



Comprehensive Management and Clinical Outcomes in Subarachnoid Hemorrhage: A Case Report

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Article History :

Received date : 2023/12/21
Revised date : 2024/04/10
Accepted date : 2024/05/25
Published date : 2024/08/17



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E-ISSN :

ISSN 3048-1368



P-ISSN :

ISSN 3048-1376



ABSTRACT

Introduction: Subarachnoid hemorrhage is a dangerous neurological condition with sudden onset, often without warning. About half of the patients die before reaching the hospital. The purpose of this case report is to evaluate the management and clinical outcomes of a 59-year-old female patient with SAH.

Case Presentation: A 59-year-old woman presented with sudden, severe headache, nausea, vomiting, and neurological deficits. CT scan revealed subarachnoid hemorrhage. She received treatment to manage intracranial pressure, blood pressure, and prevent vasospasm, with monitoring for complications like hydrocephalus. **Conclusion:** Sudden, severe headache that has never been experienced before is the most common symptom of subarachnoid hemorrhage. Management primarily aims to prevent recurrent bleeding and other complications.

Keyword: subarachnoid hemorrhage, perioperative management, delayed cerebral ischemia

INTRODUCTION

Subarachnoid hemorrhage (SAH), despite representing only 2% to 7% of all strokes, significantly contributes to stroke-related morbidity and mortality due to its occurrence in younger populations and high fatality rates. The primary cause, aneurysmal SAH (aSAH), accounts for 85% of cases, with an incidence of 6 per 100,000 annually in developed countries.¹

Recent decades have seen a decline in SAH incidence, likely due to reduced smoking and improved hypertension management. However, the condition remains more prevalent in Japan and exhibits an unexplained female predominance, with the average age of onset increasing from 52 years in 1973 to 62 years in 2002.^{2,3}

The prognosis of SAH patients is heavily influenced by delayed cerebral ischemia (DCI), a complication that has been challenging to prevent despite advances in critical care and surgical techniques.⁴ Initially, DCI was

thought to result primarily from cerebral vasospasm, but early 21st-century research, including the CONSCIOUS trials, revealed that vasospasm alone does not fully explain DCI, prompting a broader understanding of its multifactorial nature.^{5,6}

Early brain injury (EBI) is now recognized as a critical factor in SAH outcomes, occurring within the first 24 to 48 hours after hemorrhage. EBI involves increased intracranial pressure, cerebral edema, blood-brain barrier disruption, and microcirculatory dysfunction, all of which contribute to the development of DCI.^{7,8} Improved neuroimaging techniques have enhanced the detection and understanding of EBI, showing it to be more prevalent and impactful than previously thought.⁹

Effective management of SAH requires a comprehensive approach, integrating neuroimaging, nimodipine therapy, and surgical interventions like aneurysm clipping or coiling. These strategies aim to prevent rebleeding, mitigate EBI, and

reduce the risk of DCI, all of which are critical to improving patient outcomes.^{10,11}

This case report details the comprehensive management and clinical outcomes of a 59-year-old female patient with SAH, highlighting the complexities of treatment, particularly in addressing EBI and DCI. The case underscores the ongoing challenges in managing SAH despite advances in understanding DCI and EBI. While recent research has improved patient care strategies, continued investigation is crucial to developing more effective treatments and improving long-term outcomes for SAH patients.

CASE PRESENTATION

A 59 year old female patient came to the Emergency Department of Padangan Hospital with a sudden onset of severe headache that began 30 minutes prior to arrival at the hospital. The headache was intense with a pressing sensation and affected the entire head. The patient also experienced nausea and vomited three times after arriving at the hospital.

The patient also denied any previous incidents of fainting or severe headaches. However, a long-standing history of hypertension and diabetes mellitus was noted, although the patient had not been consistent with medication or regular check-ups. There was no history of high cholesterol or other serious illnesses. Upon physical examination, the patient's blood pressure was elevated, and there were signs of neurological deficits, including slightly slurred speech and difficulty responding to conversation. However, the patient denied experiencing any difficulty swallowing, issues with bowel or bladder function, fever, seizures, weakness, or tingling sensations on one side of the body. There was no history of trauma or head injury.

