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The Role of ONSD-TCD in Transforming the Approach in Critical Care Management of Acute Spontaneous High-Grade Subarachnoid Hemorhagic: A Case Report

Khairunnisai Tarimah[1], Dewi Yulianti Bisri[2]

Abstract

Background: Bleeding in the subarachnoid space (SAH) is a type of bleeding stroke (SH) with a prevalence of 10%-20% among all kinds of stroke. The incidence of SAH accounts for 5% of stroke but contributes to high disability and mortality rates. SAH that occurs in a ruptured aneurysm can cause death up to 40% within 30 days. The most common complications of SAH are re-bleeding, vasospasm, and Delayed Cerebral Ischemia (DCI). Intensive care is necessary for high-grade SAH patients. In addition to managing intracranial pressure control (ICP), it is also for close monitoring to prevent or detect complications after SAH. Transcranial Doppler (TCD) and ONSD are non-invasive monitoring techniques that are starting to be widely used in critical care. TCD can provide an overview of cerebral artery blood flow changes through flow velocity. Some studies also mention that the ICP value can be obtained through the conversion of TCD measurement results in the form of Pulse indexability (PI). Comparison between the average velocity of cerebral blood flow-internal MFV carotid arterial-iICA (generally represented by Media cerebri artery-MCA) to the value of external MFV ICA-eICA (cervical ICA) through the formula MFV iICA / MFV eICA, the results can assess the grading Lindegard ratio (Ldg) which is generally used to determine the severity of a vasospasm. ONSD is also an N.II sheath measurement whose results describe whether there is an increase in ICP.

Methode: After getting approval from the family, we reported the case of a 59-year-old woman the Sumbawa tribe of West Nusa Tenggara who came to the ED with weakness on the left side of the body and experienced suddenness when waking up from the night. Another complaint is nausea-vomiting. Patients who have hypertension > 10 years who do not routinely seek treatment. The average family history of hypertension and stroke. The patient appeared conscious (GCS E4V5M6) with TD 211/112 mmHg and a pulse rate of 87 bpm. It wasn't long before the patient had a seizure. The patient is transferred to the ICU. A CT scan of the head found SAH, IVH, and hydrocephalus. While in the ICU, the patient's consciousness decreases, GCS E2V2M5 Hunt-Hess grade 4 and WFNS score 4. Technical intubation of RSI with thiopental, fentanyl, and rocuronium is performed. Right/left ONSD 0.55/0.56 cm, right/left MFV 22.95/30.93 cm/sec, right/left PI 2.84/2.38, and right/left Lingard ratio 1.00/1.76. First-tier therapy with CSF drainage action is done cito-emergency.

[1] Trainee of Neuroanesthesia and Neuro Critical Care/ Medical Faculty Padjajaran University/Dr Hasan Sadikin Hospital-Bandung, East Java Indonesia, nisatarimah@yahoo.com, ORCID: 0009-0007-6354-2279

[2] Department Anesthesiology and Intensive Care, Medical Faculty Padjajaran University/Dr Hasan Sadikin Hospital-Bandung, East Java Indonesia, yuliantibisri@yahoo.com, ORCID: 0000-0002-7637-9710

Result: The objective of this report is to present our experience in utilizing Transcranial Doppler (TCD) and Optic Nerve Sheath Diameter (ONSD) for screening and monitoring patients with Subarachnoid Hemorrhage (SAH) resulting from the rupture of an intracerebral artery (IA) aneurysm. By integrating the diagnostic and screening outcomes from the TCD-ONSD examination with the patient's clinical condition and CT scan results, we enhanced the patient's overall condition from high grade to mild grade during their ICU treatment. Although the patient's critical period is still ongoing, with comprehensive and multidisciplinary management, the patient was able to leave the ICU with full consciousness.

Keywords: *SAH, ONSD, TCD, Pulse Indexability, Mean Flow Velocity, Lindegard, Rupture Cerebral Aneurysm.*

INTRODUCTION

Subarachnoid Hemorrhage is a type of stroke caused by bleeding in the space between the brain and the thin tissues covering the brain, known as the subarachnoid space. Chronic hypertension is a known risk factor for subarachnoid bleeding, which can occur when there is a rupture of a cerebral aneurysm in hypertensive patients (Basmala et al., 2022). Subarachnoid hemorrhage (SAH) is a significant cause of bleeding strokes, accounting for 5% of all such cases. It is a severe condition with high morbidity and mortality rates, ranging from 26% to 50%. Chronic hypertension is a significant risk factor for SAH, along with other factors such as age, smoking, family history, and use of sympathomimetic medications. Although the incidence of SAH is rare, ranging from 2 to 22 cases per 100,000, studies indicate that as many as 21% of deaths from SAH occur within 24 hours, 37% within seven days, and 44% within 30 days.(Suarez, 2007; Vivancos et al., 2014) Non-traumatic SAH (spontaneous SAH) accounts for 85% of all cases and is primarily caused by the rupture of an aneurysm (aSAH). Although aSAH represents only 3%-5% of all stroke incidence, it is responsible for 4% of stroke deaths. The rate of aSAH-related deaths increases to 27% when it occurs in people aged 65 years or older.(Basmala et al., 2022; Pratik V. Patel and Michael J. Souter, 2017)

