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TRAUMATIC SUBARACHNOID HEMORRHAGE AND CEREBRAL EDEMA FOLLOWING FOCAL BRAIN INJURY: A CASE STUDY IN A POST-TRAFFIC ACCIDENT PATIENT

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Background: Traumatic Brain Injury (TBI) is defined as neurological dysfunction, both temporary and permanent, nondegenerative and non-congenital, caused by mechanical trauma to brain tissue, either directly or indirectly. Effective and efficient diagnosis and appropriate therapeutic approaches in cases with TBI is important so that morbidity and mortality due to head trauma can be prevented. This case report on focal brain injury aims to document and analyze the clinical manifestation, management, and outcomes of this case. **Case Presentation:** A 42-year-old female patient came to the emergency room unconscious after being hit by a car. During treatment, the patient only complained of headache, nausea, and vomiting, and no abnormalities were found on neurological examination. A non-contrast head CT scan showed subarachnoid hemorrhage and cerebral edema. The treatment given was the injection of tranexamic acid, mannitol, and nimodipine, as well as symptomatic drugs such as metamizole, lansoprazole, diphenhydramine, and ondansetron. **Conclusion:** Traumatic brain injury is the main cause of subarachnoid hemorrhage (SAH), a vascular disease of the brain that causes blood to enter the subarachnoid space. The goal of treatment is to repair the bleeding source and prevent morbidity and mortality.

Keywords: Focal brain injury, Subarachnoid Hemorrhage, Cerebral edema

INTRODUCTION

Traffic Accidents (TAC) are one of the main causes of morbidity and mortality in the world, especially in the productive age group. According to data from the World Health Organization (WHO), every year around 1.3 million people die from traffic accidents and around 20-50 million others experience non-fatal injuries, many of which cause long-term disability, where Traumatic Brain Injury (TBI) is the main cause of high morbidity in TAC cases. One of the serious clinical manifestations of trauma due to TAC is head trauma, which can cause brain injury, both focal and diffuse. Focal brain injury refers to local damage to brain tissue such as cerebral contusion, epidural/subdural hematoma, and brain laceration. (Maurya, 2022)

Inappropriate and inappropriate handling of head trauma, especially focal brain injury, can result in serious complications such as increased intracranial pressure, brain herniation, permanent neurological disorders such as motor and sensory disorders, aphasia, cognitive and emotional disorders, loss of social function in daily activities, and even death. Data from the Brain Trauma Foundation (BTF) shows that 90% of severe head trauma patients who are not treated within the first hour or golden hour time have a risk of death or long-term disability. The following describes a case of a subarachnoid hemorrhage due to trauma from a recent traffic accident and how the management is

carried out until the patient's condition improves

CASE REPORT

A 42-year-old female patient came to the emergency room of Regional Public Hospital Haji East Java (September 9, 2023, at 08.45 WIB) unconscious after being hit by a car from behind. While in the emergency room, the patient was conscious and said she did not remember the incident when she was hit. The patient wore a helmet while driving, but did not remember whether the helmet came off or not during the incident. The patient felt heavy dizziness accompanied by body weakness. There was a laceration on the right temple and abrasions on both hands. The patient was unconscious after the incident and had vomited twice in the emergency room. There was no double vision and ringing in the right ear with blood coming out. The patient denied any sensation of numbness and there were no seizures.

The primary survey obtained stable airway, breathing with RR 22 times/minute and SpO2 98% and symmetrical chest movements, circulation obtained blood pressure 147/78 mmHg with a heart rate of 99 times/minute irregularly accompanied by warm dry red acral on all extremities accompanied by CRT < 2 seconds, disability obtained GCS 4/5 with isocoric pupils 3 mm in both eyes accompanied by positive pupillary reflex, exposure obtained an open wound on the left parietal region and excoriation injuries on both hands. Based on the AMPLE examination, the patient was found to have no drug or

food allergies, no history of using certain drugs, and did not remember the last time she ate. In the neurological status examination, the motor examination results were 555/555, and sensory was within normal limits. Physiological reflexes were within normal limits and pathological reflexes were negative. No abnormalities were found in the cranial nerve examination.

