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Post-Operative Management for Craniotomy Decompression in Cerebral Edema Patients Due to Cerebral Infarction in The Right Carotic System which Occurred Cerebral Vasospasm

Radian Ahmad Halimi[1*], Ardi Zulfariansyah[2]

[1] *Intensive Care Trainee, Faculty of Medicine, Padjadjaran University / Hasan Sadikin General Hospital Bandung*
[2] *Intensive Care Consultant, Faculty of Medicine, Padjadjaran University / Hasan Sadikin General Hospital Bandung*

***Corresponding Author**: Radian Ahmad Halimi, E-mail: radianhalimi@gmail.com

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ABSTRACT

Introduction: Cerebral infarction accompanied by brain edema is a life-threatening condition and requires decompression craniectomy to overcome increased intracranial pressure (ICP). Comprehensive post-operative management using non-invasive transcranial doppler (TCD) monitoring and Near Infrared Spectroscopy (NIRS) is a modality for conducting management in the intensive room (ICU).
Case Report: We reported the case of a 56-year-old man diagnosed with cerebral edema due to stroke infarction of the right carotid system accompanied by comorbidities of coronary heart disease with a heart ejection fraction of 31.6% performed craniectomy decompression. The patient was treated for 12 days in intensive care and then transferred to a semi-intensive room.
Conclusion: This case highlights the importance of using TCD and NIRS in the intensive care unit as guiding therapies in maintaining patient blood pressure, administering blood components, and early detection of complications such as cerebral vasospasm.

INTRODUCTION

Decompressive craniectomy is one of the surgical treatments for conditions of brain edema and for several pathophysiologies, including traumatic and non-traumatic brain injuries,[1] as well as to treat refractory increases in intracranial pressure caused by any cause.[2]

Management in the intensive care unit (ICU) after decompressive craniectomy is usually associated with comorbid coronary artery disease, which can cause cerebral infarction. Cerebral infarction accompanied by brain edema is a life-threatening condition characterized by an ischemic lesion volume of > 150 cm3 or > 50% of the area supplied by the middle cerebral artery (MCA) experiencing infarction. Malignant infarction in the MCA area has an incidence rate of up to 10% and a mortality rate of up to 80%.[1]

Decompressive craniectomy will interrupt the cascade of secondary brain injury (ischemic injury) due to extensive brain edema and increased ICP. Surgical intervention will reduce ICP, and cerebral blood flow will increase within 24 hours after the procedure. In the first week after decompressive craniectomy, brain swelling will occur, which will cause a loss of cerebral vascular resistance and an increase in transcapillary leakage due to the high transcapillary hydrostatic pressure gradient.[3]

The recommended ICU management that provides good outcomes is based on the ICP value. The gold standard for ICP examination is using invasive ICP monitoring. However, this examination has many risks and complications, including bleeding, iatrogenic infections, and expensive costs, and must be installed in a

room that has good sterility.[4] Currently, non-invasive ICP examination techniques are being developed using ultrasound tools, including TCD and Optic Nerve Sheath Diameter (ONSD). The Pulsatility Index (PI) value obtained using the TCD tool and the ONSD diameter value have a good correlation with the increase in ICP, therefore this examination can be used as an alternative ICP monitoring for carrying out management in the ICU.[5]

Examination of brain oxygen saturation (rSO2) using Near InfraRed Spectroscopy (NIRS) is a non-invasive examination useful for monitoring cerebral hemodynamics. This tool is a bedside, non-invasive, continuous, and real-time examination to assess oxygenation in the brain in patients with ischemic stroke/infarction (cerebral blood vessel occlusion). The function of this examination is for early detection and prevention of cortical desaturation, which can cause mortality and morbidity, such as permanent disability. Until now, there has not been much research related to the use of NIRS.[6] Here, we report a case related to the post-operative management of decompressive craniotomy in a patient with cerebral edema due to stroke infarction in the right carotid system, which resulted in cerebral vasospasm.