An aneurysm is a bulge that can occur in an artery. The most common location for an aneurysm is the anterior communicating artery (ACA) is 36%, followed by the middle cerebral artery (MCA) is 26%, the posterior communicating artery (PCA) is 18% and internal carotid arteries (ICA) is 10% cases. Posterior circulation aneurysms make up 9% of all cases, while 20% of cases involve multiple aneurysms. The incidence of associated arteriovenous malformation (AVM) is less than 2%. In neuroimaging examinations, the location of a subarachnoid hemorrhage (SAH) can generally indicate the source of the aneurysm. Aneurysmal arteries are typically found at bifurcation points within the circle of Willis. The most common locations for these aneurysms are at the bifurcation of the basilar artery, at the junction of the ipsilateral posterior inferior cerebellar artery (PICA) and vertebral artery (VA), or on the anterior communicating artery.(Marder et al., 2014)

Hemodynamic changes caused by factors like high blood pressure can lead to the formation and rupture of an aneurysm. Aneurysms can have serious consequences, and early detection and treatment are crucial for optimal patient outcomes(Penn et al., 2011). Endothelial dysfunction is believed to be a consequence of hemodynamic stress, which triggers an inflammatory response in the arterial walls. This dysfunction ultimately leads to vascular remodeling and cell death. The progressive weakening of the arterial wall is caused by macrophages, apoptosis of smooth muscle cells, and further degradation of the extracellular matrix (Chalouhi et al., 2013; Pratik V. Patel and Michael J. Souter, 2017; Sanicola et al., 2023).

A sudden and severe headache, also known as "Worst Headache of Life" (WHOL), is not a definitive sign of an aneurysmal subarachnoid hemorrhage (aSAH). Only a few patients (6-17%) who experience this type of headache will actually have aSAH. Other symptoms include seizure at onset (6%), temporary loss of consciousness (26%), and vomiting before the onset of a severe headache (69%). Some patients may experience a severe headache a few days before they present with aSAH, known as "Sentinel Headache." Physical examination usually doesn't show specific findings, but the presence of a new third cranial nerve palsy (partial or complete) should raise suspicion of an ipsilateral posterior communicating artery aneurysm.(Diringer, 2009; Pratik V. Patel and Michael J. Souter, 2017; Treggiari et al., 2023) Because SAH is a serious medical condition that requires accurate assessment of its severity, some scoring system was publish as a clinical guidance (figure 1).(de Oliveira Manoel et al., 2016; Ran et al., 2023; Rosen & Macdonald, 2005; Vivancos et al., 2014)

The primary objective of treating aSAH is to manage intracranial pressure and prevent potential complications such as seizures, re-bleeding, vasospasm, and delayed cerebral ischemia (DCI). Critical care professionals widely use non-invasive ultrasound techniques such as ONSD (optic nerve sheath diameter) and TCD (transcranial Doppler) to achieve this goal. Non-invasive procedures are preferred over invasive ones as they can be conducted at any time with minimal complications. TCD is particularly useful in cases of SAH, as it can directly detect and evaluate abnormalities in cerebral blood flow. The internal carotid arterial system supplies blood to the brain, and any changes in blood flow can have serious consequences. TCD can indirectly assess cerebral blood flow, cerebral perfusion, and intracranial pressure by measuring cerebral blood flow variability through mean flow velocity (MFV) and pulsatile index in the internal carotid arterial system.

Cerebral vasospasm is a typical complication of SAH, which typically occurs 4-14 days after the initial hemorrhage. In some cases, however, it can occur within 48 hours in 13% of patients. Cerebral vasospasm is a narrowing of the blood vessels in the brain, which reduces blood flow and can lead to delayed cerebral ischemia and cerebral infarction. TCD can accurately diagnose cerebral vasospasm by measuring mean flow velocity and Lindegaard ratio. This is crucial for predicting delayed cerebral ischemia and cerebral infarction and distinguishing cerebral vasospasm from hyperperfusion. In conclusion, non-invasive ultrasound techniques such as ONSD and TCD are crucial in managing aSAH. TCD is highly effective in detecting and assessing cerebral blood flow abnormalities, which are essential for the diagnosis and management of cerebral vasospasm. By providing accurate and timely information about cerebral blood flow, TCD can help prevent complications and improve outcomes for patients with aSAH..(Bellner, J., Romner, B., Reinstrup, P., Kristiansson, K., Ryding, E., & Brandt, n.d.; Bittar & Hannawi, 2022; Hironobu Hayashi and Masahiko Kawaguchi, 2019)

An increase in intracranial pressure (ICP) can lead to an increase in the diameter of the optic nerve sheath. This happens due to the distribution of pressure and/or volume of cerebrospinal fluid (CSF) from the intracranial to the intraorbital space. As a result, the ONSD can be evaluated and measured using ultrasound, as previously described by Ossoinig in the 1970s. Due to its anatomical structure, the ONSD measurement using ultrasound could be a useful tool for monitoring ICP in various conditions. This can include traumatic brain injuries, hydrocephalus, and other neurological conditions where an increase in ICP can be a significant concern. Additionally, during surgical procedures, an increase in ICP has been reported as a possible complication. Therefore, monitoring the ONSD can be crucial in such cases. Ocular ultrasonography is a simpler and less invasive diagnostic method of measuring ICP compared to other tools. It is a safer and quicker option that does not require the use of invasive procedures. Moreover, ONSD changes can be observed at any time during surgical procedures and follow-up, making it easier to monitor the patient's condition. Overall, ONSD measurement using ultrasound is a useful method for monitoring ICP and can contribute to better patient outcomes..