Axial head CT scan examination without contrast found no fracture in the skull and the calvaria is still intact (Figure 1). While the MSCT examination of the axial head and axial bone/SPN corona without contrast showed abnormal hyperdense lesions in the right temporoparietal and left temporal subarachnoid space, narrowed sulcus and gyrus, no midline deviation, narrowed ventricular system structure. Pons, midbrain, and cerebellum were normal, calvaria intact, and soft tissue swelling (Figure 2). Based on the examination, the diagnosis of this patient is Focal Brain Injury with traumatic subarachnoid haemorrhage and cerebral edema.

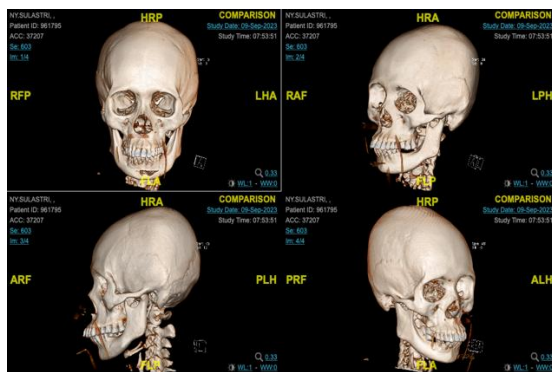


Figure 1. Axial head CT Scan Without Contrast. CT Scan result no fracture image was found and calvaria intact

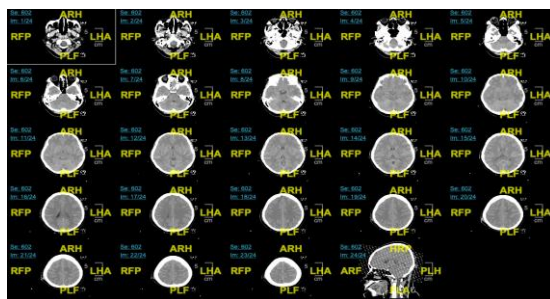


Figure 2. MSCT Skull Axial and Axial Bone/SPN Corona With Contrast. There were abnormal hyperdense lesions in the right temporoparietal and left temporal sub-arachnoid space with narrowed sulcus and gyrus without midline deviation, narrowed ventricular system structure, and soft tissue swelling. The conclusion from the examination results obtained Focal Brain Injury with traumatic subarachnoid haemorrhage and Cerebral Edema

The patient was then transferred to the room for further observation. Management given to the

patient was NaCl 0,9% infusion 1000 mL/24 hours, metamizole injection 3x1 gram, diphenhydramine injection 3x10 mg, ondansetron injection 3x8 mg, lansoprazole injection 2x30 mg, tranexamic acid injection 3x500 mg, mannitol 4x100 mL, nimodipine 4x30 mg, and active mobilization with semi fowler position. While in the medical ward, the patient was monitored for signs of increased ICP (Intracranial Pressure), respiratory status, fluid input and output, and level of consciousness. The patient was not planned to be monitored with a CT scan due to minimal bleeding, so only continued observation was carried out.

On the second day of treatment (September 10, 2023, at 2:07 pm), the patient felt dizzy and had persistent headache accompanied by nausea and vomiting. The examination results showed that the patient's consciousness was apathetic and blood pressure was 121/75 mmHg, HR 75 times/minute, RR 18 times/minute, Temperature 36.2 C, SpO2 98% without additional O2, CRT < 2 seconds with warm acral perfusion. Examination of neurological status obtained motor examination results 555/555 and sensory within normal limits. There was no increase in physiological reflexes and negative pathological reflexes. The patient's therapy was still the same as before and an additional peroral betahistine 3x6 mg was given because there were still complaints of dizziness.

