours

intravenously, an ampoule of haloperidol every 12 hours if necessary.

After being treated for 16 days, CT scan evaluation was done and the result showed abnormal hyperdense blood density lesions in the pontocerebellar cisterna, dilatation of the ventricular system, accompanied by intraventricular hyperdense lesions; sulcus, gyri, and Sylvian fissure were normal, open cisternae, and normal soft tissue, no abnormalities were found in the intracerebral cerebellum and pons. It was concluded that the patient had a subarachnoid hemorrhage and communicating hydrocephalus.

3. DISCUSSION

In the anamnesis, we found that about 3 days earlier the patient had experienced severe headaches, accompanied by projectile vomiting. The presence of seizures, memory loss, slurred speech, sore mouth, and sensibility disturbances

in the form of numbness and tingling are not present. The patient daily uses the right hand for activities and denies any weakness. From the results of the anamnesis, it can be concluded that the cause of the patient's complaints is a disturbance in the cerebrovascular system. A possible diagnosis that can be considered is cardiovascular disease (CVD) or stroke [1].

History of uncontrolled hypertension, supported by ECG results showed left ventricle hypertrophy. This is a sign of chronic hypertensive conditions. Smoking is also a risk factor for CVD [3]. Smoking increases the risk of thrombotic stroke and subarachnoid hemorrhage. The relative risk of subarachnoid hemorrhage in smokers compared to nonsmokers is 2.7 in men and 3.0 in women [4]. The patient also suffers from dyslipidemia. Research shows a relationship between high serum lipid levels and carotid artery thickening which is an indicator of atherosclerosis as evidenced by ultrasound examination [5].

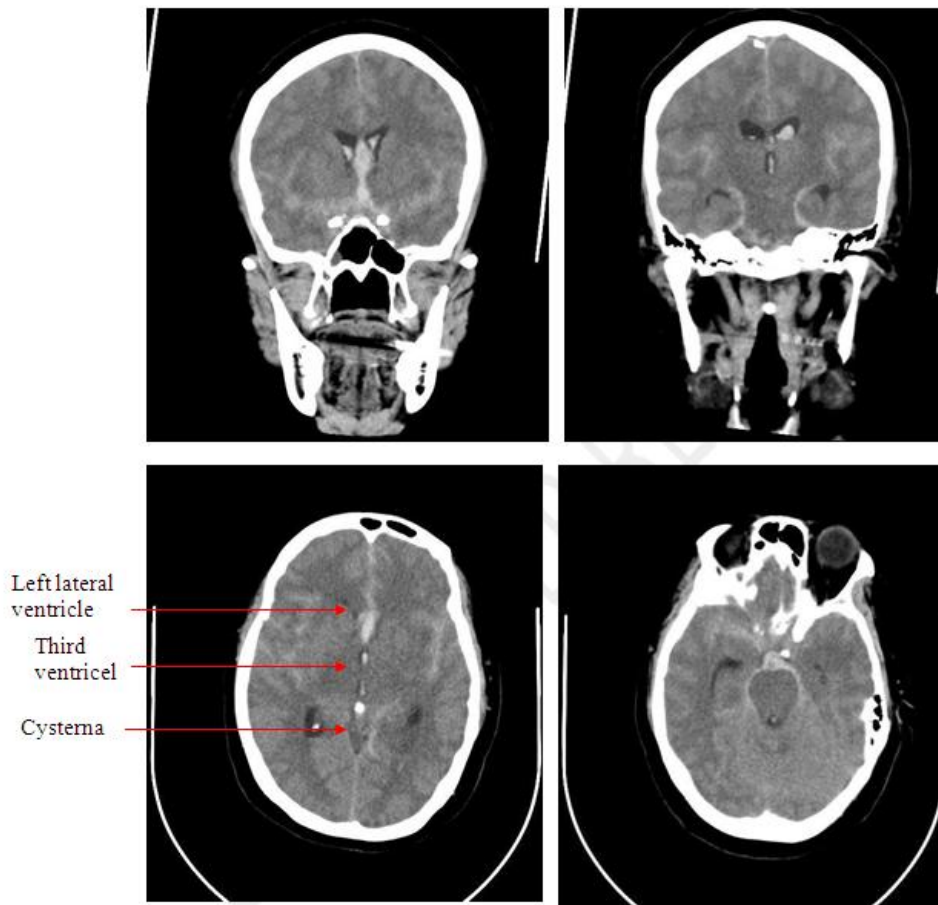


Fig. 1. CT scan of the head without contrast in the ER

A significant physical examination that we found the blood pressure was 180/100 mmHg (hypertension emergency category), accompanied by the positive meningeal sign. These findings can lead to the diagnosis of meningeal disorders [6]. In general, CVD can be classified into hemorrhagic and non-hemorrhagic CVD. To distinguish the two, when a CT scan is not yet possible, a scoring system can be used which is assessed through anamnesis. The first scoring system that can be used is Siriraj Score. From Siriraj Stroke Score (SSS) the patient was found to be +4.5 (tendency towards hemorrhagic stroke) [7].