Further examination showed a Glasgow Coma Scale of E3V4M6. The vital signs were as follows: blood pressure of 209/120 mmHg, pulse rate of 84 beats per minute, respiratory rate of 20 breaths per minute, temperature of 36°C, and oxygen saturation of 99% with a nasal cannula delivering 2-4 LPM of oxygen. The general physical

examination was unremarkable. The neurological examination revealed no meningeal signs. The pupils were round and isocoric at 3 mm/3 mm, with positive direct and consensual light reflexes in both eyes. Evaluation of cranial nerve VII (facial nerve) was challenging due to the patient's lack of cooperation. Muscle strength was symmetrical at 4/4/4/4, physiological reflexes were +2/+2/+2/+2, and no pathological reflexes were noted.

Laboratory results showed an elevated white blood cell count of 18,300/uL (reference range: 3,800 – 10,500/uL) and a random blood glucose level of 170 mg/dL. Electrolyte levels, clinical chemistry, arterial blood gas analysis, chest X-ray, and electrocardiography were within normal limits. A non-contrast head CT scan revealed bleeding in the basal system and bilateral temporoparietal lobes. The patient was diagnosed with subarachnoid hemorrhage (SAH).

The patient was subsequently admitted to the Stroke High Care Unit (HCU) with head elevation at 30 degrees. The treatment regimen included KaEn 3B infusion 1000

ml/24 hours IV, Citicoline injection 2x500 mg IV, Ranitidine injection 2x50 mg IV, Metamizole injection 3x1 g IV, Mecobalamine injection 2x500 mg IV, Vitamin B1 injection 2x50 mg IV, Nicardipine pump (4 ampoules in 50 ml PZ) at a rate of 2.3 cc/hour with a target systolic blood pressure of 130-140 mmHg, Paracetamol 3x500 mg, Amlodipine 1x10 mg, and Candesartan 1x16 mg.