("Ocular Ultrasonography to Detect Intracranial Pressure in Aneurysmal Subarachnoid Hemorrhage.," n.d.; Vitiello et al., 2022)

Case Presentation

A woman, Samawan tribe, age 59 years, BB 65 kg, TB 155 cm, arrived at the Emergency department (ED), alert and complaining of weakness on half the right side of the body with nausea and vomiting. She had a history of chronic hypertension and does not routinely take medication; the drug consumed is amlodipine 1x10 mg. Ottawa's score was 3 (age> 40 y o, neck pain, limited flexy of the neck). Blood pressure was 211/112 mmHg, heart rate was 87 bpm, and oxygen saturation in room air was 99%. Suddenly, she had a seizure and then transferred to ICU. A laboratory test was performed. Her Hb 11,4 gr/dl Hct 45% Ureum 35.5 creatinine 0,86 mg/dl sodium 140,6 meq/L potassium 2,63 meq/L. A radiological finding of a Non-contras head CT scan shows Intraventricular hemorrhage (IVH) in lateral -3rd-and 4th ventricles, acute Subarachnoid hemorrhage (SAH) interhemisfer, bilateral fissure Sylvie, ambient cistern, interpeduncular's cistern, dan right cisterna pontocerebellar, and mild communicating hydrocephalus (Figure 1). Another test is CT angiography showed Mild dilatation Right A. Carotid interna at C7 segment (± 3,61 mm) and Right MCA at M1 segment (± 2,6 mm) suspected fusiform aneurysm, Saccular aneurysm at right posterior inferior cerebellar (PICA) ± 3,2x2,7 mm, hypoplasia right A.vertebralis (VA) and left a. communicating posterior (PCA) (Figure 2). She had a progressive lowering of Hunt-Hess grading and WFNS score from 1 to 4, Modified Fisher's scale was 2, and Glasgow's coma scale from alert to E2V1M5 after a seizure.

First-tier therapy to manage elevated intracranial by the effect of hematoma was performed with intubation thiopental 5 mg/kgBW follow syringe pump 2 mg/kgBW/hours, fentanyl one mcg/kgBW, rocuronium, and lidocaine 1 mg/kgBW. The neurosurgeon consulted emergency surgery to drain Cerebrospinal fluid (CSF). Sementara menunggu jawaban dan persiapan operasi, tatalaksana farmakologi berupa pemberian nimodipine per oral 6x60 mg, control tekanan darah dengan nicardipine, optimalisasi potassium with KCL. The findings of the Transcranial Doppler and Optic Nerve Sheath Diameter (ONSD) examinations revealed an acute elevation in intracranial pressure (ICP), which subsequently led to vasospasm (figure 3).

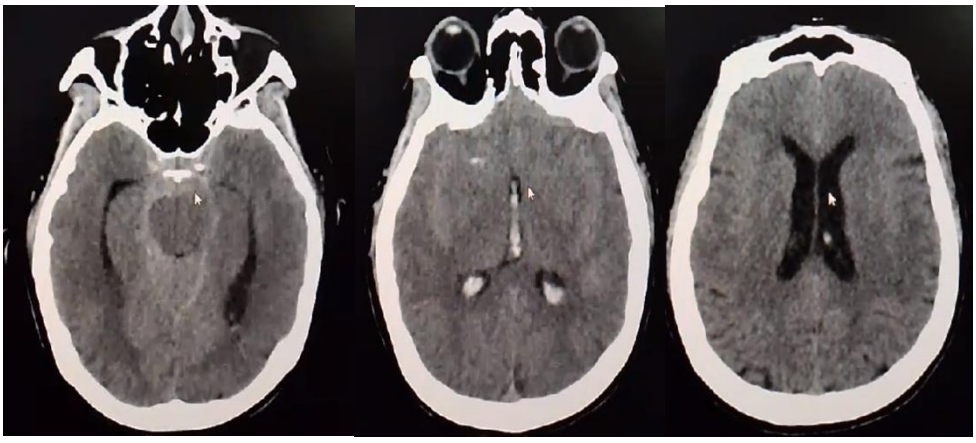

Figure 1. Non-contras head CT (NCT)

