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Ischemic Stroke

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A B S T R A C T

Ischemic stroke is a major cause of mortality and long-term disability worldwide, including in Indonesia. This condition occurs due to an interruption of cerebral blood flow caused by arterial occlusion or stenosis, leading to hypoxia and neuronal cell death. This article aims to review ischemic stroke by discussing its definition, epidemiology, etiology, pathophysiology, clinical classification, diagnosis, and management based on literature studies and a case report. The case presented involved a patient with sudden-onset right-sided weakness accompanied by headache, nausea, and vomiting. Clinical examination, Siriraj Stroke Score, and head CT scan confirmed a diagnosis of thrombotic ischemic stroke involving the left parietal and frontal lobes. Management consisted of acute-phase supportive therapy and pharmacological treatment, including neuroprotective agents, antihypertensive drugs, and supportive medications. Early diagnosis and appropriate management are crucial to reduce neurological deficits, improve functional outcomes, and enhance prognosis in ischemic stroke patients

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INTRODUCTION

Stroke is the third most common disease after heart disease and cancer, and it is the leading cause of disability worldwide. According to the American Heart Association (AHA), in 2015 the prevalence of stroke reached 33 million patients worldwide. Stroke is the fifth leading cause of death in America, with 795,000 patients per year and 129,000 deaths.

Stroke is the leading cause of death in almost all hospitals in Indonesia, accounting for around 15.4%. The Basic Health Research (Riskesdas) results from the Indonesian Ministry of Health in 2013 showed an increase in the prevalence of stroke in Indonesia from 8.3 per thousand (in 2007) to 12.1 per thousand (in 2013). The highest prevalence of stroke was in North Sulawesi (10.8 per thousand), Yogyakarta (10.3 per thousand), Bangka Belitung (9.7 per thousand), and DKI Jakarta (9.7 per thousand).

Cerebrovascular disease (CVD) is the most commonly encountered neurological disorder. Stroke is a part of CVD. According to the World Health Organization (WHO), stroke is a clinical manifestation of rapidly or suddenly developing focal or global cerebral dysfunction, lasting more than 24 hours or resulting in death, with no apparent cause other than vascular origin. According to the American Heart Association (AHA), stroke is characterized as a neurological deficit associated with acute focal injury to the central nervous system (CNS) caused by blood vessels, including cerebral infarction, intracerebral hemorrhage (ICH), and subarachnoid hemorrhage (SAH).

A stroke occurs when brain tissue is damaged due to a reduced blood or oxygen flow to brain cells. There are two types of stroke: ischemic stroke and hemorrhagic stroke. Ischemic stroke happens due to reduced blood flow, while a stroke caused by bleeding in or around the brain is called a hemorrhagic stroke. The bleeding that occurs in hemorrhagic stroke can quickly produce neurological symptoms due to pressure on the nervous structures inside the skull. Hemorrhagic stroke is less common than ischemic stroke, but hemorrhagic strokes result in more deaths.

Ischemic stroke occurs when the blood supply to a part of the brain is suddenly disrupted (ischemic) due to arterial occlusion or stenosis. This occlusion is caused by thrombosis and embolism, both of which can lead to hypoperfusion, which is a reduction or disturbance in cerebral blood flow (CBF) that results in decreased flow or intake of glucose and oxygen, thereby affecting neurological function (5).

LITERATURE REVIEW

Ischemic Stroke

According to WHO (2005), stroke is a clinical manifestation of cerebral dysfunction, either focal or global, that occurs suddenly, lasts more than 24 hours, or results in death, with no cause found other than vascular disorders (6). Non-hemorrhagic stroke is defined as a set of clinical signs that appear due to vascular causes. These symptoms last 24 hours or more, generally occurring due to reduced blood flow to the brain, causing disability or death (7). About 85% of strokes are non-hemorrhagic, occurring due to obstruction or clots in one or more large arteries in the cerebral circulation. The obstruction can be caused by a clot

(thrombus) formed within a brain vessel or distal organ vessel. A dislodged thrombus can become an embolus (8).

