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**Case Information:** A 43 years old Thai male

**Domicile:** Pathum Thani

**Healthcare coverage:** ประกันสุขภาพถ้วนหน้า

**Admitted date:** 11/5/64

**Reliability:** 90% (Data obtained from medical records)

**CC:** Weakness of left limbs with sudden severe headache 1 hour PTA

**PI:**

1.5 hour PTA- Last seen normal

1 hour PTA- Patient had sudden severe headache with nausea and 1 episode of vomiting. The patient then experienced weakness in his left arm and left leg. He also had tongue stiffness and dysarthria. There was no seizure, no loss or alteration of consciousness and no fever or any other associated symptoms.

**Past history:**

Underlying disease: no known underlying disease

Surgical history: none

Food/drug allergy: none

Current medication: none

**Family history:** no family history of DM,HT,DLP, malignancy, hematologic disease

**Social history:** no information on smoking and alcohol drinking

**Physical examinations:**

V/S: BP: 200/110, BT 37, PR110, RR22

Measurements: BW 65kg, Height 170cm, BMI 22

GA: A Thai male, alert, good consciousness, not pale, no jaundice, no dyspnea

HEENT:

Head: no traumatic lesion, normal head contour, normal scalp and hair

Eyes: no pale conjunctiva, anicteric sclera, no sunken eyeballs

Ears: normal ear pinna, no discharge/blood from ear canal

Nose: no discharge/blood from nasal cavity

Neck: no palpable cervical lymph nodes

CVS: no surgical scar, no active precordium, no heave, no thrill, normal S1,S2, no murmur, capillary refill < 2 secs

RS: normal chest contour, symmetrical chest movement, trachea in midline, resonance on percussion both lungs, clear and equal breath sound both lungs, no adventitious sound, no accessory muscles used

GI: no surgical scar, no abdominal distension, normoactive bowel sound, soft, no guarding, not tender, no mass

Extremities: no deformities, no joint swelling, no edema, no rash

CNS:

Cranial nerves:

- CNI: not tested
- CNII: normal visual acuity, visual field intact
- CNIII,IV,VI: no nystagmus, full EOM
- CNV: no facial sensation loss
- CNVII: no facial palsy
- CNVIII: normal hearing
- CNIX,X: gag reflex present
- CNXI: not tested
- CNXII: not tested

Stiff neck: positive

Babinski's sign: no response

GCS: E4V5M6, pupils 2mm RTLBE

Motor power:

Right limbs: grade V/V

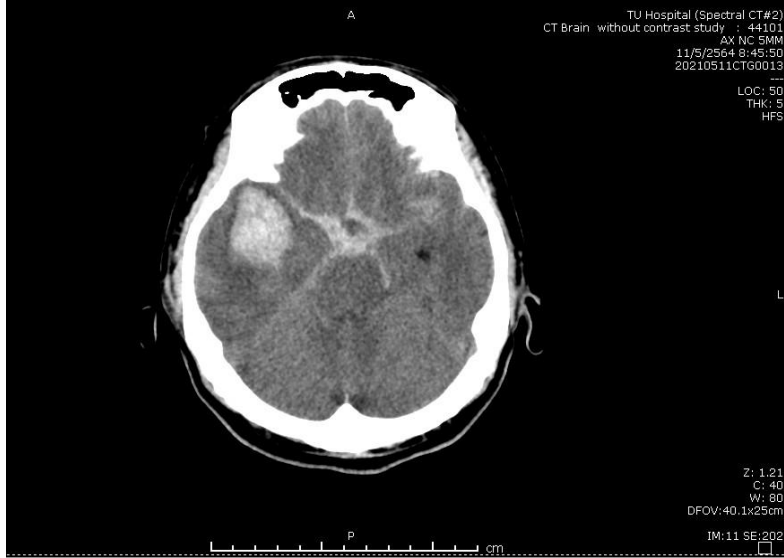
Left limbs: grade I/II

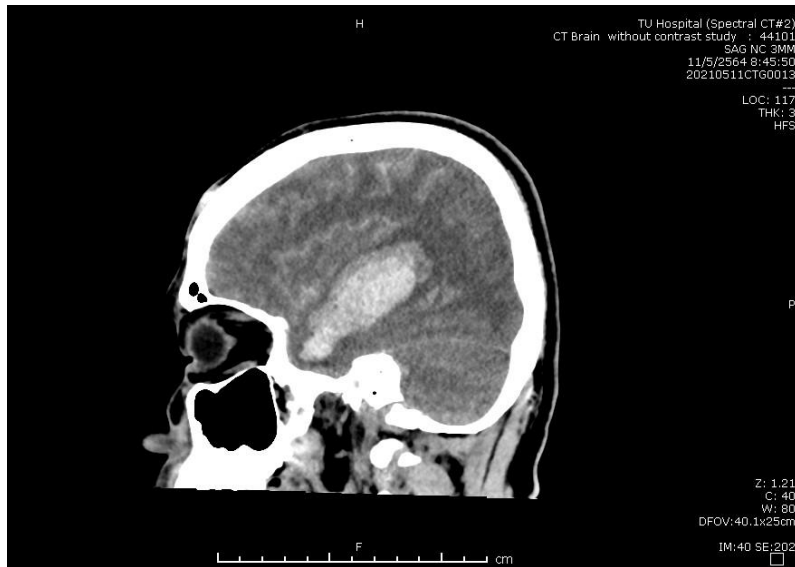
DTR 1+ all areas

### **Investigations:**

#### **CT brain non-contrast:**

- Intact bony structure
- Unremarkable paranasal sinuses, orbits and mastoid air cells
- Diffuse swelling of right cerebral hemisphere
- Midline shifted to the left (0.7cm)
- Intraventricular hemorrhage in bilateral lateral ventricles, foramen of Monroe, third ventricle, cerebral aqueduct and fourth ventricle
- Irregular shaped hyperdense lesion (intracerebral hemorrhage) with mild perilesional edema at white matter of right temporal lobe, size 4.7x3.6x4.3cm
- Diffuse SAH at suprasellar cistern and right sylvian fissure
- Right to left subfalcine herniation and right uncal herniation





**Chest x-ray:** no infiltration

**EKG 12 leads:** normal sinus rhythm, rate 111, no axis deviation, no ST elevation

**Laboratory results:**

- CBC: Hb 12.3, Hct 38.0, MCV 80.4, WBC  $21.1 \times 10^3$ , PMN 71.1, Lymphocyte 17.2, Eosinophil 0.10, basophil 0.80, Platelet  $322 \times 10^3$
- Coagulogram: PT 12.2, PTT 25.1, INR 1.10
- BUN, Cr, Electrolytes: BUN 12, Cr 0.96, Na 141, K 3.9, Cl 113, Bicarbonate 14
- Liver function test: Total protein 7.63, Albumin 4.25, Globulin 3.38, Total Bilirubin 0.46, Direct Bilirubin 0.10, AST 28, ALT 22, ALP 62
- Anti-HIV: negative
- HBsAg: negative
- Anti-HCV: negative
- Swab for Covid-19: negative

**Problem list:** Subarachnoid hemorrhage

**Discussion**

Although this patient does not possess any risk factors for aneurysmal subarachnoid hemorrhage, his clinical presentation is highly suggestive of such a condition.

Firstly, the patient presented to the hospital with a sudden onset severe headache, described as “unbearable”, which is a very typical presentation of SAH. To further support this, the patient reported a history of physical activity before the onset of the headache, which could be the trigger for aneurysm rupture. Also, upon physical examination, he was tested positive for stiff neck, and his vitals showed elevated blood pressure of 200/110 mmHg along with pulse rate of 110 bpm. Presence of left-sided hemiparesis (motor power grade I and II for arm and leg, respectively) suggests that the lesion is at the right hemisphere, specifically the subcortical area.