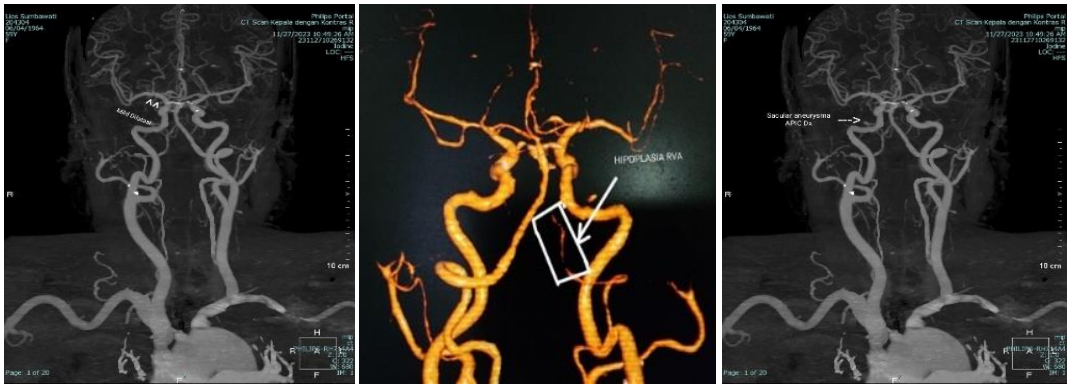

Figure 2. CT angiography

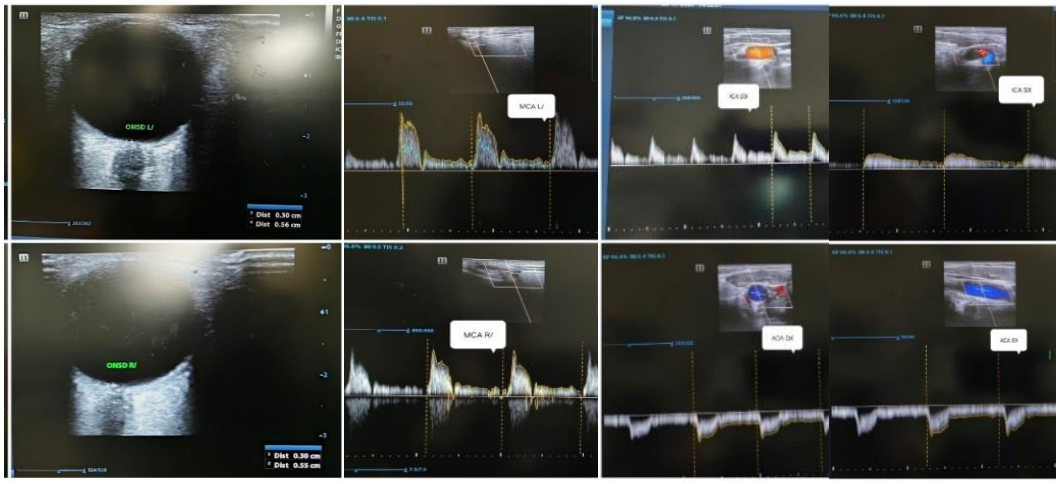

Figure 3. TCD and ONSD before surgery

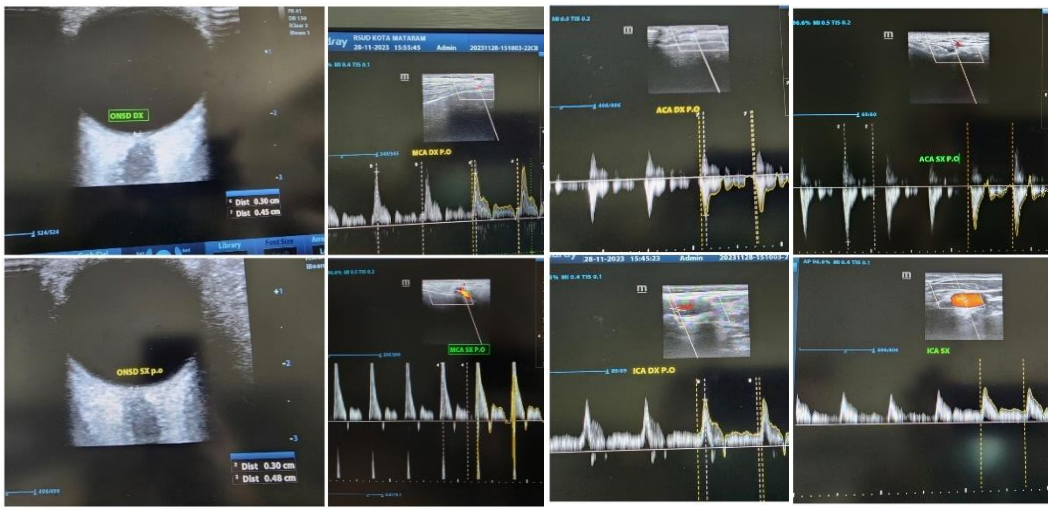

Figure 4. TCD and ONSD post drainage CSF by VP-Shunt

Diagnostics Assessment

Spontaneous SAH-IVH due to aneurysmal rupture (aSAH)

Therapeutic Intervention

The treatment goals are 1). Control of ICP and optimization of cerebral oxygenation, 2) neurological and systemic support, 3) early aneurism repair if available, and 4) prevention, monitoring, and treatment of medical complications.

Table 1.