Epidemiology

Stroke is the third leading cause of death in developed countries, after heart disease and cancer. The annual incidence is 2 per 1,000 population. In the United States, stroke ranks third as a cause of death after heart disease and cancer. Every year, 500,000 Americans suffer a stroke, of which 400,000 experience ischemic stroke and 100,000 suffer hemorrhagic stroke (including intracerebral and subarachnoid hemorrhage), with 175,000 people dying (9). In 2007, the prevalence of stroke in Indonesia was found to be 8.3 per 1,000 population, and those diagnosed by healthcare professionals were 6 per 1,000 population. This indicates that about 72.3% of stroke cases in the community have been diagnosed by healthcare professionals. National data released by the Ministry of Health of the Republic of Indonesia states that stroke ranks first as the leading cause of death (10).

Etiologi

The causes of non-hemorrhagic stroke include :

Thrombosis

Thrombotic stroke can be divided into stroke in large blood vessels (including the carotid artery system) and small blood vessels (including the Circle of Willis and the posterior circulation). The most common site of thrombosis is the branching points of the cerebral arteries, particularly in the distribution area of the internal carotid artery. Arterial stenosis can cause blood flow turbulence, thereby increasing the risk of atherosclerotic thrombus formation (plaque ulceration) and platelet aggregation. Other causes of thrombosis include polycythemia, sickle cell anemia, fibromuscular dysplasia of the cerebral arteries, and prolonged vasoconstriction due to migraine disorders. Any process that causes cerebral artery dissection can also lead to thrombotic stroke (for example, trauma, thoracic aortic dissection, arteritis) (11).

Embolism

The areas most commonly affected by embolic stroke are in the anterior circulation (branches of the internal carotid artery) and in the vertebrobasilar arteries. Sources of emboli include, among others (12):

Pathophysiology

Non-hemorrhagic stroke is caused by a decrease in blood flow or even a complete stoppage in certain areas of the brain, which can cause neurons to stop functioning. Disruption of blood flow in the brain can lead to impaired supply of oxygen and glucose. If this supply disruption occurs beyond the cells' tolerance limit, it can result in cell death. Conversely, if blood flow can be restored promptly, the damage can be minimized.

Ischemic neuronal injury is a rapidly developing biochemical process. A lack of oxygen and glucose can deplete the cell's energy reserves, which are needed to maintain membrane potential and transmembrane ion gradients. Potassium leaving the cell triggers depolarization, calcium influx, and also stimulates the release of glutamate via glial glutamate transporters. Synaptic glutamate will activate excitatory amino acid receptors coupled with calcium and

sodium ion channels. The influx in postsynaptic neurons and dendrites will cause depolarization and acute edema. Excessive calcium influx will result in the activation of calcium-dependent enzymes such as proteases, lipases, and nucleases. Enzymes along with their metabolic products (eicosanoids and free radicals) will cause the breakdown of the plasma membrane and cytoskeletal elements, which can result in cell death. This sequence of events is called excitotoxicity due to the involvement of excitatory amino acids such as glutamate (11,13).

If the ischemia that occurs is not extensive, it can allow cells to survive longer, such as at the border between the ischemic area and the area that still receives adequate perfusion, known as the penumbra. This biochemical process can involve the expression of proteins such as Bcl (B-cell lymphoma)-2 protein and caspases (pro-enzymes for cysteine proteases). These proteins are involved in cell apoptosis (13).

Classification

Non-hemorrhagic stroke can be found in 4 clinical stages or time divisions, including :

Transient Ischemic Attack (TIA)

TIA describes an acute episode of neurological deficit that is short-lasting, less than 24 hours, and resolves without any residual symptoms.

Residual Ischemic Neurological Deficit (RIND)

RIND is almost the same as TIA but lasts more than 24 hours and recovers completely within less than 3 weeks.

Progressive stroke

Stroke with neurological deficits that occur gradually and reach their peak within 24-48 hours (carotid system) or 96 hours (VB system) with incomplete recovery within 3 weeks.