CASE

A 56-year-old man with the main complaint of decreased consciousness accompanied by weakness in the left limbs, slanted mouth, and slurred speech since 2 days before admission to the hospital. Five days earlier, the patient had undergone PCI, and it was discovered that he had coronary heart disease involving 3 blood vessels (CAD 3VD). On physical examination, the patient obtained GCS 13 (E3M5V5), blood pressure 155/95 mmHg, pulse: 68 x/m, SpO_2: 95% with nasal cannula 3 lpm, respiratory rate: 28 x/min and temperature 36.3ºC. Fundoscopy showed papilla edema dextra. Motor examination revealed left hemiparesis 5|2 5|2 and edema in the lower extremities. Physiological reflex examination +/++, Babinski pathological reflex -/+, and NIHSS 11 scoring. Initial laboratory results showed hemoglobin (Hb) levels of 16.4 g/dL, hematocrit 49.2%, leukocytes 12,850/µL, urea 46.2 mg/dL, creatinine 1.31 U/L, sodium 141 mmol/L, Potassium 4.0 mmol/L, Magnesium 2.3 mmol/dL, Calcium 4.6 mmol/dL.

The chest x-ray results showed cardiomegaly accompanied by aortic atherosclerosis and no bronchopneumonia. Results of electrocardiography (ECG) examination, sinus rhythm, Left ventricular hypertrophy (LVH), PRWP ec LVH and old anteroseptal myocardial infarction (OMI), inferior ischemia. Echocardiography examination revealed dilatation in all cardiac chambers, decreased ejection fraction (LVEF 31.6%) with inferior akinetic and hypokinetic other areas, diastolic dysfunction, mild mitral regurgitation, and normal right ventricular contractility. The patient was then consulted to the Anesthesiology Department for decompressive craniectomy with a diagnosis of cerebral edema ec stroke infarction of the right carotid system accompanied by CAD 3VD, post-PCI (5 days before admission), Idiopathic Dilated Cardiomyopathy (IDCM), Chronic Heart Failure (CHF) Functional class II, Hypertension stage I, Stable COPD.

Intraoperative Management

Pre-induction TCD and ONSD examinations were carried out, resulting in a right pulsatility index (PI) of 1.4 and a left PI of 1.83. The right ONSD value was 6.3 mm, and the left ONSD was 1.3 mm. Intraoperative management was carried out by induction using dobutamine support 5 mcg/kg/minute and noradrenaline 0.05 mcg/kg/minute with an operation duration of 5 hours, intraoperative bleeding 2000 cc, intraoperative hemodynamics stable with the lowest blood pressure 70/56 mmHg after induction of anesthesia. During the operation, the dura mater was found to be intact, pale, and tense, then the brain appeared swollen and had minimal pulsation. The cranium was opened to 12x10 cm.

Treatment in ICU

On day one of treatment, PI-TCD results showed good results (<1.2), and NIRS results showed oxygenation of both parts of the brain was in good condition. The patient was then awakened by changing the propofol to dexmedetomidine, and the administration of mannitol was tapered off.

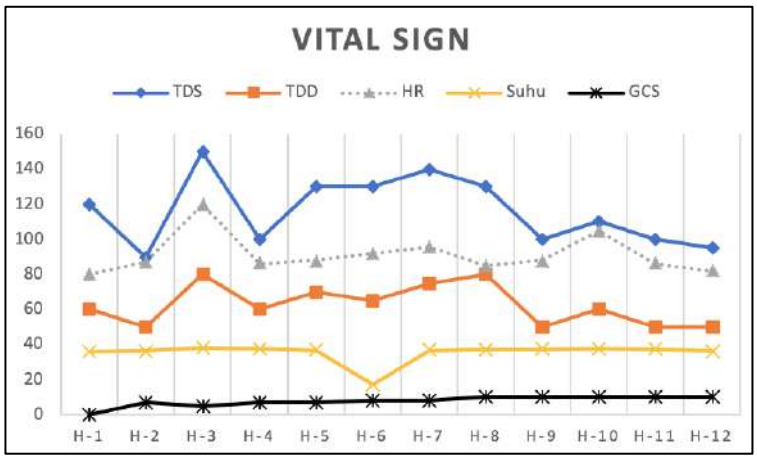


Figure 1. TDS, Systolic Blood Pressure; TDD, Diastolic Blood Pressure; HR, Heart Rate; GCS, Glasgow Coma Scale.