	ED	First arrival ICU	Post-op ICU D-1
A	Patent airway +, no intervention	Patent airway	On ventilator
B	Spontan regular breathing, RR 24-27 x/min	Hyperventilation RR 20-27 x/min	
C	BP 211/112 (145) mmHg ➔ Start nicardipine adjusted dose to target SBP 140-180 mmHg, MAP 70-90 mmHg ➔ Nimodipin oral 6x60 mg	BP 172/107 (129) mmHg	
D	GCS E4V5M6 Hunt&Hess score 2	Stupor GCS E2V1M5- R2 Hemiparese Sx Hunt&Hess Score 4 Intubation : Tiopol -fentanyl-rocuronium Nimodipin 6x60 mg	FOUR Score E4M3B4R1 Tiopol 1-3 mg/kgBW/hrs Fentanyl 0,25 mikro/kgBW/hrs
E	pCO2 32 mmHg Hb 11,4 gr/dl Hct 33,4% Normothermy, normoglycaemia, Nirmal limit electrolyte	pCO2 56 mmHg	Hb 10,9 gr/dl Hct 32% Sodium plasma 140,8 Potassium plasma 3,03
F	NCT Head CTA	ONSD-TCD Urgent EVD/VP-shunt	ONSD-TCD

Outcomes and Follow-Up

After undergoing a VP shunt, patients are treated with ventilators for up to 12 hours, followed by ddi weaning and hemodynamic stability. While in the ICU, the patient undergoes ONSD-TCD series and evaluations of consciousness and neurological function (table 1 attachment). After 12 hours, the patient is extubated and moved to a room on the fifth day of treatment in the ICU. At this point, the patient's SBP is 124/76 mmHg HR 62 bpm, and they are well conscious. The Hunt & Hess Score has improved from 4 to 1. The patient continues to receive treatment of nimodipine 6x60 mg and lactulose syrup 3x1 tablespoons, along with other supportive drugs.

DISCUSSION

We managed the case of a 59-year-old female patient who presented with SAH arising from spontaneous risk factors, namely chronic hypertension and a family history of the same. It is well-established in various studies that hypertension is a significant risk factor for stroke, bleeding, and SAH.(Arismendi-Morillo et al., 2008; Djelilovic-Vranic et al., 2017; Rosen & Macdonald, 2005; Suarez, 2007) The symptoms manifested by patients upon their arrival at the emergency department are more likely to be indicative of stroke symptoms, particularly hemiparesis. Sorimachi et al have reported a similar occurrence in patients with ICA aneurysm rupture, with a prevalence of up to 2.9%. A possible mechanism behind hemiparesis without hematoma is the interaction between transient ipsilateral hemispheric functional failure caused by the impact of aneurysmal rupture and transient ischemia of the perforators originating from the internal carotid artery.(Sorimachi et al., 2019) Medical research indicates that nausea and vomiting are frequently experienced, up to 69% of the time, prior to the onset of headaches. Furthermore, seizures typically occur within 24 hours of the rupture of aSAH in proximal ACA and are a significant risk factor for poor prognosis, particularly when affecting individuals under the age of 51.(Darkwah Oppong et al., 2021) The presence of an unruptured intracranial aneurysm (IA) or elevated intracranial pressure (ICP) following a rupture may result in compression of the third and sixth cranial nerves (CN III, CN VI), leading to cranial nerve palsies. Additionally, IA rupture may trigger seizures in certain patients. It is imperative to monitor such complications closely to prevent further neurological damage.(Diringer, 2009; Treggiari et al., 2023)

As many as 85% with non-traumatic hemorhagic stroke occur due to cerebral aneurysm rupture.(Basmala et al., 2022; Pratik V. Patel and Michael J. Souter, 2017) Upon the patient's arrival at the Emergency Department, they have reported experiencing symptoms that have commenced less than an hour ago. As a result, we have conducted the Ottawa SAH assessment to guide the SAH diagnostic testing process (Figure 4).(Hoh et al., 2023; Treggiari et al., 2023) In cases of subarachnoid hemorrhage (SAH), grading scales like Hunt-Hess and WFNS, as well as Fisher/modified Fisher's scale, are commonly used. Our patients tend to have unfavorable outcomes when they arrive with high grades of Hunt-Hess and WFNS, with a score of 1 that rapidly drops to 4 within 6 hours. A Vasograde predictor tool has been developed by De Oliviera et al through RCT cohort to predict delayed cerebral ischemia (DCI) occurrence. This tool combines WFNS score with mFisher's scale.(De Oliveira Manoel et al., 2015) It is essential for patients who are at risk of vasospasm-DCI to undergo serial Transcranial Doppler (TCD) monitoring to ensure appropriate follow-up.