On the other hand, the patient did not experience a sentinel headache before the onset of the aneurysmal rupture, and had no risk factors contributing to the diagnosis, which is atypical. Thus, further investigation through CTA imaging could prove useful.

### Further investigation:

- CTA brain: Rupture right middle cerebral artery aneurysm



### Management:

- Admit to ICU Neurosurgery ward
- NPO
- Record vital signs, intake and output
- DTX q 6 hrs, keep 100-180mg%
- Elevate head 30 degrees
- Absolute bed rest
- Group match LPRC 4 units, FFP 4 units
- NSS 1000ml IV rate 80ml/hr
- Medications:
  - Dilantin 100mg IV q 8 hrs
  - Losec 40 mg IV OD
  - Nicardipine (20mg + NSS 100ml) rate 25ml/hr titrate, keep BP<140/90mmHg
  - Morphine 3 mg IV prn q 4 hrs
  - Plasil 10 mg IV prn q 4 hrs
- Set OR for right craniotomy with aneurysm clipping with clot removal

## Introduction

Subarachnoid hemorrhage (SAH) is defined as the presence of bleeding in subarachnoid space; this space is bounded by the pia mater and the arachnoid mater. The hemorrhage can be either spontaneous (non-traumatic) or traumatic; evidence illustrates that the latter aetiology is more common. Aneurysmal hemorrhage is the main cause of spontaneous subarachnoid hemorrhage.

## Epidemiology

Overall, the incidence of SAH is 9/100,000 person per year. The incidence also increases with age (peak at 50-60 years old), female gender, and is more common in winter than in the summer. In terms of case mortality, this has evidently declined between the years 1980 and 2005 from 50% down to 35%; this may be due to better diagnostic tools, ability to perform early repair of ruptured aneurysm, and improvement in medical management.

Modifiable Risk Factors	Non-modifiable Risk Factors
<ul style="list-style-type: none"><li>● Smoking</li><li>● Hypertension</li><li>● Heavy alcohol intake</li><li>● Low serum cholesterol</li></ul>	<ul style="list-style-type: none"><li>● Increase age</li><li>● Female sex</li><li>● Autosomal Dominant Polycystic Kidney Disease</li><li>● Family history of SAH (Two first degree relative with SAH)</li></ul>

## Clinical Presentation

Sentinel headache (sudden severe headache) occurring days to weeks before aneurysm rupture may represent warning leak (minor hemorrhage consisting of around 35 ml of blood).

The most typical symptom of aneurysmal SAH is sudden severe headache with no specific localisation, often expressed as the “worst headache of my life”, usually reaching its peak intensity within the first hour after onset. Clinical suspicion of SAH is further supported if the aforementioned symptom is associated with prior physical activities or emotional stress. Other associated symptoms include loss of consciousness, vomiting, neck pain and stiffness. It is important to note that 22% of SAH patients die before reaching the hospital.

Moreover, bleeding location, presence or absence of hydrocephalus, elevated intracranial pressure, ischemia, infarction, or hematoma all play a role in determining neurologic signs that may be detectable. Involvement of the third cranial nerve may produce loss of pupillary light reflex. Preretinal hemorrhage in Terson syndrome predicts poorer prognosis.

## Diagnosis

All patients suspected of aneurysmal SAH are recommended to undergo initial brain CT scan without contrast. The detection rate of SAH depends on the volume and location of the bleeding. This CT scan would also provide information about the risk of possible complications of SAH, including angiographic vasospasm and delayed cerebral ischemia; the Fisher Scale is an example of a grading system developed to systematically classify CT scan findings in order to predict an individual’s prognosis.