The second day of treatment showed a decrease in right brain oxygen saturation followed by an increase in PI to 1.4. The NE dose was then increased, and the patient was given a 1-unit PRC transfusion. The right brain rSO2 value increased again, although the left brain rSO2 decreased but was not significant (<20).

On the third day of treatment, the patient experienced a fever with a body temperature of 38.1 degrees Celsius. The patient was planned to have a repeat thorax x-ray examination and sputum culture examination due to greenish productive sputum, as well as an escalation of antibiotics to ceftazidime and levofloxacin.

On day 4th of treatment, there was a decrease in left brain rSO2 to below the critical value with a decrease in left brain mean flow velocity (mFv). This occurs when vasopressor weaning is performed, and blood pressure decreases to 100/60 mmHg. The patient was given vasopressor medication again and was planned for a 2-unit PRC transfusion with prior intravenous administration of 20 mg furosemide; then, the patient's rSO2 again increased to NIRS right brain to 70 and left brain to 59. The patient was planned to have a CT-scan or DSA examination to confirm whether there was a blockage. Left cerebral blood vessels and was given thromboembolic prophylaxis using subcutaneous heparin 2x5000 IU. The results of a chest x-ray examination revealed cardiomegaly with aortic atherosclerosis without pulmonary obstruction and no bronchopneumonia.

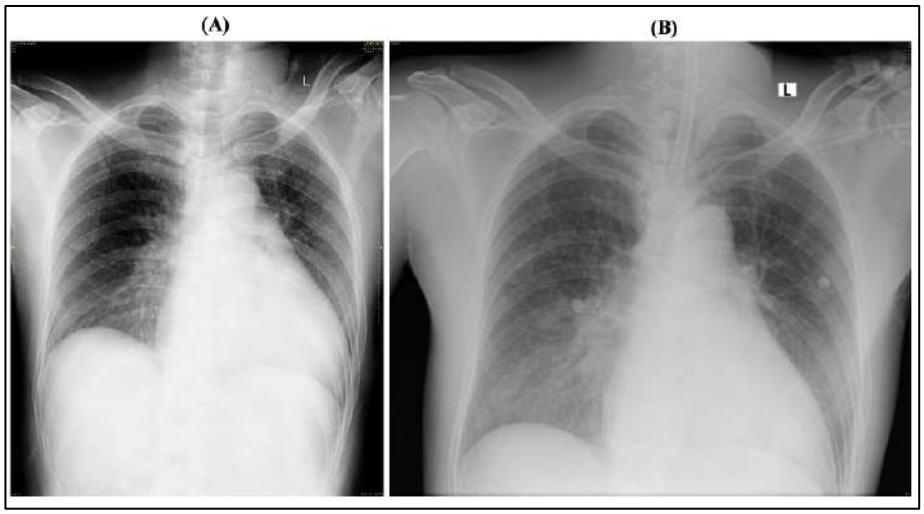


Figure 2. (A). Pre-operative chest x-ray; (B) Post-operative chest x-ray on day 4

From day 5th to day 7th of treatment, cardiology colleagues gave the patient anti-platelet aspirin and clopidogrel, and there was an increase in GCS to 8t (E3M5Vt) on the 6th day of treatment. On day 8th of

treatment, when a repeat TCD examination was carried out, it was found that mFv had increased to 146.7. The right internal carotid artery mFv was examined and the mFv value was 41.91, with a Lindergard ratio value of 3.5. Based on this, the patient was diagnosed with right MCA vasospasm and planned to be given nimodipine and undergo a repeat CT scan. The patient was also planned to be given 20% albumin because he had a hypoalbumin condition. Based on the results of the repeat CT scan examination showed an improvement compared to the results of the first CT scan.