Diagnosis

The diagnosis of stroke is made based on the presence of sudden, diverse neurological clinical symptoms ranging from focal motor symptoms, sensory symptoms, higher function disturbances, to impaired consciousness. These symptoms may be accompanied by headache, nausea and vomiting, seizures, neck stiffness, and so on. The diagnosis of stroke, like other diagnoses in the field of Neurology, includes clinical, topographical, and etiological diagnosis.

Clinical Diagnosis

The clinical diagnosis of stroke is established from a neurological physical examination, where symptoms consistent with the disease progression and signs corresponding to the affected area of cerebral blood vessels are found. Disorders of the carotid system lead to: visual disturbances, speech disorders, dysphasia or aphasia if the dominant cerebral hemisphere is affected, motor disturbances, contralateral hemiplegia/hemiparesis, and sensory disturbances. Disorders of the vertebrobasilar system lead to: visual disturbances, blurred vision or blindness if the occipital lobe is affected, cranial nerve disorders if the brainstem is involved, motor disturbances, coordination problems, drop attacks, sensory disturbances, consciousness disturbances, and combinations of these.

Topical Diagnosis

According to Bamford's classification, the diagnosis of stroke can be divided into:

Total Anterior Circulation Infarct (TACI) if you meet 3 of the symptoms below:

- Hemiparesis with or without sensory disturbances (contralateral to the lesion)
- Contralateral hemianopia
- Higher function disorders: dysphasia, visuospatial deficits, hemineglect, agnosia, apraxia

Partial Anterior Circulation Infarct (PACI) if it meets 2 of the symptoms below or just 1, but it must be a high-level functional disorder:

- Hemiparesis with or without sensory disturbances (contralateral to the lesion)
- Contralateral hemianopia
- Higher function disorders: dysphasia, visuospatial deficits, hemineglect, agnosia,
  apraxia

Lacunar Circulation Infarct (LACI) if:

- Pure motor disorders
- Pure sensory disorders
- Hemiparesis with ataxia

Posterior Circulation Infarct (POCI) if it produces symptoms:

- Double vision
- Difficulty swallowing
- Dizziness

Etiological Diagnosis

The etiological diagnosis of stroke is divided into two types: ischemic stroke and hemorrhagic stroke. The gold standard used to determine the etiology is a head CT scan. Supportive examinations such as laboratory tests (blood and urine), electrocardiogram, echocardiogram, chest X-ray, lumbar puncture, electroencephalogram, arteriography, and Doppler sonography are required to assist in the etiological diagnosis of hemorrhagic stroke (intracerebral, subarachnoid) or ischemic stroke (embolism, thrombosis) and to identify risk factors.

Physical Examination

An important component of the physical examination includes the assessment of vital signs (blood pressure, pulse rate, respiratory rate, and temperature) as well as a neurological examination, which encompasses the evaluation of mental status and level of consciousness, cranial nerve assessment, motor and sensory function, physiological reflexes, and pathological reflexes.

Supportive Examinations

CT scan (Computer Tomografi Scan)

This modality can accurately and quickly differentiate between hemorrhagic stroke and non-hemorrhagic stroke because patients with non-hemorrhagic stroke require the administration of thrombolytics as soon as possible. In addition, this examination is also useful for determining the

anatomical distribution of the stroke and ruling out differential diagnoses of stroke (hematoma, neoplasm, abscess).

MRA (Magnetik Resonan Angiografi)

MRA has also been proven to be able to identify vascular lesions and occlusions earlier in acute stroke. Unfortunately, this examination and other MRI tests are quite expensive and require a relatively long examination time.

Cerebral angiography

Cerebral angiography can help determine the specific cause of a stroke, such as bleeding or arterial obstruction.

Chest X-Ray Examination

A chest X-ray examination can show the condition of the heart, whether there is enlargement of the left ventricle, which is one of the signs of chronic hypertension in stroke patients.

Electrocardiography

This examination can be used to check for the presence or absence of myocardial infarction, arrhythmia, or atrial fibrillation, which can be risk factors for stroke.

Echocardiography

This examination can be used to determine the presence or absence of heart abnormalities that may cause embolic stroke.

