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Case Report

Case Series Report: Subarachnoid Hemorrhage and ICU Management

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Abstract

Background : Subarachnoid hemorrhage is a neurological syndrome with complex systemic complications. Rupture of an intracranial aneurysm causes acute extravasation of arterial blood under high pressure into the subarachnoid space and often into the brain parenchyma and ventricles. Bleeding triggers a complex series of events, which can ultimately lead to early brain injury, delayed cerebral ischemia, and systemic complications.
Cases : There were six cases of subarachnoid hemorrhage. Some patients come clinically with severe headache and loss of consciousness. The patient has been treated quickly and aggressively and even put on a ventilator for complications of respiratory failure, support for shock and management of aneurysm clipping and EVD. Rapid and precise diagnosis in the management of patients with SAH is of paramount importance, within the first few hours after the onset of SAH. The risk for early neurologic damage and high rates of severe long-term complications necessitated aggressive early management.
Conclusion : Rapid diagnosis and attentive management of patients with SAH are essential, as early deterioration is possible within the first few hours after the onset of SAH. The risk for early neurologic damage and high rates of severe long-term complications necessitated aggressive early management. Prevention and Management of Complications. The most common complications were pneumonia, aspiration, respiratory failure/distress, sepsis and imbalance electrolyte (hyponatremia). Approximately 50% of deaths after SAH are due to medical complications.

Keywords : Subarachnoid hemorrhage, aneurysm, vasospasm, early brain injury

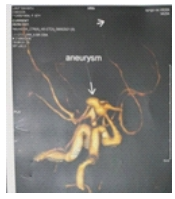
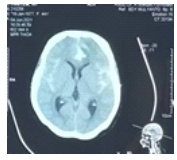
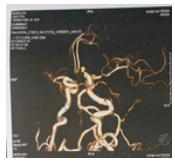
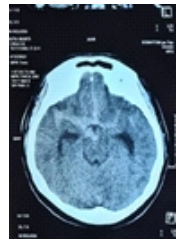
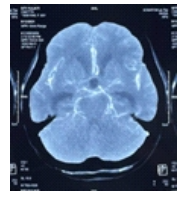
INTRODUCTION

Subarachnoid hemorrhage (SAH) is a mechanical disorder of the intracranial vascular system that causes blood to enter the subarachnoid space.[1] SAH due to aneurysm rupture is a neurological syndrome with complex systemic complications.[1,2] Rupture of an intracranial aneurysm causes acute extravasation of high-pressure arterial blood into the subarachnoid space, brain parenchyma, and ventricles.[3,4] Hemorrhage triggers a series of complex events that can ultimately cause early brain injury, delayed cerebral ischemia, and systemic complications.[5] Although patients with severe SAH (World Federation of Neurosurgical Societies grade 4 and 5) have a higher risk of early brain injury, delayed cerebral ischemia, and systemic complications, early and aggressive management in this patient population has reduced overall mortality rates from over 50% to 35% in the past four decades.[6] Management of SAH poses a unique challenge for neurointensivists, requiring good intensive care unit (ICU) care and neurosurgical intervention techniques. This article discusses several case illustrations of SAH in the neurological ICU and related management given.

