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Successful Management of Ischemic Stroke: A Case Report and Literature Review

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ABSTRACT

Introduction: Stroke is a clinical condition characterized by the sudden onset of focal (or global, in cases of coma) neurological symptoms that persist for over 24 hours or result in death, with no clear cause other than a vascular issue.

Case Description: Male, 62 years old, with chief of complaint of mouth dropped to the right since 1 day before admission. Complaints made the patient speak more difficult, slow and unable to form sentences. Physical examination show parese CN. VII UMN dextra. Siriraj score -5 and Head NCCT show cerebral infarction in the internal capsula interna. Patient was diagnosed with ischemic stroke.

Discussion: The use of a scoring system can be useful when there are no brain imaging facilities that can clearly distinguish the causative pathology of stroke. criteria for diagnosis of ischemic stroke are symptoms of global neurological deficits or one / several focal neurological deficits that occur suddenly with evidence of brain imaging images (CT scan or MRI). The most common differential diagnosis is hemorrhagic stroke.

Conclusion. Early recognition and appropriate treatment is important to cure acute ischemic stroke to achieve good quality of life and prevent further infarct to brain tissue.

Keyword: Ischemic Stroke, Management, Case Report

1. Introduction

Stroke, as defined by the World Health Organization, is a clinical condition characterized by the sudden onset of focal (or global, in cases of coma) neurological symptoms that persist for over 24 hours or result in death, with no clear cause other than a vascular issue. Strokes are generally categorized into three main types: ischemic, hemorrhagic, and subarachnoid hemorrhage. Ischemic stroke occurs when a blood vessel is blocked, restricting blood flow to the brain, while hemorrhagic stroke results from a ruptured blood vessel that leads to bleeding within the intracranial space. Depending on where the bleeding occurs, hemorrhagic strokes can be further classified as intracerebral or subarachnoid hemorrhages. Ischemic strokes account for approximately 60–80% of all stroke cases. This article focuses on acute ischemic strokes and their treatment. In patients with stroke, around 30% of sufferers experience permanent disability and become dependent on other

people. A person's risk of stroke increases with age and the risk doubles every 10 years after a person is over 55.1 years old and stroke is often accompanied by morbidity related to the patient's age. In this case report, we report a successful patients management of a 62-year-old male patient with ischemic stroke.[1]

Among stroke patients, around 30% suffer from permanent disabilities that make them reliant on others for assistance. The likelihood of experiencing a stroke increases with age, doubling every decade after an individual turns 55 years old, and the morbidity associated with stroke often correlates with the patient's age. This case report discusses the successful management of a 62-year-old male patient who experienced an ischemic stroke.[2]

2. Case Description

Male, 62 years old, with chief of complaint of mouth dropped to the right since 1 day before admission. Complaints made the patient speak more difficult, slow and unable to form sentences. The complaint occurred suddenly when the patient woke up. The patient was a smoker and has a history of uncontrolled hypertension. On physical examination, the patient was found to have a blood pressure of 180/100 mmHg. General status showed no abnormalities. Neurological examination showed parese N. VII UMN dextra with asymmetry of the patient's right lip, leaning to the right, and deviation of the tongue to the right when protruded. Motor examination is within normal limits. On the Siriraj score, a score of -5 was obtained with the interpretation of ischemic stroke and the Gajah Mada score showed no loss of consciousness, headache, and Babinski reflexes with the interpretation of ischemic stroke. The patient was given a Head NCCT show a subacute cerebral infarction in the internal capsule sinistra (Figure 1). The patient was treated with citicoline, candesartan, and loading acetylsalicylic acid of 320 mg for 2 days, followed by 80 mg daily. improvement in language function where the patient could return to speaking and forming sentences.

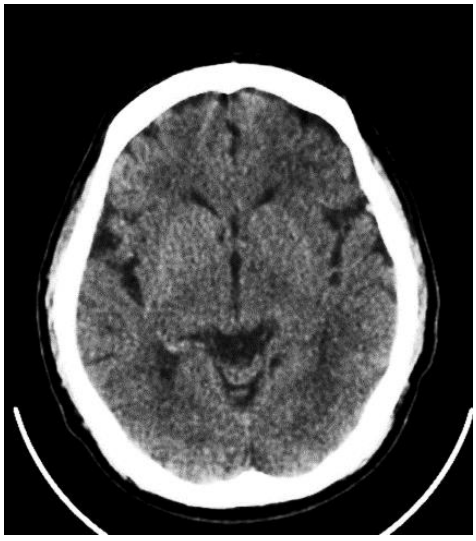

Figure 1. Head NCCT show subacute cerebral infarction in the capsula interna

3. Discussion

Stroke risk factors are divided into modifiable risk factors, including hypertension, diabetes mellitus (DM), smoking, obesity, gout, and hypercholesterolemia, and non-modifiable risk factors, such as age, gender, and ethnicity. The pathophysiology of hypertension causes changes in the blood vessels. The changes start from thickening of the tunica intima and increased endothelial permeability

by long-standing hypertension, especially in arteries with small size, which is about 300-500 μm (perforate branches). Hardening of the vessel wall may result in impaired autoregulation, in the form of difficulty to contract or dilate to changes in systemic blood pressure. If there is a sudden drop in systemic blood pressure, brain perfusion pressure becomes inadequate, leading to brain tissue ischemia.[3]