USG

Ultrasound can be used to detect the presence of stenosis or occlusion in the internal carotid artery.

Blood Test

Blood tests that can be done include a complete blood count, cholesterol test, and blood sugar test.

Management

Therapy for ischemic stroke is divided into the acute phase and the post-acute phase.:

Acute Phase (day 0 – 14 after disease onset)

The goal of treatment at this stage is to save neurons that are suffering from dying and to prevent other accompanying pathological processes from disturbing or threatening brain function. The actions and medications given must ensure adequate blood perfusion to the brain. Therefore, optimal function is maintained:

- Respiration: the airway must be clear and unobstructed
  Heart: must function properly; monitor ECG if necessary
  Blood pressure: should be maintained at an optimal level; monitored to ensure it does not reduce brain perfusion
- Blood sugar: high blood sugar in the acute phase should not be lowered drastically, especially if the patient has chronic diabetes mellitus
  Fluid balance: if the patient is critical or comatose, fluid, electrolyte, and blood acid-base balance must be monitored

The use of drugs to restore blood flow and metabolism in the brain affected in the ischemic area (ischemic penumbra) still generates differing opinions.

Prognosis

One-third of patients with cerebral infarction will experience a deterioration of their neurological status after treatment. This is partly due to brain edema and maturation of cerebral ischemia. Large infarctions causing hemiplegia and decreased consciousness occur in 30-40% of cases. About 10% of patients with ischemic stroke recover with normal function. The prognosis is worse in patients with congestive heart failure and coronary heart disease.

METHODS

Problem Solving Methods

RESULTS AND DISCUSSION

The patient was brought by family to the emergency room of RSUD Cut Meutia with complaints of weakness in the right limbs since 2 days before hospitalization. The family stated that the complaint appeared suddenly when the patient woke up in the morning. The family said that the weakness in the right hand is equally as severe as the weakness in the right leg, making it difficult for the patient to sit and walk. Weakness in the limbs occurs continuously and is not influenced by activity. The complaints are also accompanied by headache, nausea, vomiting, and numbness in the right hand and foot. The Siriraj stroke score is -2 (non-hemorrhagic stroke). A head CT scan examination showed an infarction in the left parietal and frontal lobes, indicative of thrombotic ischemic stroke.

According to the WHO, stroke is defined as rapidly developing clinical signs due to focal or global brain dysfunction, with symptoms lasting 24 hours or more, which can cause death, without any other cause besides vascular. Ischemic stroke is divided into two major categories, namely stroke caused by cerebral thrombosis and stroke caused by cerebral embolism. Non-hemorrhagic stroke due to a thrombus occurs because of reduced blood flow in a specific area of the brain through a stenosis process. A thrombus is the formation of a clot of platelets or fibrin in the blood, which can block veins or arteries and cause ischemia and local tissue necrosis. Thrombosis is the result of changes in one or more of the main components of hemostasis, which include coagulation factors, plasma proteins, blood flow, vessel surfaces, and cellular constituents, particularly platelets and endothelial cells. Arterial thrombosis is a complication of atherosclerosis that occurs due to the rupture of atherosclerotic plaques. The most common site of thrombosis is at arterial branching points, especially in the distribution area of the internal carotid artery. Arterial stenosis can lead to turbulent blood flow. The energy required for neuronal activity comes from glucose metabolism and is stored in the brain in the form of glucose or glycogen for supply usage for about 1 minute. If there is no blood flow for more than 30 seconds, the EEG pattern will flatten; if more than 2 minutes, brain tissue activity stops; if more than 5 minutes, brain tissue damage begins; and if more than 9 minutes, a person can die. When blood flow to brain tissue stops, the oxygen and glucose required for ATP production decrease, leading to a reduction in Na+ K+ ATPase, and consequently, the membrane potential will drop. K+ moves into the