CASE REPORT

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Onset	7 day(s) prior to admission	5 day(s) prior to admission	1 day(s) prior to admission	4 day(s) prior to admission	1 day(s) prior to admission	5 day(s) prior to admission
Chronology	Patient had weakness on the left limb and limited mobility for 3 days prior to admission. The patient also complained of headache, drooling, and slurred speech. The patient was then taken to Panti Wilasa Hospital and diagnosed with hemorrhagic stroke. The patient experienced a decreased level of consciousness and was immediately treated with surgery in the Emergency Department at RSDK Hospital. The patient received postoperative care in the Intensive Care Unit.	One day ago, the patient experienced a sudden onset of decreased level of consciousness after daytime activities. The family then brought the patient to RSDK, where a head CT scan revealed intracranial hemorrhage. The patient underwent hematoma evacuation surgery and received postoperative care in the RSDK ICU for recovery after a basal skull craniotomy. .	One day ago, a patient experienced sudden severe headache described as throbbing accompanied by vomiting while engaging in photocopy activity. The patient then took paracetamol to relieve the pain and the symptoms improved. However, during rest, the patient suddenly felt weakness on the right side and experienced a decrease in consciousness.	-4 days ago, the patient complained of severe headache with throbbing pain and vomiting, but remained conscious. The patient then experienced a sudden decrease in consciousness. --A CT scan was performed, revealing intracranial hemorrhage. The patient was then referred to RSDK for further management. -The patient's hypertension was uncontrolled.	-1 day ago, the patient suddenly experienced a decrease in consciousness. Headache (+), seizure (+). -The patient then underwent a head CT scan which revealed intracranial hemorrhage. -The patient was subsequently admitted to the ICU. -DM (-), uncontrolled hypertension, taking amlodipine. -The patient has been experiencing severe headaches for 1 month, accompanied by dark shadows in the right eye.	5 days ago, the patient experienced sudden vertigo and severe headache. The patient felt like the surrounding environment was spinning, with the vertigo occurring frequently and with unpredictable duration. The patient also experienced weakness on the right side of the body and vomiting. -The patient also complained of severe headache throughout the head. According to the family, the patient's speech was incoherent and had difficulty communicating. The patient also felt nauseous and had dry mouth. Urination and bowel movements were within normal limits. -There is a history of uncontrolled hypertension.
GCS and Vital sign	GCS E1M1Vet, BP 133/75, pulse 72x/mins, RR 12x/mins, temp	GCS E1M1Vet, BP 67/46, pulse 88x/mins, RR 12x/mins, temp	GCS E1M1Vet, BP 141/86, pulse 107x/mins reguler, RR 14x/mins,	Moderately ill, GCS E4M6V5, BP 186/75, pulse 78x/mins, RR	Severely ill GCS E4M6V5, BP 151/71, pulse 81x/mins, RR	Severely ill. GCS E4M6V4, BP 117/64 pulse 82x/mins, RR