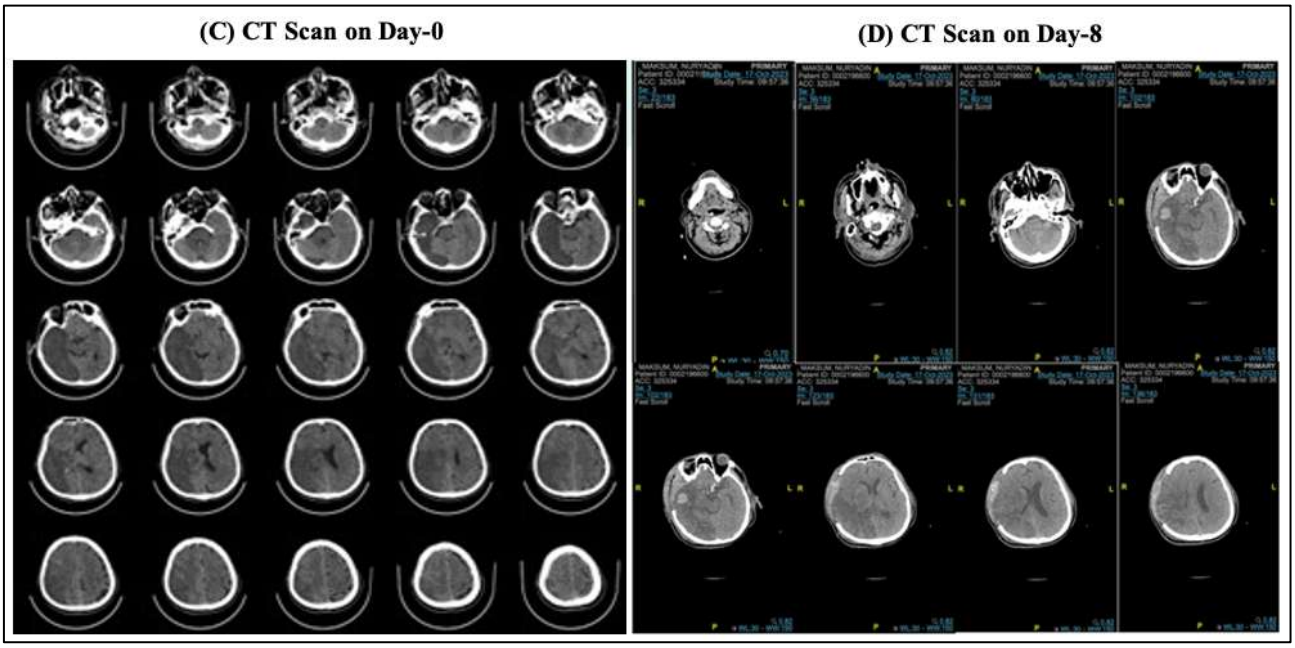
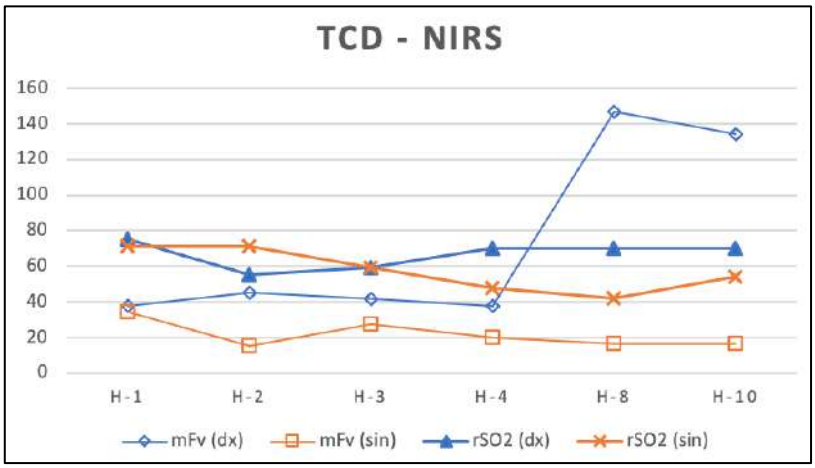


Figure 3 (C). Widespread infarction in the subcortical cortex of the right parietotemporooccipital lobe which compressed and narrowed the surrounding sulci and gyri, right sylvian fissure, third ventricle, and right lateral ventricle and caused a midline shift of 0.7 cm to the left, and there were no visible signs of intracranial hemorrhage; (D) There was an impression of intracerebral hemorrhage in the right parietal lobe, extensive infarction in the subcortical cortex of the right temporopaietooccipital lobe which compressed and narrowed the surrounding sulcus and gyrus, right sylvian fissure, 3rd ventricle and right lateral ventricle and caused a midline shift of 0.51 cm to left direction.