Ischemic stroke is initiated by a thrombus or embolism that results in brain cells experiencing metabolic disturbances, because they are not supplied with blood, oxygen, and energy. Emboli are caused by obstruction of blood vessels from more proximal sites. A thrombus is formed by the process of atherosclerosis in the aortic arch, carotid artery, or cerebral blood vessels. This process is initiated by endothelial injury and inflammation which results in the formation of plaque on the blood vessel wall. The plaque will progressively become thicker and sclerotic. Platelets will then adhere to the plaque and release factors that initiate the coagulation cascade and thrombus formation. Symptoms usually worsen immediately or only briefly and then disappear again as soon as the embolism is dislodged distally, as in TIA.[4]

Surrounding the infarcted brain cell area is usually only a temporary metabolic and perfusion disorder called the penumbra region. This area can still be salvaged if immediate blood flow restoration (reperfusion) is performed, thus preventing more extensive cell damage, which means preventing disability and death. However, if the penumbra cannot be saved, it will become an infarcted area. Infarction is not only caused by obstruction, but also by inflammation, blood brain barrier (BBB) disruption, neurotoxic substances due to hypoxia, decreased collateral microcirculation blood flow, and reperfusion management.[5]

A simple screening to recognize the symptoms and signs of stroke can be abbreviated to FAST, which includes facial drop, arm weakness, speech difficulties, and time to seek medical help. FAST has a sensitivity of 85% and specificity of 68% to establish stroke, as well as good reliability in doctors and paramedics. The main physical examination includes level of consciousness with the Glasgow coma scale, cranial nerve weakness, motor weakness, sensory deficits, autonomic disorders, and impaired cognitive function.[5]

The use of a scoring system can be useful when there are no brain imaging facilities that can clearly distinguish the causative pathology of stroke. However, the score system cannot be confirmed in the stroke pathology that occurs. This is because the clinical manifestations in hemorrhagic stroke with a small volume of bleeding can resemble ischemic stroke. Similarly, the clinical manifestations of extensive ischemic stroke with increased ICP are similar to hemorrhagic stroke. The Siriraj stroke score is a scoring system that is often used to distinguish ischemic or hemorrhagic stroke (Table 1). The system calculates with the formula: [(2.5 x consciousness) + (2 x vomitus) + (2x headache) + (0.1 x diastolic pressure) - (3 x atheroma) - 12] with interpretation if the total score < 1 is ischemic stroke, if the score > 1 is intracerebral hemorrhage, and if the score 0 is considered doubtful.[6]

The criteria for diagnosis of ischemic stroke are symptoms of global neurological deficits or one / several focal neurological deficits that occur suddenly with evidence of brain imaging images (CT scan or MRI). The most common differential diagnosis is hemorrhagic stroke (if brain CT/MRI has not been performed). Brain imaging using CT scan is the gold standard in the diagnosis of hemorrhagic stroke. CT scan is superior in detecting direct hemorrhage based on hypodensity images in the brain parenchyma compared to MRI which requires comparison of multiple image sequences.[7]

General management includes airway stabilization with continuous monitoring of vital signs in the first 72 hours. Intubation with ETT or laryngeal mask airway is indicated in hypoxic patients (pO_2 < 60 mmHg or pCO_2 > 50 mmHg), stroke, or aspiration patients. Hemodynamic stabilization

with IV crystalloid or colloid is performed. If systolic BP is below 120 mmHg and fluids are adequate, vasopressor agents can be titrated, such as medium/high doses of dopamine, norepinephrine or epinephrine with a target systolic BP of 140 mmHg. Cardiac monitoring during the first 24 hours after the onset of ischemic stroke. Intracranial pressure control is carried out with attention to worsening symptoms. Intracranial pressure monitoring is performed in patients with GCS < 9 and patients with decreased consciousness with a target intracranial pressure < 20 mmHg and brain perfusion pressure > 70 mmHg. If necessary, it can be done to maintain normoventilation (pCO2 35-50 mmHg). Seizure control was performed by administering a slow bolus of diazepam 5-20 mg followed by phenytoin bolus 15-20 mg/kg. Administration of rTPA such as alteplase is given in acute ischemic stroke with onset <6 hours intravenously according to protocol at a dose of 0.6-0.9 mg/kg.[8]

4. Conclusion

Early recognition and appropriate treatment is important to cure acute ischemic stroke to achieve good Quality of Live and prevent further infarct to brain tissue.

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