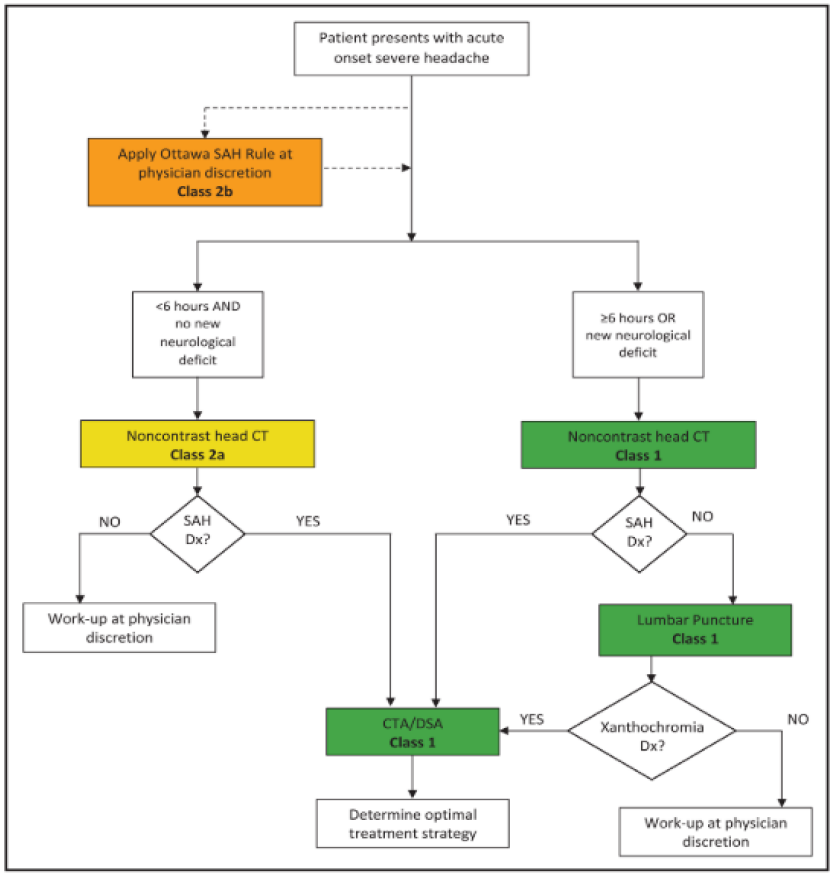

Figure 5.

Adopted from 2023 Guideline for the Management of Patients With Aneurysmal Subarachnoid Hemorrhage: A Guideline From the American Heart Association/American Stroke Association,2023

Transcranial Doppler (TCD) represents a non-invasive monitoring tool that has found significant application in the critical care domain. Its unique ability to evaluate cerebral blood flow and perfusion renders it a highly suitable candidate for deployment in cases of head trauma. The technique involves the use of ultrasonic waves to non-invasively measure blood flow velocity in the major cerebral arteries, enabling clinicians to assess cerebral perfusion and identify abnormalities associated with brain injury. The real-time insights offered by TCD make it a valuable tool in the management of critical care patients..(Razumovsky, 2023) Fatima and her colleagues have unequivocally established the efficacy of transcranial doppler ultrasound (TCD) as a reliable prognostic tool for patients with traumatic brain injury (TBI) through a comprehensive randomized controlled trial meta-analysis.(Fatima et al., 2019) Transcranial Doppler (TCD) is an effective tool for detecting cerebral vasospasm in cases of aneurysmal subarachnoid hemorrhage (aSAH). This is due to its ability to assess pulsatile index (PI), which widens its usefulness.(Bittar & Hannawi, 2022; Rigamonti, 2008) Transcranial Doppler (TCD) is a diagnostic tool employed in various fields to evaluate cerebral blood flow, cerebral autoregulation, and cerebral perfusion by measuring mean flow velocity (MFV) and pulsatility index (PI). The PI value, when converted, can also help determine intracranial pressure (ICP).(Bellner, J., Romner, B., Reinstrup, P., Kristiansson, K., Ryding, E., & Brandt, n.d.)

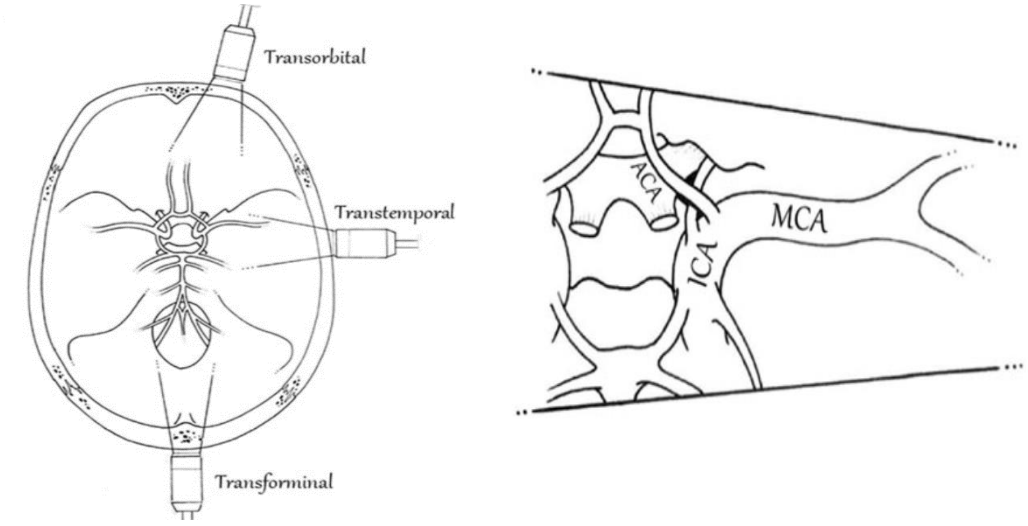

Figure 6. Schematic probe approach of TCD

Transcranial Doppler (TCD) is a technique that uses ultrasonic waves to calculate the velocity of blood flow in a vessel. TCD ultrasound probes emit ultrasonic waves (fo) through the tissue at a known speed (c). The waves reflect off the moving red blood cells (RBCs) in the vessel, producing an echo at a different frequency (fe). This shift in frequency (Doppler shift) allows for the calculation of velocity (v), which needs to be corrected for the angle of insonation (θ). If the angle of insonation is less than 30°, the degree of error will be less than 15%..(Gomez et al., 2021) In our case, transtemporal windows were selected to detect MFV in MCA and ACA, with MFV values in MCA-ACA < 100 cm/sec.