In addition, if we use other scoring systems such as the Gajah Mada Stroke Algorithm (ASGM), it also shows the presence of bleeding stroke. However, because this scoring system has low sensitivity and specificity, other investigations are still needed to make a definite diagnosis. From the anamnesis, physical examination, neurological examination, and scoring system that has been carried out, it can be considered a possible diagnosis in the patient is CVD hemorrhagic et causa subarachnoid hemorrhagic (SAH).

In subarachnoid hemorrhage, the etiology is mostly due to rupture of the aneurysm of the brain blood vessel. An aneurysm is an injury caused by hemodynamic pressure on the artery wall branching and bending. Sudden rupture of a vascular aneurysm will cause the subarachnoid space fulfilled with blood and causes the manifestations as a severe headache felt by the patient. The presence of blood in the subarachnoid space will also irritate the meningeal layer which is then shown clinically as nuchal rigidity. In some cases, 3-12 hours after the bleeding this sign of nuchal rigidity may disappear. However, the absence of this nuchal rigidity, cannot immediately rule out the diagnosis of subarachnoid hemorrhage [7,8]. Subarachnoid hemorrhage does not always show a focal neurological deficit but can be found in intraparenchymal bleeding, compression of cranial nerves, or ischemic lesions due to vasospasm [8].

The complimentary examination of choice is a CT scan without contrast because of its high sensitivity (close to 100% if performed within the first 12 hours after the stroke attack) and can determine the location of bleeding more accurately. This examination is still the gold standard for CVD hemorrhagic. From the results

of the first CT scan (Fig. 1), results showed hyperdense lesions that filled the ambient and basal cisternae areas accompanied by hyperdense lesions with dilatation of the right lateral ventricles and third ventricles, suggesting subarachnoid hemorrhage and intraventricular hemorrhage [7,9].

Furthermore, during the treatment period, the patient underwent a head CT scan without contrast for the second time to evaluate any improvement and progress of the disease. From the results of the second CT scan, it was found that the bleeding density hyperdense lesion filled the pontocerebellar cistern with dilatation of the ventricular system and it was concluded that the patient had communicating hydrocephalus.

To assess hydrocephalus by using a CT scan or MRI, we need to assess the size of the temporal horns (TH) from end to end >2mm (if there is no hydrocephalus, the temporal horn is difficult to see). We can also assess the frontal horn (FH) divided by the distance between the internal tabula (ID). There may also be a frontal horn from lateral ventricular ballooning. In addition, we can also use the Evans Ratio by assessing the ratio of FH to the maximum distance from the biparietal diameter. It will be diagnosed as hydrocephalus if the Evans ratio is >30% [10].

Hydrocephalus is a condition characterized by the increases in intracranial pressure due to the accumulation of cerebrospinal fluid (CSF) in the ventricular system of the brain due to production, flow, and absorption of cerebrospinal fluid imbalance. Hydrocephalus is categorized into non-communicating hydrocephalus (obstructive) and communicating hydrocephalus. Obstructive hydrocephalus easily occurs when there is an obstruction of CSF flow in the ventricle. The blockage can occur in the ventricular foramen. Meanwhile, communicating hydrocephalus occurs when there is an increase in CSF pressure without a blockage between the ventricular and subarachnoid systems. This can be caused by impaired absorption of CSF, excess CSF production, or venous drainage insufficiency [11,12].

Chen et al explained the relation between SAH and the occurrence of hydrocephalus. SAH can cause hydrocephalus in 2 mechanisms: (1) the mechanism of flow resistance from CSF (acute, obstructive, non-communicating type), and (2) the mechanism of arachnoid granulation blockage due to scar (delayed, non-obstructive,

and communicating type). In communicating type, after the SAH attack, the subarachnoid space will be filled with blood cells. Thickening of the leptomeninges is seen with deposits of hemosiderin (assessed histologically), this condition disrupts CSF absorption to reach the vein system [12].

Initial general management aimed at preventing worsening of the patient's condition includes (1) primary survey (ABC in cardiopulmonary resuscitation), (2) management of hypertension, (3) fluid and electrolyte balance, (4) gradual headache management from mild analgesics (paracetamol to codeine) to heavy analgesics (morphine injection) [13,14].