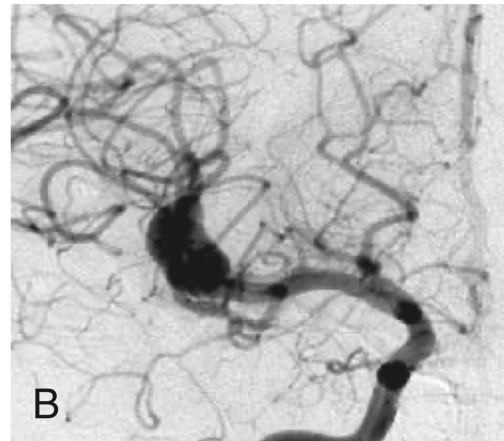


Lumbar puncture is indicated if the initial CT scan came out negative, provided that patient has no contraindications to undergo lumbar puncture. These contraindications are:

- abnormal blood clotting
- increased intracranial pressure as a result of a space-occupying lesion
- suspected arteriovenous malformation
- infection at puncture site

Presence of erythrocytes in the CSF confirms hemorrhage, though traumatic tapping must be ruled out.

In the same way, if both CT scan and lumbar puncture provide negative results for SAH, CTA or catheter digital subtraction angiography (DSA) is recommended.



**A, CT scan** demonstrating an aneurysm in the right basal cistern (*asterisk*)

**B, Angiography** showing a proximal complex fusiform middle cerebral artery aneurysm

## Initial Management of Aneurysmal Subarachnoid Hemorrhage

1. **Stabilization:** ABCD
  - maintain patent airway, breathing, cardiovascular function, and controlling seizure
  - If GCS  $\leq$  8, intubate patient with endotracheal tube
2. **Severity grading:** perform as soon as possible, since it acts as a predictor of neurologic complications and outcome
  - Hunt and Hess grading based on initial neurologic examination

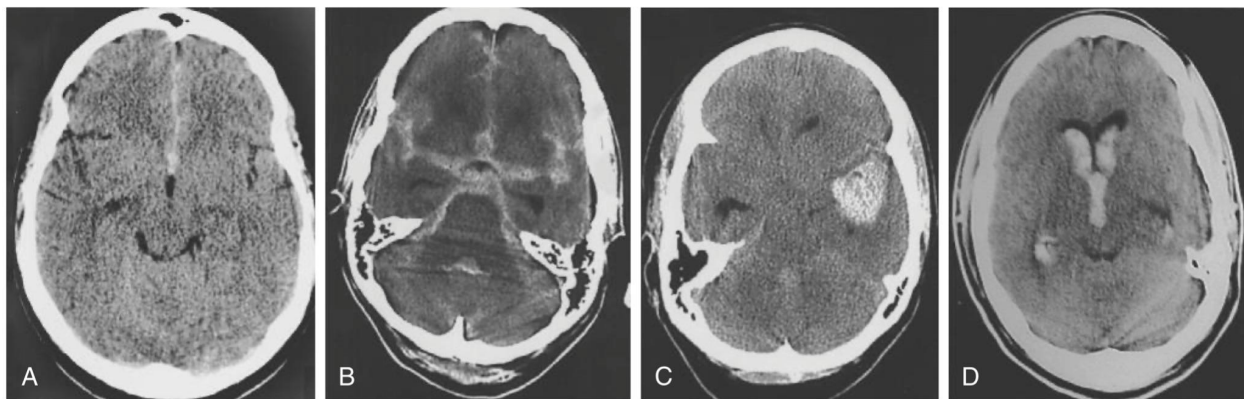
Grading	Neurologic status
0	Unruptured aneurysm
I	Asymptomatic or mild headache and slight nuchal rigidity
II	Moderate to severe headache, nuchal rigidity, no neurologic deficit except cranial nerve palsy
III	Drowsy, confused, or mild focal neurologic deficit
IV	Stupor, mild or severe hemiparesis, possible early decerebrate rigidity, vegetative disturbance
V	Deep coma, decerebrate rigidity, moribund appearance

- World Federation of Neurological Surgeons grading scale

Grade	GCS score	Motor deficit
I	15	Absent
II	13-14	Absent
III	13-14	Present
IV	7-12	Present or absent
V	3-6	Present or absent

- Fisher grading based on amount of blood seen on CT scan (predict risk of vasospasm)