Cerebral vasospasm can be predicted by observing a high mean flow velocity on transcranial Doppler (TCD) and is often associated with delayed cerebral ischemia and cerebral infarction. The mean flow velocity and Lindegaard ratio are commonly used to diagnose cerebral vasospasm accurately using TCD. These measures help distinguish it from hyperperfusion. (Hironobu Hayashi and Masahiko Kawaguchi, 2019) Although Cerebral vasospasm and DCI typically occur between days 3–14 following aSAH with a peak incidence at 7–10 days, with a wide spectrum of symptoms and signs including newly progressive cephalgia, confusion, a change in the level of consciousness, or new focal neurologic deficit(Pratik V. Patel and Michael J. Souter, 2017), but the routine daily measurement of TCD velocities in patients with SAH is recommended until the expected period of cerebral vasospasm for up to 21 days.(Bittar & Hannawi, 2022) Transcranial Doppler (TCD) is the highly recommended monitoring method for detecting cerebral vasospasm after aneurysmal subarachnoid hemorrhage, according to the American Heart Association.(Connolly ES Jr, Rabinstein AA & Al., 2012) Recent research of a systematic review reveals that TCD monitoring boasts a high sensitivity rate (90%) and negative predictive value (92%) concerning the association between cerebral vasospasm and delayed cerebral ischemia. However, it is worth mentioning that current diagnostic practices for delayed cerebral ischemia rely only on the mean middle cerebral arterial flow velocity cutoff value (typically 120 cm/s in most studies).(Kumar G, Shahripour RB, 2016) Characteristics of TCD values to establish vasospasm-DCI(Bittar & Hannawi, 2022).

Table. 2

	Sensitivity	Specificity	MFV Value To Vasospasm	
MCA	67-88%	72-99%	> 120 cm/sec	120-200 cm/sec , Mild to Moerate >200 cm/sec severe (87%PPV)
ACA	18-50%	65%	≥120 cm/sec	or increase MFV >50% or 50 cm/sec than baseline
BSA	76,9%	79%	>85 cm/sec	>115 cm/sec is high risk for DCI modified Lindegaard ratio=MFV BA/MFV EVA >2
PCA	42-48%	69-72%	>110 cm/sec	

Optical nerve sheath diameter has been widely used as a predictor of ICP enhancement in various neurological conditions. A meta-analysis suggests that the use of ONSD can be a predictor of ICP improvement. In a narrative review of ONSD in various types of surgery(Lee, 2019) Bernardo et al concluded that B-ONSD has a high sensitivity to detect elevated ICP.(De Bernardo et al., 2022) A study of ONSD in SAHs that had undergone CSF drainage with EVD and ICP repair, reported that ONSD was still widening. This is likely due to impaired retraction of the optic nerve sheath due to the accumulation of blood that forms fibrin in the intraorbital SAS space. (Bäuerle et al., 2016) Another study in line, Baurle et al found that ONSD was not shown to play a role as monitoring in improving outcomes of SAH patients.(Rigamonti, 2008). Given that ONSD can not only be measured by ultrasound but can be measured through MRI, Murat et al on conveyed a threshold value of 6.1 mm, the sensitivity and specificity of SAH was 72%. (Yesilaras et al., 2017) In our case, the patient's ONSD value when in high grade was in the range of 0.55 – 0.56 cm. Although with an insignificantly increased value, the clinical patient supports an eICP.

Various scoring systems are utilized to predict the mortality and survival rate of patients with aSAH. The patients under our care are currently categorized as high-grade, with poor outcomes expected while in the ICU. Specifically, they have a Hunt-Hess grade and WFNS score of 4. After seizures occur, a reduction in GCS E2V1M5 can result in acute complications such as rupture-rebleeding. To alleviate ICP caused by the pressure of a massive hematoma, intubation is performed, which also guarantees sufficient cerebral perfusion to avoid secondary brain injury caused by hypoxia. However, this procedure further elevates the risk of mortality. During intubation and airway management, it's important to avoid any blood pressure changes that could lead to a rupture or re-bleeding. Elevated intracranial pressure (ICP) can cause increased transmural pressure (TMP), which can trigger such events. To ensure a smooth intubation without any blood pressure changes, Thiopental is used as an induction agent because it has a lower effect on cerebral metabolic rate of oxygen consumption (CMRO2) and does not significantly decrease cerebral blood flow (CBF) or cerebral perfusion pressure (CPP). Hence, Thiopental is preferred over barbiturates. The maximum decrease in CMRO2 induced by Thiopental is around 55 to 60%. (Kundra et al., 2014; Santra & Das, 2007) Agents used to blunt laryngoscopic response to intubation we administer I.V. lidocaine, 1 mg/kg 2-3 min before intubation. Rocuronium is a nondepolarizing agent which has no effect on CBF or ICP and is a suitable alternative for rapid sequence induction in a dose of 0.6-1.2 mg/kg.(Kundra et al., 2014)