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
	36.3°C, SpO2 100% assisted with ventilator mode pressure A/C RR 12 PEEP 5 FiO2 60%.	36°C, SaO2 94% assisted with ventilator mode PSIMV PS 5 PEEP 5 FiO2 30%.	temp 36.8°C, SpO2 100% assisted with ventilator SIMV PEEP 5 FiO2 50%, volume 500cc.	22x/mins, temp 36.8°C, SpO2 100% with NRM 7 lpm	20x/mins, temp 36.4°C, SpO2 100% nasal canule 3 lpm	18x/mins, SpO2 97%
Neurological Deficit	Decreased level of consciousness, spastic bilateral hemiparesis, more severe on the left side. Central paresis of the left Nerve VII	Decreased level of consciousness, spastic bilateral hemiparesis, more severe on the right side. Central right-sided facial nerve palsy.	Decreased level of consciousness, bilateral spastic hemiparesis, with more severe symptoms on the right side. Central right-sided paresis of the facial nerve (N VII).	Neurologic deficit-	Right-sided nasal hemianopsia	Right spastic hemiparesis Central right-sided facial nerve palsy (N. VII)
BGA	pH7.435 pCO2 35.7mmHg PO2 102.6mmHg HCO3 17.8 AaDO2 286.8mmHg PFR: 171 Moderate ARDS	pH7.090 pCO2 78.9 PO2 78.2 BEecf-6.4 BE (B)-7.2 SO2c89.6 HCO3 23.4 A-aDO247.7 *PFR 260* Mild ARDS	H(T) 7.376 PCO2(T) 51.3 PO2(T) 130.0 HCO3- 28.9 TCO2 30.4 BE (B) 3.5 SO2c 98.3 A-aDO2 92 PFR 280 Mild ARDS	FIO2 33.0 pH(T) 7.421 PCO2(T) 31.6 PO2(T) 145.4 HCO3- 20.3 TCO2 21.3 SO2c 99.0 A-aDO2 52.9 PFR 439	BGA pH 7.423 pCO2 36.9 PO2 137.1 HCO3 26.5 FIO2 32.0 PFR 428	pH7.328 pCO243.5 PO278.4 FIO2 32.0 pH(T) 7.330 PCO2(T) 43.3 PO2(T)77.9 HCO3- 22.3 TCO223.7 BE (B)-3.5 SO2c94.8 A- aDO298.3 PFR 243 Mild ARDS
Lab	Hemoglobin12.1 Leukocyte 10.600 Procalsitonin0.12 CRP 14.84	Hemoglobin11.2 Leukocyte14.4 CRP 0.26 Natrium 130	-prokalsitonin 3.67 -Leukocytosis 20.300 - CRP 21.08	Leukocyte 14.400 CRP 0.26 Hiponatremia126 Routine urine test: Yeast Cell 1085.4 YEAST: (+)	Leukocyte 21.900 CRP 1.73 ROUTINE URINE TEST YEAST (+) HYPHA (+)	Hemoglobin 11.1g/dL Leukocyte 13.900 CRP 27.62
Grading SAH	Fisher gr IV, WFNS gr 3 Hunt and Hess 5	Fisher gr IV, WFNS gr 3 Hunt and Hess 5	FISHER grade II, WFNS grade 1 Hunt and Hess 3	FISHER grade II, WFNS grade 1 Hunt and Hess 1	FISHER gr III WFNS 1. Hunt and Hess 3	FISHER gr III WFNS 1. Hunt and Hess 3
Radioimaging	Result of Head CT Angiography on 09/06/2021 - The image shows a saccular aneurysm in the proximal right posterior communicating artery with the dome towards the posterolateral direction (length 5.1 mm, width 9 mm, dome 6 mm) that receives supply from the right internal carotid artery at segment C7 (neck 3.2 mm) and the right posterior communicating	The MSCT angiography of the head revealed a saccular aneurysm on the left internal carotid artery segment 7 (size ± AP 0.88 x CC 0.71 x LL 0.85 cm) with a narrow neck (neck size ± 0.34 cm) and a dome directed towards the superolateral left. Suspected ruptured anterior communicating artery aneurysm with subarachnoid hemorrhage.	MSCT ANGIOGRAPHY OF THE HEAD WITH CONTRAST (12/9/2021) FINDINGS: There is a saccular aneurysm seen in the left anterior cerebral artery, segment A1 (size ± AP 0.32 x CC 0.35 x LL 0.39 cm) with a narrow neck (neck size ± 0.16 cm) and dome directed towards the anterosuperior aspect on the left. Intracerebral hemorrhage is observed along	MSCT angiography of the head showed a saccular aneurysm at the branching of the left anterior cerebral artery (A1) and anterior communicating artery (size: AP 0.22 x CC 0.30 x LL 0.53 cm) with a wide neck (visualized neck size + 0.2 cm) and dome towards the anterosuperiorme dial right. There was subarachnoid hemorrhage in the anterior cerebral interhemispheric fissure, Sylvian	The MSCT angiography of the head showed a decreased subarachnoid hemorrhage, intraventricular hemorrhage in the posterior horns of the lateral ventricles and the fourth ventricle, infarction in the right temporoparietal lobe, cerebral edema with signs of increased intracranial pressure, communicating hydrocephalus, pneumocephalus	A head CT scan showed subarachnoid hemorrhage, lacunar infarction in the right and left external capsule, obstructive hydrocephalus, no signs of increased intracranial pressure at present, and right mastoiditis. The CT angiography of the head showed no signs of aneurysms, AVMs, or other vascular abnormalities, subarachnoid hemorrhage,