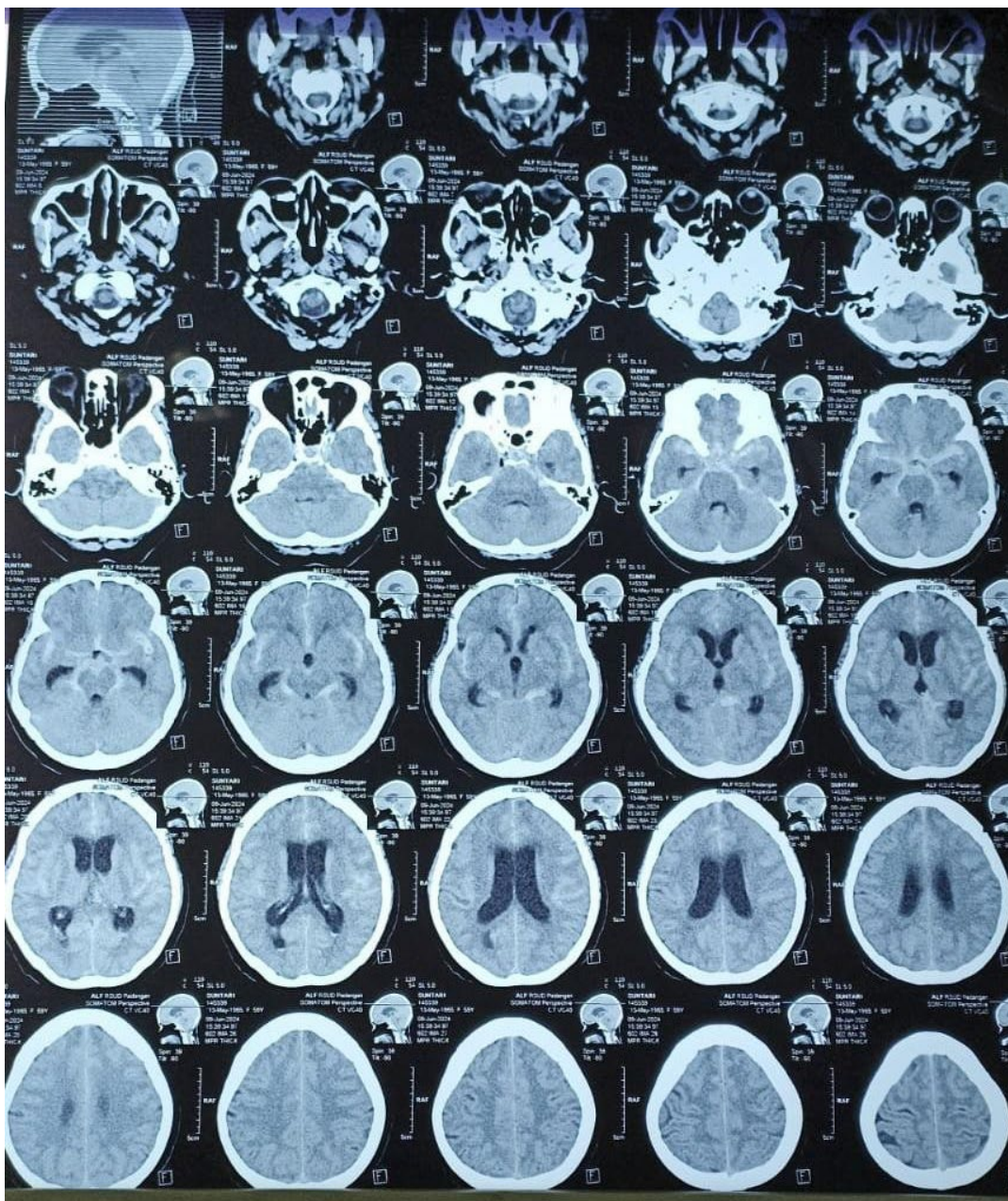


Figure 1. Non-contrast Head CT Scan shows cerebral edema and subarachnoid hemorrhage (SAH) in the basal region and bilateral temporo-lateral lobes.

DISCUSSION

Hemorrhagic stroke, accounting for approximately 15-20% of all strokes, occurs when a vascular lesion ruptures, causing bleeding into the subarachnoid space (subarachnoid hemorrhage or SAH) or directly into brain tissue (intracerebral hemorrhage or ICH).¹² This condition leads to sudden neurological symptoms, often accompanied by severe headaches due to increased intracranial pressure. Spontaneous subarachnoid hemorrhage (SAH) is primarily caused by the rupture of brain aneurysms and is responsible for about 5% of all stroke cases. Approximately 85% of spontaneous SAH cases are due to aneurysm rupture, while the rest may result from non-aneurysmal conditions such as arteriovenous malformations (AVMs).¹³

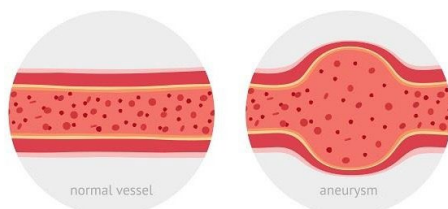


Figure 2. Difference between normal vessel and aneurysm

Epidemiology shows that the incidence of SAH increases with age, with a higher prevalence among women compared to men, especially between ages 45 and 55. Additionally, SAH incidence varies by region and ethnicity, with higher rates observed among Japanese and Finnish populations compared to those near the equator, and African Americans have double the incidence compared to Caucasians. Risk factors include smoking, cocaine abuse, excessive alcohol consumption, and hypertension. Genetic conditions and connective tissue disorders like Marfan syndrome and Ehlers-Danlos syndrome also contribute to aneurysm formation.¹⁴