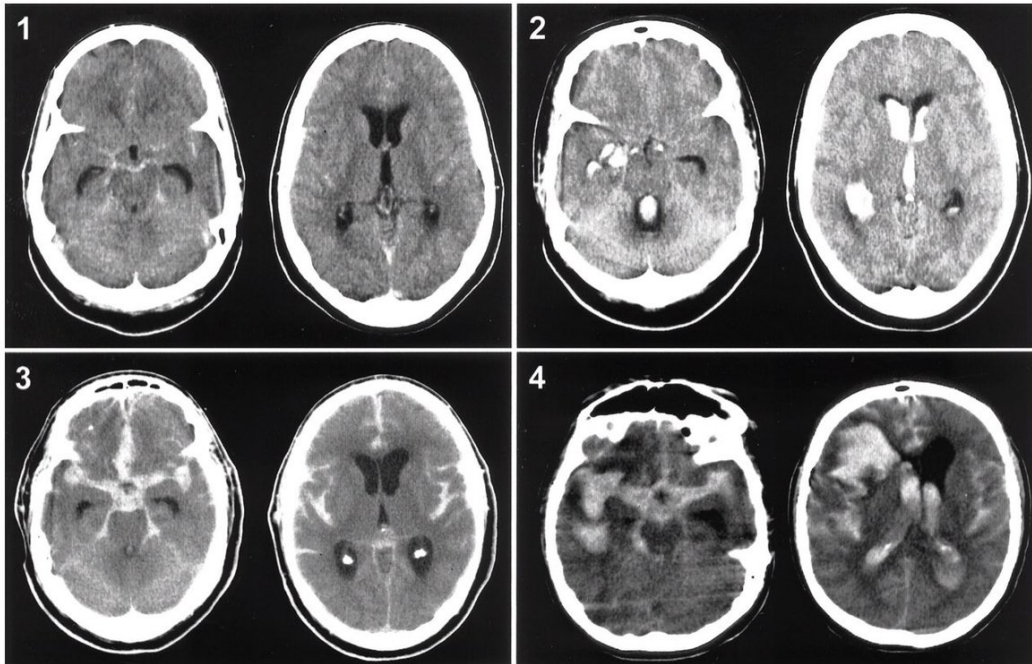
Grade	CT scan findings
1	No blood
2	Diffuse thin layer of subarachnoid blood (vertical layer <1 mm)
3	Localised clot or thick layer of subarachnoid blood (vertical layer $\geq$ 1 mm)
4	Intracerebral or intraventricular blood with diffuse or no subarachnoid blood



Fisher grading: **A**, grade 2; **B**, grade 3; **C** (intracerebral hemorrhage) and **D** (intraventricular hemorrhage), grade 4.

- Modified Fisher grading based on CT scan findings

Grade	CT scan findings				Description
	No SAH	Focal or diffuse thin SAH	Focal or diffuse thick SAH	IVH	
0	+	-	-	-	No SAH; no IVH
1	-	+	-	-	Thin diffuse/focal SAH; but no IVH
2	-	+	-	+	Thin diffuse/focal SAH with IVH
3	-	-	+	-	Thick focal/diffuse SAH; but no IVH
4	-	-	+	+	Thick focal/diffuse SAH; with IVH



**Figure: Modified Fisher grading**

**Grade 1:** diffuse thin SAH without bilateral IVH

**Grade 2:** thin SAH with bilateral IVH

**Grade 3:** thick cisternal clot without bilateral IVH

**Grade 4:** Thick cisternal clot with bilateral SAH

Reproduced from Claassen J, Bernardini G, Kreiter K et al. Effect of Cisternal and Ventricular Blood on Risk of Delayed Cerebral Ischemia After Subarachnoid Hemorrhage. Stroke. 2011;42(9):2012-2020. doi:10.1161/hs0901.095677