Treatment of hypertension with a fast-acting agent (nimodipine), which is the best vasospasm preventer according to the AHA with level A evidence. This CCB group can reduce the incidence of ischemia of neurological deficits with its neuroprotective effect on calcium influx in damaged neurons. Antihypertensives should be given carefully. Our target is to reduce it to premonitory blood pressure or 15-20% if systolic pressure >200 mmHg, diastolic >150 mmHg, and MAP >130 mmHg. If systolic blood pressure is 180mmHg or MAP >130 mmHg accompanied by symptoms of increased ICP, then blood pressure should be lowered with continuous or intermittent intravenous antihypertensive drugs with blood pressure monitoring every 15 minutes to a MAP of 110 mmHg or up to a blood pressure of 160/90 mmHg. In this patient, the blood pressure was 180/100 mmHg in ER and the MAP was 127 mmHg, this needs to be lowered [15].

Hemorrhagic stroke patients admitted to the ICU with an indication of hematoma volume >30 mL, the presence of intraventricular hemorrhage with hydrocephalus, and clinical deterioration. In this patient, the initial CT scan revealed a subarachnoid hemorrhage with intraventricular hemorrhage and hydrocephalus. The patient had decreased consciousness. This makes it possible to be treated in the intensive care unit for intensive monitoring.

Non-medical management given to patients includes family and patient motivation. The patient has lied with an elevated head about 30 degrees. Prevention of recurrent bleeding in subarachnoid hemorrhage is very important because it is estimated that 35-40% of cases occur in the first 4 weeks in patients who are still alive on the first day. The incidence of rebleeding

occurs in the first 6 hours, and approximately 20% on the first day.

Prevention of cerebral ischemia is influenced by the amount of blood that appears during the first CT scan, a marked decrease in consciousness after an attack, the presence of hypovolemic or hyponatremic conditions, excessive antihypertensive medication causing hypoperfusion, and the presence of meta-analytical evidence suggesting that antithrombotic drugs can trigger the cerebral ischemia.

The patient was also treated for hydrocephalus. There are 3 principles in the treatment of hydrocephalus: reducing the production of cerebrospinal fluid by giving acetazolamide tablets, improving the relationship between the site of cerebrospinal fluid production, and the release of cerebrospinal fluid into extracranial organs. Drug treatment in hydrocephalus is only delayed surgical intervention and is considered ineffective for the long term [16].

Our patient also received surgical intervention for the installation of a shunt. The principle of shunt placement is to establish a connection between the cerebrospinal fluid (ventricle/lumbar) and the final cavity for drainage (peritoneum, right atrium, pleura). There are several options for shunt installation that can be done such as ventriculoperitoneal (VP) shunt, ventriculoatrial (VA) shunt, lumboperitoneal (LP) shunt, etc. The choice between each shunt installation is based on the patient's condition [15].

The last option in treating hydrocephalus if the installation of a shunt is not possible or there is a threat of herniation (impending herniation) is by doing a serial lumbar puncture to decrease the CSF pressure and allow the CSF absorption by the arachnoid villi [15].

The prognosis in patients with SAH is generally dubia ad bonam, unless other factors aggravate the patient's condition such as changes in mental condition and the presence of neurological deficits on examination. In this patient, there were no aggravating factors, so it can be estimated that the patient's prognosis is dubia ad bonam. In addition, to determine the prognosis, the Hunt and Hess Grading Scale can also be used. This tool is used to measure the risk or severity of subarachnoid hemorrhage based on the first neurological examination.

From the results of the examination, the patient was categorized in Grade I with a mild headache and meningeal irritation. Patients with the Grade I Hunt and Hess grading scale have a much better prognosis than Grade II to V with good outcomes found in almost 95% of patients.

4. CONCLUSION

Stroke attack due to a rupture of arteriovenous malformation that occurs suddenly really requires proper diagnosis and prompt treatment to identify the source of bleeding, determine the next definitive treatment, and prevent the complications. Subarachnoid hemorrhages are preceded by a severe headache. In this case, the physical examination showed a decrease of consciousness, emergency hypertension, and meningeal signs (in this case we found positive Brudzinsky). Blood laboratory results showed leucocytosis, thrombocytosis, and hyperlipidemia as one of the risk factors for atherosclerosis that precedes aneurysms. Siriraj Stroke Score indicates the probability of a hemorrhagic stroke (+4.5 points). MRI was not performed in this patient, the diagnosis was confirmed by radiological CT scan of the head which showed SAH and IVH.

Appropriate treatment generally results in a good prognosis as long as there are no other factors that aggravate the patient's condition include the neurological deficit symptoms. This patient was discharged home in a stable condition after being hospitalized with antihypertensive, neuroprotectant, other symptomatic medications, and ventriculoperitoneal shunt surgery.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

CONSENT AND ETHICAL APPROVAL

As per international standard or university standard guideline patient's consent and ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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