Pathophysiology of SAH is primarily linked to aneurysm rupture, typically at arterial bifurcations within the Circle of Willis. Ruptures are attributed to either congenital weaknesses in vessel walls or degenerative changes that alter the elasticity of the vessel's intima. Aneurysms averaging 6-7 mm in size are most commonly associated with

rupture. The ruptured aneurysm can cause severe disability and death.¹⁵

Clinical evaluation employs several scales to standardize patient assessment and track clinical changes. The Glasgow Coma Scale (GCS), introduced in 1974, is used to assess neurological function based on eye opening, verbal response, and motor function. The Hunt and Hess scale, World Federation of Neurological Surgeons (WFNS) scale, and Fisher scale help determine the severity and prognosis of SAH based on clinical and imaging findings.¹⁶

Initial management of SAH includes airway stabilization, often necessitating intubation if the GCS is low or the patient cannot maintain airway patency. Blood pressure control is critical, with target systolic pressure recommended to be <160 mmHg or mean arterial pressure (MAP) <110 mmHg. Aggressive blood pressure reduction is avoided to preferred due to its lower associated disability rates compared to surgical clipping. Additionally, external ventricular drainage (EVD) may be required for managing acute

prevent complications like rebleeding, which can increase the risk of ischemia and worsened outcomes. Antihypertensive medications like nicardipine are used, with dosage carefully adjusted. Antifibrinolytics such as tranexamic acid can prevent rebleeding but should be used cautiously due to potential complications like thromboembolism. Supportive care includes pain management, antiemetics, and antiepileptic drugs to address common symptoms and prevent further complications.¹⁷⁻¹⁹

Delayed cerebral ischemia (DCI), occurring 3-14 days post-SAH, is managed with nimodipine, a calcium channel blocker effective in reducing DCI risk.²⁰ The decision between endovascular coiling and surgical clipping for aneurysm repair depends on factors such as aneurysm location, shape, and patient health. Endovascular coiling is generally preferred to avoid hydrocephalus, which can occur in up to 20% of SAH patients.²¹⁻²³

Prognosis for hemorrhagic stroke is influenced by factors such as the volume and location of bleeding, the patient's age, and initial blood

pressure levels. Patients with lower Hunt and Hess and WFNS scores generally have a better prognosis. Severe outcomes are often associated with larger hemorrhage volumes, posterior fossa involvement, older age, and initial GCS scores <4. With

comprehensive management, including intensive care and supportive therapies, some patients show significant improvement, though recovery can be slow and challenging.^{24,25}

TABLE 8 - 1 Glasgow Coma Scale

The GCS is the most common scoring system used to describe the level of consciousness following a brain injury. It is used to help gauge the severity of an acute brain injury. The scale is a simple, reliable, and objective way of recording initial and subsequent neurological function and correlates well with outcome following severe brain injury. The GCS has three subscales that measure the following functions:

Eye opening (E)

- 4 = spontaneous
- 3 = to voice
- 2 = to pain
- 1 = none

Verbal response (V)

- 5 = normal conversation
- 4 = disoriented conversation
- 3 = words, but not coherent
- 2 = no words, only sounds
- 1 = none