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
	artery (neck 1.9 mm) accompanied by a daughter saccular aneurysm with the dome towards the posteromedial direction (neck 4.1 mm, dome 6.1 mm) - Dilatation of the left and right posterior communicating arteries with hypoplasia of the left and right posterior cerebral arteries in the segment.		with perifocal edema in the left frontotemporal lobe, which is compressing the left lateral ventricle and causing midline shift to the right (± 0.6 cm). Intraventricular hemorrhage is also present. Subarachnoid hemorrhage is detected. There is a subdural hemorrhage in the left frontoparietotemporal region. Signs of increased intracranial pressure are apparent.	fissure bilaterally, cortical sulci of the frontal and temporal lobes bilaterally, and cavum septum pellucidum. There was also intraventricular hemorrhage in the left lateral ventricle, old infarction in the left corona radiata and anterior limb of internal capsule, ventriculomegaly, and signs of increased intracranial pressure.	in the right frontal region, and subgaleal hematoma in the right frontotemporal region.	intraventricular hemorrhage, hydrocephalus, signs of increased intracranial pressure, or right mastoiditis.
BGA						
Surgical Procedure	The patient underwent craniotomy for hematoma evacuation and also had an external ventricular drain (EVD) placed.	The patient underwent a post craniotomy of the skull base procedure which involved wrapping of the ruptured giant aneurysm located in the left ICA-MCA and placement of an EVD for management of subarachnoid hemorrhage.	Post clipping of ruptured aneurysm at left A. Com (12/9/21) with intracerebral hemorrhage (ICH), subarachnoid hemorrhage (SAH), and intraventricular hemorrhage (IVH) complications, EVD was performed.	Subarachnoid hemorrhage due to the rupture of an aneurysm in the IC-Acom (Internal Carotid-Anterior Communicating) artery, and underwent a clipping surgery to address the issue fixed with VP shunt	Spontaneous subarachnoid hemorrhage due to a ruptured IC-Pcom aneurysm on the right side, with a WFNS score of 1 and a Fisher grade III. The patient underwent a craniotomy for clipping of the aneurysm and also had a lumbar drain placed.	Spontaneous subarachnoid hemorrhage (SAH) due to a ruptured IC Pcom aneurysm on the right side. The aneurysm was subsequently clipped, and the patient underwent a ventriculostomy (EVD) procedure.
Complications	Mild hyponatremia (132) Obstructive hydrocephalus Bronchopneumonia Azotemia (urea level of 94) Seizures Respiratory arrest and cardiac arrest Declared deceased	Bronchopneumonia Hypoalbuminemia Electrolyte imbalance (hypernatremia)	Bronchopneumonia Sepsis Electrolyte imbalance	Electrolyte imbalance UTI	UTI	Electrolyte imbalance UTI

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
	Mild hyponatremia (132) Obstructive hydrocephalus Bronchopneumonia Azotemia (urea level of 94) Seizures Respiratory arrest and cardiac arrest Declared deceased	Bronchopneumonia Hypoalbuminemia Electrolyte imbalance (hypernatremia)	Bronchopneumonia Sepsis Electrolyte imbalance	Electrolyte imbalance UTI	UTI	Electrolyte imbalance UTI
Condition progression	After 6 days of hospitalization, the patient experienced dyspnea followed by 3-5 minutes of generalized tonic-clonic seizures, and subsequently had cardiac arrest. Upon examination, respiratory failure was observed with a heart rate of 120 beats per minute, blood pressure of 87/65 mmHg, SpO2 of 89%, and cold extremities. On the 6th day of hospitalization, the patient was declared deceased.	The patient experienced seizures, fever, desaturation, and eventually cardiac arrest.	On the 40th day of treatment, the patient experienced desaturation, cardiac arrest, and was pronounced dead.	The patient is currently neurologically intact and experiences intermittent headache.	The patient was discharged with improvement. Headache subsided, neurologically intact.	Patient discharged with improvement. Headache present, fully conscious.

DISCUSSION

Subarachnoid hemorrhage (SAH) is a complex neurovascular syndrome with profound systemic effects and is associated with high disability and mortality. Although there has been a decline in mortality rates in the past three decades due to better management strategies, unfortunately, the 30-day mortality and pre-hospital mortality rates remain high. Assessment of onset, initial GCS, Hunt and Hess score, and patient complications play an important role in determining the patient's prognosis outcome.[1] Prevention and management of complications play an important role. The most common complications include pneumonia, aspiration, respiratory failure/distress, sepsis, and electrolyte imbalances (hyponatremia).[3–6] Around 50% of deaths after SAH are caused by medical complications.[5]

In the first, second, and third cases, a long onset assessment, poor initial GCS score, Hunt and Hess score >3, and complications such as blood pressure, hypoxia, fever, and other signs of systemic inflammation associated with mortality suggest the possibility of early brain injury as the cause of poor patient prognosis.[7] The initial GCS upon arrival at the hospital and the Modified Fischer Scale are strong indicators for assessing the severity level at the onset of SAH and also predicting in-hospital mortality, the loss of consciousness at the onset reflects the occurrence of transient intracranial circulation cessation, which is associated with poor outcomes, and the presence of cerebral edema at the onset of SAH.[7–10] Referral factors in the process of transferring patients to tertiary referral hospitals also play a role in determining the mortality rate of patients with SAH, using the Hunt and Hess grading which has been consistently used epidemiologically (Hunt and Hess Table).[11,12] In cases one, two, and three, patients have undergone stabilization management for ABC, TTV, prevention of vasospasm, prevention of rebleeding, and monitoring of clinical signs of increased ICP. They have also received ventilator support for respiratory failure,