Figur 4. TCD, Transcranial Doppler; NIRS, Near InfraRed Spectroscopy; mFv (dx), mean flow velocity dextra; mFv (sin), mean flow velocity sinistra; rSO2 (dx), right brain oxygen saturation; rSO2 (sin), left brain oxygen saturation.

On day 9th of treatment, antibiotic de-escalation was carried out based on the culture results, which found Acinetobacter baumannii bacteria that were sensitive to the antibiotic amikacin, and vasopressor weaning was carried out therefore no vasopressor drugs were used and blood pressure was found to be good. On day10th of treatment, there was an improvement in his brain condition, which was marked by a decrease

in mFv of the right MCA and an increase in rSO2 of the left brain, but there was an increase in temperature accompanied by an increase in the number of leukocytes. On day 12th of treatment, the patient was planned to move to the high care room, and based on the TCD MCA examination of the right brain, the mFv value was found to be 72.5, with a PI value of 1.3, which indicated that there was improvement in the vasospasm condition.

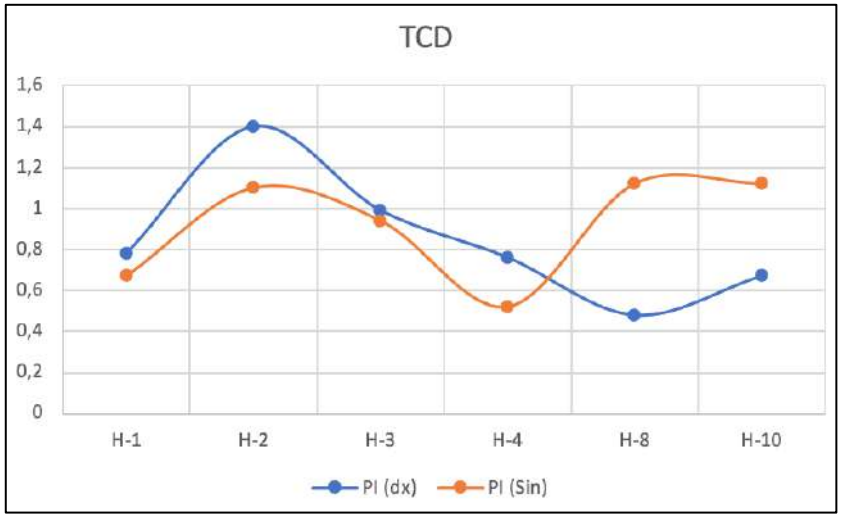


Figure 5. TCD, Transcranial Doppler; PI (dx), Pulsatility Index dextra; PI (sin), Pulsatility Index sinistra.

DISCUSSION

This case report discusses comprehensive treatment in the intensive care unit for a patient with a large infarct stroke accompanied by comorbid coronary heart disease with blockages in the 3 main blood vessels of the heart. Treatment in the ICU is carried out with the principle of ensuring that secondary brain injury does not occur due to hypoperfusion or brain hypoxia, which is carried out by non-invasive brain hemodynamic monitoring using TCD and NIRS.

At the start of the emergency room, the patient was planned to undergo a decompressive craniectomy because, based on the results of the CT-scan examination, it was found that a large infarction in the cortical area of the right parietotemporooccipital lobe was pressing and narrowing the surrounding sulcus and gyrus and causing a midline shift of up to 7 mm to the left even though there were no signs. -signs of intracranial hemorrhage. This is a life-threatening condition (characterized by an ischemic lesion volume > 150 cm3 or > 50% of the area supplied by the middle cerebral artery (MCA) experiencing infarction. This condition will usually clinically worsen within 24-48 hours if not treated immediately and has a mortality rate of up to 80%.[1]