### 3. Admit and refer to specialists

#### 4. Acute care

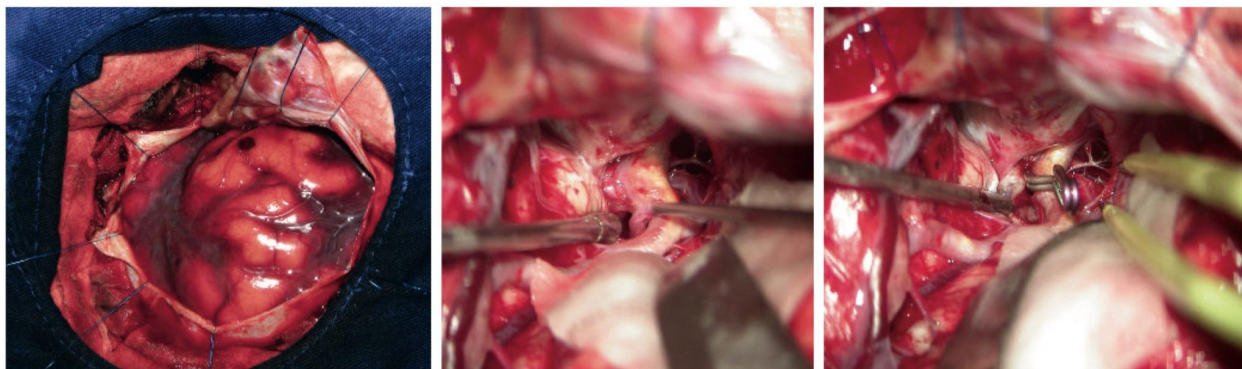
- Control blood pressure: mainly prevents “rapid variation in BP”
  - Prior to repair: keep normotensive SBP <160-180 mmHg (prevent rebleed) but >100 mmHg (prevent cerebral ischemia/stroke) by labetalol, hydralazine or nicardipine
  - After aneurysm repair: no treatment is needed to control hypertension
- Antithrombotic reversal: discontinue all antithrombotics and anticoagulant until definitive treatment with surgery or coiling is executed
  - Give platelet transfusion prior to surgery in patient who is thrombocytopenic (platelet count <100,000) or received antiplatelets
  - Warfarin: reversed by using vitamin K, FFP, or prothrombin complex
  - DOACs: use specific antidote
- DVT prophylaxis:
  - Not generally necessary in Asian population (less likely occurred than in caucasians)

- Prior to surgery: intermittent pneumatic compression device or graduated compression stocking
- 24 hrs after surgery: start UFH or LMWH
- Seizure prophylaxis: controversial
  - For treatment (not prophylaxis): Phenytoin 15-19 mg/kg IV load (max 50 mg/min) then 100 mg IV/PO q 8 hr, phenobarbital, carbamazepine, or levetiracetam
- Maintain euvolemia:
  - mainly use normal saline solution,
  - target normal electrolytes,
  - monitor input and urine output, and serum sodium daily
- Nimodipine: prevent vasospasm
  - Given to all patients (60mg q 4 hr) enterally
  - Duration: 21 days, Adjust dose to avoid hypotension
- Pain control: recommend starting with paracetamol → tramadol, codeine → morphine
- General care:
  - Bed rest until the aneurysm is obliterated or several days from hemorrhage
  - Elevate head 30 degrees
  - Restrict visitors (avoid unnecessary stimulation)