On the third day of treatment (September 11, 2023, at 15.19 WIB), the patient's complaints are still feeling dizzy, headache, nausea, and vomiting. Consciousness compos mentis with the results of vital signs examination blood pressure 105/65 mmHg, HR 62 times/min, RR 20 times/min, SpO 98% without additional O2, Temperature 36 C, CRT < 2 seconds accompanied by warm acral perfusion. Neurological status examination obtained the same results, namely motor 555/555 and sensory within normal limits. There was no increase in physiological reflexes and negative pathological reflexes. Tranexamic acid injection therapy was stopped on this day and mannitol was planned for tapering off to 3x100. Metamizole injection was stopped and replaced with ibuprofen injection 3x800 mg.

On the fourth day of treatment (September 12, 2023, at 16:12 WIB), the patient felt that complaints of dizziness were more dominant than headache nausea, and vomiting. The patient's consciousness was composmentis with the results of vital signs examination of blood pressure 116/67 mmHg, HR 68 times/minute, RR 20 times/minute, SpO 98% without additional O2, Temperature 36.7 C, CRT < 2 seconds accompanied by warm acral perfusion. Examination of neurological status obtained motor examination results 555/555 and sensory within normal limits. Physiological and pathological reflexes were not found to be abnormal. The patient's therapy for mannitol was reduced to 2x100 mg and added gabaxa (L-Glutamine) injection 1x1 mg, and ondansetron injection to 3x4 mg.

On the fifth day of treatment (September 13,

2023, at 15.27 WIB), complaints of dizziness from the patient began to decrease as well as headache, nausea, and vomiting. The patient's consciousness was composmentis with the results of vital signs examination of blood pressure 126/77 mmHg, HR 65 times/minute, RR 20 times/minute, SpO₂ 98% without additional O₂, Temperature 36.4 C, CRT < 2 seconds accompanied by warm acral perfusion. Examination of neurological status obtained motor examination results 555/555 and sensory within normal limits. Physiological and pathological reflexes were not found to be abnormal. Mannitol therapy was reduced to 1x100 mg and planned to be stopped the next day.

On the sixth day of treatment (September 14, 2023, at 3:47 pm), the patient felt better and there were no significant complaints. The patient's consciousness was composmentis with the results of vital signs examination of blood pressure 125/85 mmHg, HR 65 times/minute, RR 18 times/minute, SpO₂ 98% without additional O₂, Temperature 36.2 C, CRT < 2 seconds accompanied by warm acral perfusion. Examination of neurological status obtained motor examination results 555/555 and sensory within normal limits. Physiological and pathological reflexes were not found to be abnormal. The patient was given analgesic 3x500 mg, lansoprazole 2x30 mg, and betahistine 3x6 mg. The patient was planned to go home due to improved clinical condition.

DISCUSSIONS

The patient initially did not complain of severe headache due to a period of post-traumatic retrograde amnesia. Headache, dizziness, nausea, and vomiting developed a few hours later were in line with meningeal irritation from local blood and progressive ICP elevation due to mild edema (Maher et al., 2022).

The GCS remained 15 since baseline suggesting that intracranial pressure did not surge massively as in aneurysm rupture and there was likely only a tear in the capillary or superficial vein in the temporoparietal area so that bleeding was only localized and did not lead to widespread blood distribution (Lazier et al, 2023). In patients with traumatic brain injury (TBI) and subarachnoid hemorrhage (SAH), factors like age, GCS score, presence of traumatic SAH, and other injuries significantly impact outcomes, with certain conditions exacerbating the injury and increasing mortality risk (Fitri et al., 2021) Older age (especially >65) is associated with a higher risk of mortality. Pre-existing conditions like hypertension, diabetes, and other medical comorbidities can negatively impact TBI outcomes (Laing et al., 2022) This patient did not have any of these risk factors.