Decompressive craniectomy aims to reduce ICP by opening the cranium as wide as possible so that the pressure in the brain decreases. Therefore brain perfusion returns to optimal. In patients with ischemic stroke or infarction, cytotoxic, ionic, and vasogenic brain edema can occur. Cytotoxic edema occurs immediately after an ischemic condition in the brain tissue, causing edema at the cellular level. Cytotoxic edema causes changes in ion concentrations at the blood-brain barrier (SDO), causing changes in the sodium concentration gradient, which will later create a driving force for the emergence of ionic and vasogenic edema.[7]

Based on this, it is necessary to monitor ICP so that we can immediately intervene if intracranial hypertension occurs. Currently, ICP monitoring is widely used using non-invasive examination tools (TCD, ONSD) because invasive ICP examination devices (which are the gold standard for ICP examination) have many disadvantages, including the risk of bleeding and infection, and are quite expensive.[5]

We perform routine TCD examinations in the ICU to evaluate intracranial pressure trends (not accurate ICP values) and see the results of our therapy, as well as to identify possible complications such as vasospasm.

Apart from that, we also carried out continuous brain oxygenation checks because previously, this patient was suspected of having a thrombus in the blood vessels in the carotid system of the brain. Non-invasive ICP examinations have now been widely studied for predicting ICP values. However, based on research that has been conducted, it was found that the PI value on the TCD examination has a moderate correlation with the ICP value. Therefore, this PI value should be used to see trends in ICT values but not as a reflection of the actual value.[7,8]

We carry out an NIRS examination to assess rSO2 levels or brain oxygen saturation. In patients with conditions of decreased heart function, there can be a condition of decreased oxygen levels in the brain; apart from that, in these patients, there is an increase in ICP, which requires greater systemic pressure to maintain perfusion pressure in the brain. In this patient, there had been a previous decline in heart function, so during intraoperative surgery, inotropic and vasopressor drugs were given to maintain adequate blood pressure and adequate cerebral perfusion pressure (CPP) in the brain, and this NIRS device was used as a parameter of oxygen adequacy. In brain tissue (as guiding therapy to target blood pressure to maintain adequate CPP). In conclusion, if the oxygen adequacy value is good, then CPP is considered adequate. However, the NIRS examination tool has several weaknesses namely the rSO2 examination cannot differentiate which blood vessels will be analyzed (arterial, capillary, or venous), where 70% of the blood vessels in the brain are veins, so the majority of what is read is venous saturation, and the second is Extracranial contamination such as melanin can interfere with NIRS readings. However, NIRS examination can still be useful for seeing trends in the oxygenation picture in the brain.[9,10]

Immediate post-operative care in the ICU, the patient underwent anesthetic sedation using propofol to reduce oxygen consumption because the patient had comorbid coronary heart disease with decreased heart function (LVEF 31.6%). Therefore, it is necessary to ensure that the patient's oxygen delivery is in optimal condition before the patient is awakened and the ventilator is weaned; the aim is to prevent secondary brain injury and ischemic heart conditions, which can aggravate the previous heart condition.[10]