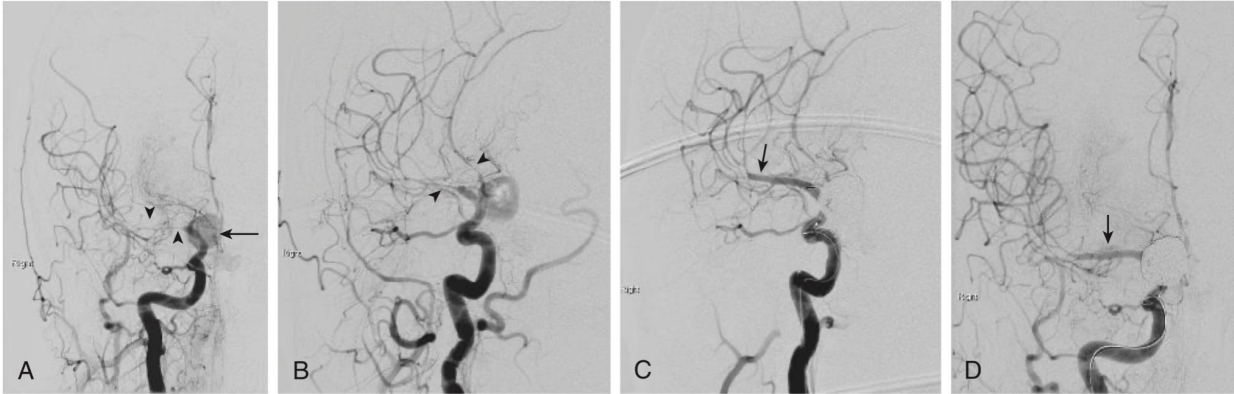
#### **Aneurysm treatment (AHA/ASA class I recommendations)**

'Surgical clipping or endovascular coiling' to completely obliterate the aneurysm should be performed as early as feasible (improved outcome if performed within 24 hours) to reduce the risk of rebleeding.

Antifibrinolytics should be avoided, despite definitive treatment not being plausible, since it increases the risk of cerebral infarction.



Picture above demonstrates the intraoperative view of **left carotid-posterior communicating artery aneurysm** and **surgical clipping** being performed to repair the aneurysm



**A**, an **arrow** pointing at a right-sided posterior communicating artery aneurysm

**A and B**, **arrowheads** indicating right middle cerebral artery vasospasm and anterior cerebral artery narrowing

**C and D**, aneurysm was occluded via **endovascular coiling** technique

**C and D**, **arrows** revealing **balloon angioplasty** performed onto the M1 segment of the middle cerebral artery

## Specific complications of subarachnoid hemorrhage

1. Rebleeding:
  - Untreated ruptured aneurysm has the risk of rebleeding of 4% in the first 24 hours, then 1.5% per day, until 19% in the first 2 weeks.
2. Vasospasm and delayed cerebral ischemia
  - Incidence of angiographic vasospasm in ruptured aneurysm is 50-90%
  - **Risk factor:**
    - i. large volume of persistent subarachnoid clot (higher risk being associated with higher grade in **Modified Fisher scale**),

Modified Fisher Grading	Risk for Vasospasm
0: no SAH or IVH	Very low risk
1: focal or diffuse thin SAH, no IVH	Low risk
2: focal or diffuse thin SAH with IVH	Moderate risk
3: Focal or diffuse thick SAH, no IVH	High risk
4: Focal or diffuse thick SAH with IVH	Very high risk

- ii. loss of consciousness,
  - iii. poor neurological status on admission,
  - iv. cigarette smoking, DM, hyperglycemia, and pre-existing hypertension
- Occur within 2 weeks after rupture, no earlier than day 3, and peak at day 7
  - Prevention of cerebral vasospasm

Prevention of Cerebral Vasospasm	
General measures	Augmentation of subarachnoid clot clearance
Endovascular coil repair/ microsurgical clipping	Clot lysis (intracisternal rtPA or urokinase)
Maintain euvolemia ( $\geq 3$ L/day of isotonic fluids)	Lamina terminalis fenestration (more preferable than clot lysis)
Prevent anemia (Hb $< 9$ g/dL)	Lumbar CSF drainage
Prevent hyponatremia	"Head shaking"
Maintain normal to high SBP	Combined treatment
Optimize ventilation and oxygenation	
Prevent high ICP (and maintain CPP) with EVD	
Nimodipine 60mg q 4 hr (by mouth/NG tube)	