The patient was not subjected to surgery, but only observation because the speed of action greatly affects the worsening of symptoms (such as

pharmacological management described in the next paragraph). Periodic CT scan evaluation is not necessary because it is adjusted to the patient's condition, and it is said that it is less useful in cases with minimal bleeding. In this case, there is only minimal bleeding so there is no need for routine CT Scan imaging examinations (Griswold et al, 2022).

In this case, the patient was given Nimodipine, which is a calcium channel blocker, which has a significant effect on SAH, which is treated with nimodipine. These drugs mainly affect cerebral vascular smooth muscle cells, which help relax and dilate blood vessels, improve blood flow, and reduce the risk of SAH complications. These drugs are also recognized to significantly improve patient prognosis. As a result, it is imperative to create innovative treatment approaches for SAH that focus on factors associated with early brain injury (Li et al., 2023).

In addition, the administration of tranexamic acid in patients with head injury resulted in a significant reduction in mortality, bleeding growth, and bleeding volume growth (JX et al., 2024).

The uses of mannitol in these patients include reducing intracranial pressure because mannitol is a hyperosmolar solution that draws fluid from brain tissue into the bloodstream, potentially lowering ICP. Current evidence suggests that mannitol is more effective in lowering ICP in TBI patients compared to barbiturates and is recommended by guidelines. (Syahrul et al., 2024)

The semi-fowler position, 30-degree head-up position, is a position to raise the head of the bed at an angle of about 30 degrees. The body is in a parallel position, so it can reduce intracranial pressure and increase oxygen to the brain. Providing this intervention can lower intracranial pressure, thereby affecting cerebrovascular dynamics, which can meet the oxygen demand in the brain. An upturned head position can increase blood flow in the brain to maximize brain oxygenation. Increased oxygen in the brain can increase metabolism, which is characterized by increased consciousness and other vital signs (Basuki et al., 2024).

During the subsequent early stages, stabilization of patients with SAH is required through the use of extensive vasodilation strategies, optimization of cerebral blood flow and metabolism, improved anti-inflammatory response, reduction of oxidative stress, inhibition of platelet aggregation, and other relevant interventions. This multifaceted method aims to stop several important mechanisms of brain injury. This allows for better neurovascular protection and ultimately affects patient prognosis. (Li et al., 2023)

In this patient, there was no skull fracture after being involved in a traffic accident in this case, but there was subarachnoid hemorrhage (SAH) due to traumatic brain injury, raising interesting questions related to the mechanism of injury and also the prognosis of this patient was very good so that after being hospitalized for several days, he could be discharged due to the rapid and appropriate treatment so that there was minimal disability.

CONCLUSION

Traumatic brain injury is the leading cause of subarachnoid hemorrhage (SAH), which is a vascular disease of the brain that causes blood to enter the subarachnoid space, impairing brain perfusion and function with the main causes being road traffic accidents, falls, and violence. Brain injury begins with an increase in ICP due to extravasation of hemorrhagic blood into the subarachnoid space, ventricles, and parenchyma. This increase in ICP, combined with acute vasoconstriction and microthrombosis, disrupts cerebral perfusion pressure and cerebral blood flow, causing global ischemia. The disease presents with sudden, explosive, dramatic head pain lasting 1-2 seconds to 1 minute. Its diagnosis becomes difficult in patients who are neurologically intact and experience rapid headache pain. As a result, many patients have to undergo computed tomography (CT) to find SAH, but if the first CT does not show the right result, the next traditional step is lumbar puncture. Calcium channel blockers have a great effect on SAH, which is treated with nimodipine. Stabilization of patients with SAH is required through extensive use of vasodilation strategies, optimization of cerebral blood flow and metabolism, improved anti-inflammatory response, reduction of oxidative stress, inhibition of platelet aggregation, and other relevant interventions. The two main approaches to SAH are endovascular coiling and open surgical clipping. The choice of intervention is determined by several factors, such as patient complications and aneurysm anatomy, as well as the availability of surgical expertise. Patients with normal levels of consciousness have a lower risk of mortality, while patients with lower levels of consciousness have a higher risk of death and disability.

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