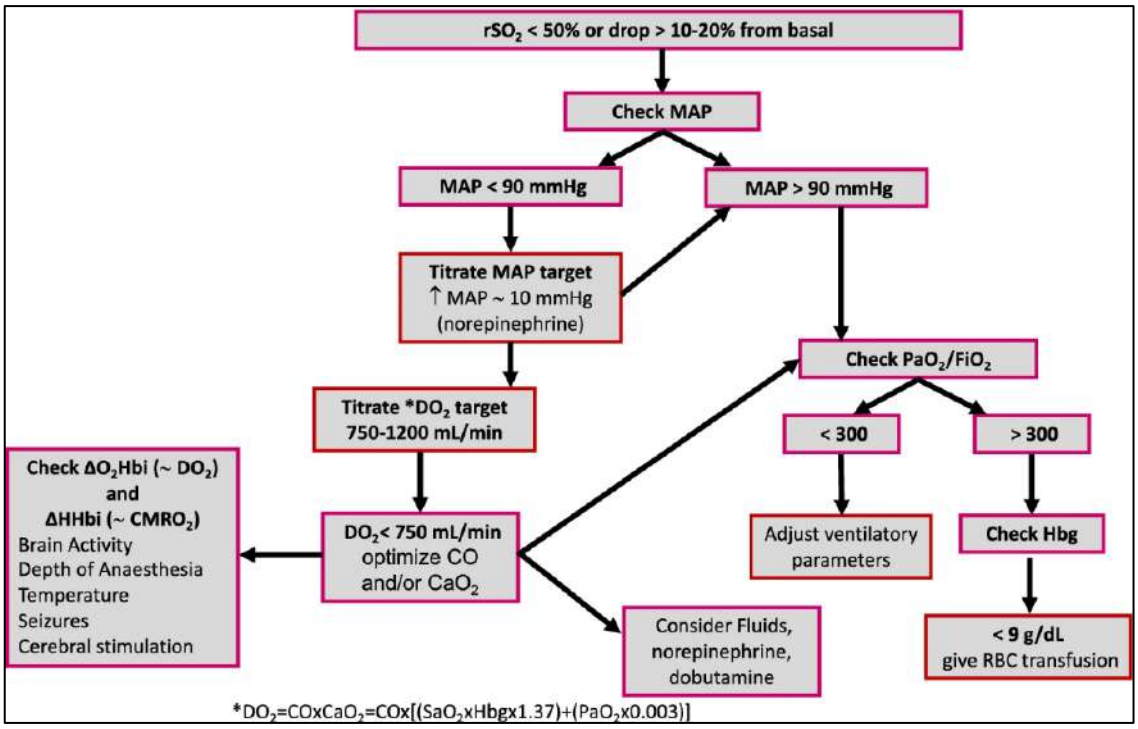


Figure 6. Brain Desaturation Management Algorithm

The patient's hemodynamic condition on the first to second day was in good condition even though he was still using hemodynamic support drugs however, on the second day, there was a decrease in the rSO2

value (>20 points) in the right brain, which may have been caused by interference with brain oxygen delivery due to a decrease in hemoglobin levels. Therefore we increased the vasopressor dose and administered 1 unit of PRC blood components, and shortly after the vasopressor was increased and the PRC was given, the rSO2 value increased again. On the 4th day, the patient's rSO2 value again decreased to below the critical value (<50), so it was decided to re-administer 2 PRC blood components with the patient previously given a loop diuretic to prevent fluid overload because the patient had low heart function. We carry out this management based on management guidelines using continuous monitoring (Figure 6).[11]

The rSO2 condition continued to improve until the 7th day of ICU treatment however, on the 8th day of treatment, there was a decrease again in the rSO2 value in the left brain to below the critical value but hemodynamically, the patient was in a stable condition and the blood hemoglobin value was also optimal, but when the TCD examination was carried out in the right MCA, the mFv value was 146.7, which is included in the criteria for suspicion of vasospasm and needs to be proven by calculating the Lindergard ratio by calculating the ratio of MCA mFv to right carotid artery mFv. Then, the patient had the right internal carotid artery mFv checked, and the mFv value was found to be 41.91 if the Lindergard ratio value was calculated as 3.5. Based on this, the patient was diagnosed with mild vasospasm in the right MCA and planned to be given nimodipine and undergo a repeat CT scan. This patient underwent a CT-scan evaluation because vasospasm is usually caused by intracranial bleeding, and based on the CT-scan results, intracerebral bleeding was found in the right parietal lobe.[12,13]

Table 1. Diagnostic criteria for cerebral vasospasm

Mean flow velocity cm/s	Lindegaard ratio	Vasospasm or hyperemia and grade
<120	<3	Hyperemia
>120	3-4	Mild vasospasm + hyperemia
>120	4-5	Moderate vasospasm + hyperemia
>120	5-6	Moderate vasospasm
>180	3-4	Predominently moderate hypermia + mild vasospasm
>180	4-6	Moderate vasospasm + hyperemia
>180	>6	Severe vasospasm