- Management of vasospasm
- Keep normovolemia (CVP 8-10 mmHg, PCWP 14-16 mmHg), Hb >9 g/dL, SpO2 >95%, optimal intracranial pressure with proper EVD position
- **Triple-H therapy**: Hypervolemia, Hypertension (induce 20mmHg increase in SBP, then titrate according to clinical response using NE/phenylephrine), and Hemodilution
- Symptomatic vasospasm & moderate to severe angiographic vasospasm in comatose patients => vasopressor infusion → if fail to improve: “angioplasty” (endovascular treatment)
- Indications for early balloon angioplasty:
  - Proven large artery angioplasty
  - No new cerebral infarction or hemorrhage
  - No or minor clinical response to SBP >200 mmHg
  - Unstable cardiopulmonary status

### 3. Hydrocephalus

- Incidence following SAH is 20%
- Indication of EVD insertion: ventriculocranial ratio 20-25% greater than P95 for age with depressed level of consciousness

Upper 95% Confidence Value for Ventriculocranial Ratio, by Age	
Age	95% Confidence Value
<30	0.16
<50	0.18
<60	0.19
<80	0.21
<100	0.25

### 4. Intraventricular hemorrhage (IVH)

- Intraventricular thrombolysis with rTPA may accelerate clearance of the hemorrhage and ICP reduction in large aneurysmal IVH
- Ruptured aneurysm should be repaired prior to administration of fibrinolytic drugs

### 5. Increased intracranial pressure: can occur with/without hydrocephalus

- Management: intracranial hematoma removal, ventricular drainage, assisted ventilation, sedation, pharmacological paralysis, maintenance of normal body temperature and sodium, seizure prophylaxis, 20% mannitol/ hypertonic saline boluses, short period of hyperventilation
- Decompressive craniectomy can also reduce ICP
- Maintain CPP above 70 mmHg



6. Intracerebral hemorrhage
  - It is most commonly arising from distal anterior cerebral artery, middle cerebral artery and anterior communicating artery aneurysm.
  - Treatment: Craniotomy for hematoma evacuation (indicated in deteriorating level of consciousness with/without signs of herniation) with surgical clipping performing simultaneously.
7. Seizure
  - Prophylactic antiepileptic drug use is not recommended.
  - Antiepileptic agent is used in patient who has seizure before or to immediately terminate the seizure

### Neurological complications of subarachnoid hemorrhage

Complication	Death	Disability
Rebleeding	6.7	0.8
Vasospasm	7.2	6.3
Hydrocephalus	0.3	1.4
Direct effect of SAH	7.0	3.6
Intracerebral hemorrhage	1.0	1.0
Complication of intracranial surgery	1.7	2.3
Other	2.0	1.2

### Recurrence, Outcome and Prognosis

Patients diagnosed with aneurysmal SAH are at risk of recurrent SAH despite having had complete obliteration of the current aneurysm.

Initial and recurrent rebleeding is most responsible for mortality observed in SAH patients within the first 30 days after aneurysm rupture. Studies have reported 0.2% rate of rebleeding for coiling and 0.02% for clipping after the first year. Neurocognitive impairment and epilepsy are the common morbidity seen in SAH survivors.

It is evident through a systematic review that prognosis of SAH is determined based on neurological status, age, aneurysm repair modality, Fisher grade of CT clot burden, premorbid history of hypertension, aneurysm size, and aneurysm location. Thus, evaluation of the aforementioned factors could provide useful information for the patient as well as their relatives in terms of long term clinical outcome.

## References

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[https://www.uptodate.com/contents/aneurysmal-subarachnoid-hemorrhage-epidemiology-risk-factors-and-pathogenesis?search=subarachnoid-hemorrhage&source=search\\_result&selectedTitle=5~150&usage\\_type=default&display\\_rank=5](https://www.uptodate.com/contents/aneurysmal-subarachnoid-hemorrhage-epidemiology-risk-factors-and-pathogenesis?search=subarachnoid-hemorrhage&source=search_result&selectedTitle=5~150&usage_type=default&display_rank=5). Published 2021. Accessed June 11, 2021.