extracellular space, while Na and Ca ions accumulate inside the cell. This causes the cell surface to become more negative, resulting in membrane depolarization. At the onset of depolarization, the cell membrane is still reversible, but if it persists, structural changes occur, causing brain tissue death. This condition occurs immediately if perfusion drops below the threshold for tissue death, that is, when blood flow decreases to below 10 ml / 100 grams / minute. As a result of oxygen deficiency, acidosis occurs, which causes enzyme function disorders due to the high levels of H ions. Furthermore, acidosis leads to cerebral edema, characterized by cell swelling, especially in glial tissue, and affects microcirculation. Consequently, there is an increase in vascular resistance and then a decrease in perfusion pressure, resulting in the expansion of ischemic areas (14). The patient received Asering intravenous fluid therapy aimed at meeting the patient's fluid needs and serving as a medium for administering injectable medication. The patient also received Inj Citicoline 500 mg every 12 hours, Inj Omeprazole 40 mg every 12 hours, Inj Ondansetron 2 mg every 12 hours, Drip PCT every 12 hours, Drip Sohobion/H, and Amlodipine 5 mg once daily.

Citicoline is a neuroprotective drug. Neuroprotective drugs are one of the therapies aimed at reducing cell damage due to the disruption of blood flow that delivers oxygen.

Amlodipine is an antihypertensive drug belonging to the calcium channel blocker class, recommended by PERDOSSI in the management of hypertension in stroke patients. Amlodipine is known to provide good protective effects for stroke patients by inhibiting calcium influx, leading to muscle relaxation. In addition, this drug is an effective therapeutic agent in lowering systolic and diastolic blood pressure compared to other antihypertensive drugs in stroke patients (16).

Omeprazole is a drug in the proton pump inhibitor class that functions to inhibit the production of HCL or stomach acid. This drug is used to prevent the occurrence of stress ulcers in the gastrointestinal tract as a side effect of using antiplatelet drugs and non-steroidal drugs (16).

Sohobion is a neurotropic vitamin supplement designed to maintain nerve health and relieve tingling, cramps, and numbness caused by a deficiency of Vitamins B1, B6, and B12. The use of vitamins in stroke patients is related to the levels of homocysteine in the blood. Homocysteine is one of the risk factors for stroke, and when its levels are high in the blood, the risk of stroke increases. High homocysteine levels have atherogenic and prothrombotic properties. Blood homocysteine levels are determined by the essential amino acid methionine, which is influenced by genetic factors, vitamin intake, and folic acid. Consuming vitamins and folic acid can help reduce homocysteine levels in the blood (17).

CONCLUSIONS AND RECOMMENDATIONS

A case has been reported under the name Mr. I, who came to the ER at RSUD Cut Meutia with complaints of weakness in the right limbs for the past 2 days. The patient's family stated that the complaint appeared suddenly when the patient woke up in the morning. The family mentioned that the weakness in the right arm was as severe as the weakness in the right leg, making it difficult to sit and walk. This limb weakness has been continuous and is not affected by activity. The complaints are also accompanied by headache, nausea, vomiting, and numbness in the right hand and foot. Based on the examination, the findings were GCS E4M6V5, blood pressure 140/90 mmHg, pulse 83/min, respiration 21/min, temperature 36.5°C, and SpO2 95%. Neurological status examinations, cranial nerves, sensory, and autonomic tests that could be performed showed normal results, while the motor examination revealed weakness in the right limbs with a Siriraj score of -2.

Based on the anamnesis, physical examination, and supporting examinations, the patient was diagnosed with ischemic stroke. The patient received both pharmacological and non-pharmacological therapy aimed at reducing the severity of the disease and accelerating the patient's recovery process. The prognosis for this patient, based on quo ad vitam, functionam, and sanationam, is likely to be recovered/good (dubia ad bonam).

FURTHER STUDY

Further studies are needed to evaluate ischemic stroke management using a larger number of patients to strengthen clinical evidence beyond single case reports. Future research should focus on comparing the effectiveness of neuroprotective therapy, antihypertensive management, and supportive treatment in improving neurological outcomes and functional recovery. Longitudinal studies are also recommended to assess long-term prognosis, recurrence rates, and quality of life in ischemic stroke patients, as well as the role of early rehabilitation and risk factor control in preventing disease progression.

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