TABLE 1
Hunt and Hess

Grading Hunt and Hess grade	Total Mortality	Study Population (%)	Mortality Rate (%)
Mild-moderate headache	12/342	19.5	3.5
Severe headache or cranial nerve palsy	6/186	15.5	3.2
Drowsiness, confusion, or weakness on one side of the body (focal neurological deficit)	30/319	26.6	9.4
Stupor	42/173	14.4	23.6
Coma	127/180	15.0	70.5
Total	216/1200	100.0	18.0

IHK: Immunohistochemistry; MG: Modified Giemsa

support for shock, and EVD management.[2] However, in accordance with the poor initial GCS and Hunt and Hess grade, and increased ICP that impaired the patient's level of consciousness, respiration, and cardiovascular system function, severe disturbances occurred.

In cases of subarachnoid hemorrhage, 66.6% of deaths are determined by brain death and 33.3% are determined by cardiac death. Subarachnoid hemorrhage has a 50% mortality rate during hospitalization. Factors that influence mortality include pre-operative GCS values that are related to Hunt and Hess grading, with Hunt and Hess values > 3 resulting in death in all cases.[13]

In the fourth, fifth, and sixth cases, rapid and accurate diagnosis and management of SAH patients become a very important concern.[2] A good initial GCS upon arrival at the hospital with a score of >13 and a Hunt and Hess score of <3 indicate a good prognostic value. The occurring complications can be well managed. The outcome of SAH can vary significantly, from full recovery to severe disability or death, depending on the severity of the initial bleeding and the potential complications that usually occur within the first 2 weeks after the bleeding. The level of consciousness is considered the most important early predictor of outcome.[1] Patients with a normal level of consciousness have a low risk of mortality. Patients treated with a low level of consciousness have a higher risk of mortality and disability, namely with a Glasgow Coma Scale (GCS) score less than 13, with Hunt and Hess grades 4 and 5.[10] In cases four, five, and six, patients have undergone stabilization management including airway, breathing, and circulation (ABC), monitoring of vital signs, prevention of vasospasm, prevention of rebleeding, and monitoring of increasing signs of intracranial pressure (ICP). Ventilator support was provided for respiratory failure, and shock support was given. In case four, aneurysm clipping and VP Shunt were performed, in case five, aneurysm clipping and lumbar drain were performed, and in case six, aneurysm clipping and EVD were performed.[14,15] Case four, five, and six had a good initial GCS and Hunt and Hess score <3, so the patients showed improvement with mild headache symptoms. The assessment of onset, initial GCS, Hunt and Hess, and patient complications plays an important role in determining the patient's prognosis outcome.[1]

It is still important to note, however, that this report is comprised of a limited sample size of six cases, and thus the prognosis outcome is subject to compounding health factors and statistical variations. Nevertheless, the cases provided shows the significance of prompt and proper initial assessment, while immediate patient management and emergent complications during and after procedure play a significant role to the patients' prognosis outcome.

CONCLUSION

From the 6 cases included in this research, the patients in cases 1, 2, and 3, all of whom showed moderate to severe reduction of consciousness evident by the higher Hunt and Hess score, experienced an increase in mortality. Patients in cases 4, 5, and 6 which showed comparatively lower score experienced a decrease in mortality. The initial assessment of patient's condition is subject to both the severity and extent of symptoms prior to receiving treatment. Nevertheless, in all cases, the first few hours after the onset of SAH carries the risk of early neurological damage and high rates of poor long-term outcomes,[9,16] which underline the need for aggressive early management. The assessment of onset, initial GCS, Hunt and Hess grade and patient complications play an important role in the outcome of the procedure and prognosis of the patient.[17] During the procedure, stabilization of ABC (airway, breathing, circulation) is

crucial in the initial management and control of blood pressure is important to prevent further bleeding, with the use of anti-fibrinolytic drugs and therapy for coagulopathy potentially necessary in some cases.[18] Multimodal monitoring using transcranial doppler, CT angiography, DSA, MRI/MRA, and SPECT.[19,20] In summary, the top priority in the management of SAH is early identification, with the severity measured by all grading parameters used in this report (Hunt and Hess, FISHER, and WFNS) showing a close link to the prognosis outcome. Furthermore, the considerations during patient management proper management are ICP control, complications, and prevention and management of DCI.[14]

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