When a condition of cerebral vasospasm occurs, there is a decrease in rSO2 in the healthy brain, this may be because the area that is damaged (right brain) experiences a decrease in ICP compared to the healthy area, and the patient's blood pressure at that time may be too high (because at that time the medication should be vasopressors have begun to be reduced), resulting in a condition of cerebral vasoconstriction in healthy parts of the brain, even though in the damaged area there is a condition of vasospasm. This causes the inverse cerebral steal phenomenon (Robin Hood syndrome), which means that more blood flows to areas that have lower vascular resistance and brain pressure. Based on this idea, we weaned the noradrenaline drug until it stopped, and on the 10th day of treatment, the left brain rSO2 increased again, with the mFv value trending downwards. When the TCD was re-examined on day 12, the right MCA mFv value was 72.5.[13]

The dose of mannitol was tapered off starting from the first day of treatment in the ICU until it was stopped on the second day. This is because, based on clinical symptoms, the patient had undergone a decompressive craniectomy, and the TCD-PI examination showed that the patient's ICP had decreased and the patient had a condition of decreased heart function, therefore there was a fear of fluid overload when administering mannitol. The dose of mannitol needs to be reduced gradually to prevent rebound intracranial hypertension.[14]

Tranexamic acid and vitamin K are given after the operation is completed, and the patient is treated in the ICU. Administration aims to prevent brain hemorrhage or bleeding from surgical wounds in the post-operative phase. Based on a study, it is stated that patients who use tranexamic acid and vitamin K will experience a decrease in prothrombin time (PT) by 2.7 times and activated partial thromboplastin time (aPTT)

by 1.6 times compared to those who do not use it. 15 However, the use of tranexamic acid should not be given for a long period of time because it can increase the risk of venous thromboembolism (VTE), although this is still controversial.[16]

On the 3rd day of treatment, the patient experienced an increase in body temperature, and a decrease in saturation although not significant, the phlegm became greenish productive, so a resistance culture result was carried out and antibiotic escalation was carried out according to the germ map at our hospital. On the 9th day of treatment, the culture results showed that Acinetobacter baumannii was sensitive to the antibiotic amikacin, so antibiotic de-escalation was carried out according to the resistance culture results.

On the 4th day of treatment, we gave a prophylactic dose of subcutaneous heparin to prevent VTE because the patient was at high risk for venous thromboembolism with risk factors for paresis in the lower extremities, bed rest for more than 3 days. This patient was also given the anti-platelet aspilet and clopidogrel for anti-platelet therapy in ischemic stroke patients because, based on a study, it was stated that combination therapy of clopidogrel and aspilet in ischemic stroke and infarction patients would reduce major ischemic events but increase the risk of bleeding for up to 90 days compared to single aspilet therapy. Based on a repeat CT-scan examination on this patient which was carried out on the 8th day, it was found that there was intracerebral bleeding which was probably caused by the administration of dual anti-platelet and anti-coagulant prophylaxis. At that time, subcutaneous heparin was only stopped, but clopidogrel and aspirin were continued; perhaps the administration of clopidogrel was stopped, and only aspirin was continued because the administration of aspirin alone did not significantly affect the incidence of additional bleeding.[17]

The patient underwent a tracheostomy on the 4th day of treatment. This patient underwent a tracheostomy with the aim that the patient could be weaned from the ventilator early without waiting for adequate consciousness to improve. Based on a retrospective study and meta-analysis, it is stated that early tracheostomy in ischemic stroke patients with poor GCS levels can shorten the length of stay in the ICU, thereby reducing the incidence of ventilator-associated pneumonia (VAP). So, early tracheostomy has a positive effect on the prognosis of patients with severe stroke infarction who require mechanical ventilation.[18]

CONCLUSION

The main principle of management in this patient is to carry out measurable therapy using TCD and NIRS examination tools. So that we have more accurate targets (blood pressure, time for transfusion, and anti-vasospasm medication given) and so that the treatment of patients in the ICU can be more comprehensive.

DECLARATIONS

This article is already approved by the Ethics Committee of Hasan Sadikin General Hospital

CONSENT FOR PUBLICATION

The Authors agree to publication in Journal of Society Medicine.

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COMPETING INTERESTS

The authors declare that there is no conflict of interest in this research.

AUTHORS' CONTRIBUTIONS

All authors contributed equally to the conception, study, execution, analysis, and interpretation of the data. The authors agreed to publish this work and agreed to be accountable for it.

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