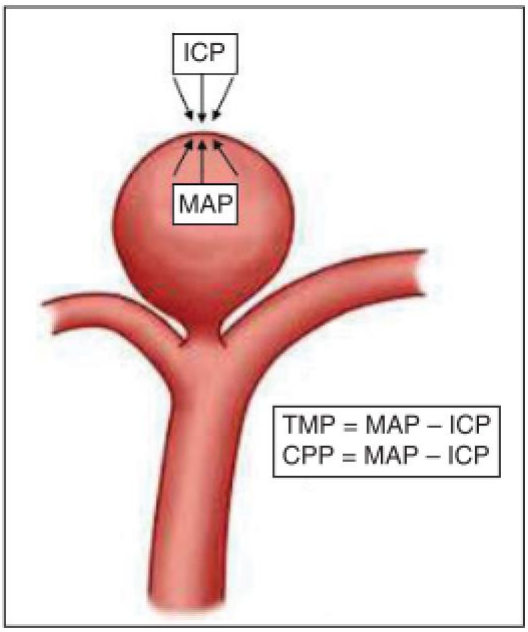

Figure 7. Transmural pressure = Mean arterial blood pressure (MAP) — intracranial pressure (ICP); cerebral perfusion pressure = MAP — ICP

Using a mechanical ventilator after intubation in critical care must be concern to VAP, so patient who require mechanical ventilation for >24 hours, implementation of a standardized ICU care bundle is recommended to reduce the duration of mechanical ventilation and hospital-acquired pneumonia (strong recommendation). Maintain Volume status euvolemia and using mineralokortikoid to treat natriuresis and hyponatremia is level 2a.(Hoh et al., 2023) Improving cerebral perfusion oxygenation CPP) is obtained by maintaining blood pressure (CBF) within the normal autoregulatory threshold and lowering ICP, based on the formula CPP= MAP-ICP. The target of reducing BP in patients with SAH to prevent rebleeding based on the AHA Guideline is SBP <160 mmHg or <180 mmHg.(Hoh et al., 2023) However, a meta-analysis suggests that SBP > 160 mmHg is a risk factor for early rebleeding in SAH.(Tang C, Zhang TS, 2014) The use of antihypertensive medications to lower BP is recommended. Calcium Channel blockers mostly use to lowering base on many studies. (nicardipine, nimodipine). Liu et al in a meta-analysis concluded that nimodipine was highly effective at treating SAH and significantly improved patient outcomes with minimal adverse events.(Liu & Li, 2022) Nimodipine is a medication that can easily cross the blood-brain barrier and enter the nervous system due to its high lipid solubility. As a calcium channel blocker, it decreases the influx of Ca2+ in brain cells and prevents the development of free radicals. This promotes the relaxation of vascular smooth muscle, resulting in a reduction in vasospasm. Furthermore, nimodipine has anti-free radical and antagonistic effects on endothelin neurotoxicity, which enhances the tolerance of nerve cells to ischemia and hypoxia. This leads to improved neurological function, decreased cerebral ischemia-related mortality, and global cerebral infarction following SAH, ultimately resulting in a better prognosis. (Liu & Li, 2022; Minhas et al., 2022; Vivancos et al., 2014) We used a nimodipine to reduce BP in this case with concern, according to NSCC oral nimodipine 6x60 mg is a strong recommendation to reduce risk of cerebral vasospasme and the injury but because nimodipine as an antagonist Ca hypotensive is strong enough to hypotension effect then a dose of 3x30 mg is recommended.(Frontera et al., 2016; Treggiari et al., 2023) In addition to antihypertensives given to SAH, also antithrombolytic drugs (tranexamic acid) show a good outcome contribution to the prevention of rebleeding (strong recommendation) .(Hoh et al., 2023)(Ran et al., 2023)(Minhas et al., 2022)

CONCLUSION

Subarachnoid hemorrhage (SAH) is a severe medical condition that frequently results in stroke and high mortality rates. For patients with moderate-severe grade SAH, multidisciplinary management and intensive care unit (ICU) care are typically essential. Non-invasive techniques, such as optic nerve sheath diameter-transcranial Doppler ultrasound (ONSD-TCD), are preferred by physicians to monitor intracranial pressure in real-time, especially for patients who have experienced an aSAH rupture. By utilizing uONSD-TCD, physicians can promptly identify and minimize complications that may arise during the critical 21-day period following an aSAH rupture. However, due to the extended critical period necessitating ICU care, we face challenges in performing serial ultrasound follow-ups, rendering it difficult to monitor patients for early complications related to LOS. The question remains, what is the optimal duration for close monitoring with uONSD and TCD to decrease the risk of post-SAH complications, which are the most frequent cause of mortality? In other words, what is the appropriate length of time for patients with aSAH to receive semi-intensive care to avoid such complications? Further research is needed on this, and perhaps a system of scoring patient criteria based on the need to receive close monitoring in anticipation of post-aSAH complications.

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