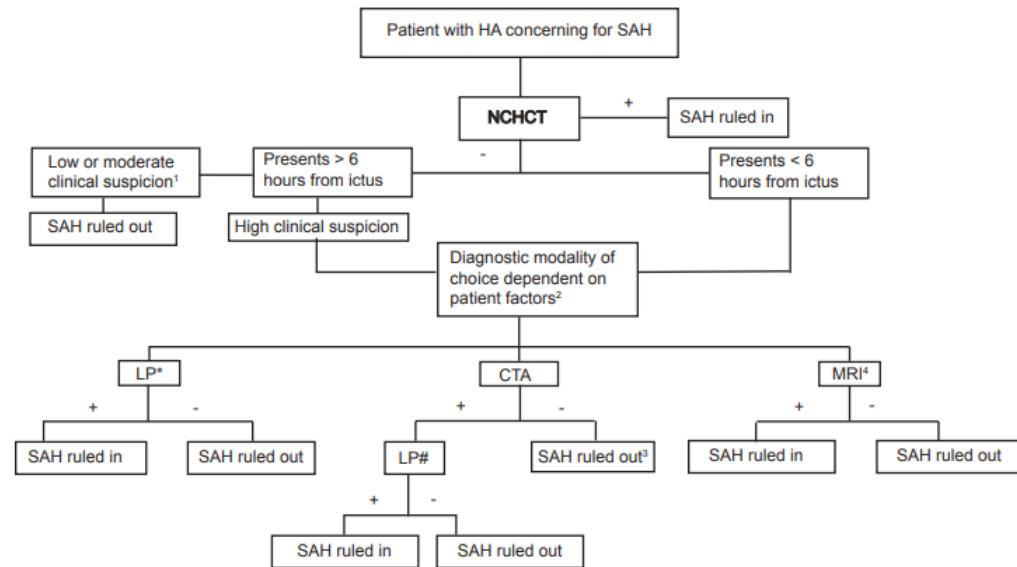
Motor response (M)

- 6 = normal
- 5 = localized to pain
- 4 = withdraws to pain
- 3 = decorticate posture (an abnormal posture that can include rigidity, clenched fists, legs held straight out, and arms bent inward toward the body with the wrists and fingers bent and held on the chest)
- 2 = decerebrate (an abnormal posture that can include rigidity, arms and legs held straight out, toes pointed downward, head and neck arched backward)
- 1 = none

Clinicians use this scale to rate the best eye opening response, the best verbal response, and the best motor response an individual makes. The final GCS score or grade is the sum of these numbers.

Adapted from Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: A practical scale. *The Lancet*, 2(7872), 81–84; Teasdale, G., Murray, G., Parker, L., & Jennett, B. (1979). Adding up the Glasgow Coma Score. *Acta Neurochirurgica Supplementum*, 28(1), 13–16.

Figure 3. Glasgow Coma Scale



HA, headache; SAH, subarachnoid hemorrhage; NCHCT, non-contrast head computed tomography; LP, lumbar puncture; CTA, computed tomography angiography; MRI, magnetic resonance imaging.

¹With criteria met for Perry study [Perry et al. BMJ 2011]⁴⁷

²Patient factors include anticoagulation status, patient willingness to undergo LP, history of lumbar spinal fusion or other surgery, and time from ictus (with longer time favoring MRI)

³Caveat for this strategy includes the potential to miss aneurysms < 4 millimeters

⁴MRI is an acceptable diagnostic at > 24 hours from ictus, prior to this sensitivity is lacking.

^{*}This is the recommended strategy by AHA/ASA, ACEP, and these authors

^{*}Recommended to decrease the false positive rate of CTA.

Figure 4. PSA diagnosis algorithm for patients with sudden severe headache

Table 1. Hunt and Hess Score

Score	Description	Survival
1	Asymptomatic, or mild headache, mild neck stiffness	70%
2	Severe headache with meningeal signs and possible cranial nerve deficit	60%
3	Decreased consciousness with mild focal neurological deficit	50%
4	Stupor, moderate to severe hemiparesis, early decerebration	20%
5	Deep coma, decerebration	10%

Table 2. World Federation of Neurological Surgeons Score

Score	GCS	Clinical Symptoms
1	15	No motor deficit (-)
2	13-14	No motor deficit (-)
3	13-14	Motor deficit present (+)
4	7-12	Motor deficit ($\pm$), may or may not be present
5	3-6	Motor deficit ($\pm$), may or may not be present

CONCLUSION

Subarachnoid hemorrhage (SAH), primarily due to aneurysmal rupture, poses significant morbidity risks, notably through delayed cerebral ischemia (DCI). Perioperative management focuses on meticulous blood pressure regulation, nimodipine therapy, and selecting appropriate aneurysm repair to optimize patient outcomes.

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