

Sub Arachnoid Hemorrhage

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Disclosures

- None



Epidemiology

- ~ 3% of all strokes
- Incidence : 2-16/1 lakh population
- Women > men
- Incidence increases with advancing age ; mean age of detection 50 yr
- 80% have a cerebral aneurysm, 15% - no source of bleed, 5% - AVM, vasculitis.
- Median mortality ~ 30%, steadily decreasing
- Significant morbidity – physical, cognitive, mood



Risk factors for SAH

- **Nonmodifiable Risk Factors**
  - Age
  - Female sex
  - Prior history of aneurysmal subarachnoid hemorrhage
  - Family history of subarachnoid hemorrhage
  - History of aneurysm in first-degree relatives (especially in two or more relatives)
- **Modifiable Risk Factors**
  - Hypertension
  - Cigarette smoking
  - Heavy alcohol use
  - Sympathomimetic drug use (eg, cocaine)
- **Other**
  - Certain genetic disorders (eg, autosomal dominant polycystic kidney disease, type IV Ehlers-Danlos syndrome)
  - Anterior circulation aneurysms are more likely to rupture in patients who are younger than 55 years of age
  - Posterior circulation aneurysms are more likely to rupture in men
  - Significant financial or legal problems within the past 30 days
  - Cerebral aneurysms of more than 7 mm in diameter



Clinical features

- Sudden and severe headache
- Nausea, vomiting, photophobia
- Neck rigidity
- Loss of consciousness
- Atypical features – Seizures, acute encephalopathy
- Sentinel headache



Focal physical findings in SAH

Findings	Likely Cause
Third nerve palsy	Usually posterior communicating aneurysm; also posterior cerebral artery and superior cerebellar artery aneurysms
Sixth nerve palsy	Elevated intracranial pressure (false localizing sign)
Combination of hemiparesis and aphasia or visuospatial neglect	Middle cerebral artery aneurysm, thick subarachnoid clots, or parenchymal hematomas
Bilateral leg weakness and abulia	Anterior communicating artery aneurysm
Ophthalmoplegia	Internal carotid artery aneurysm impinging upon the cavernous sinus
Unilateral visual loss or bitemporal hemianopia	Internal carotid artery aneurysm compressing optic nerve or optic chiasm
Impaired level of consciousness and impaired upward gaze	Pressure on the dorsal midbrain due to hydrocephalus
Brainstem signs	Brainstem compression by basilar artery aneurysm
Neck stiffness	Meningeal irritation by the presence of subarachnoid blood
Retinal and subhyaloid hemorrhages	Sudden increase of intracranial pressure
Preretinal hemorrhages (Terson syndrome)	Vitreous hemorrhage due to severe elevations of intracranial pressure

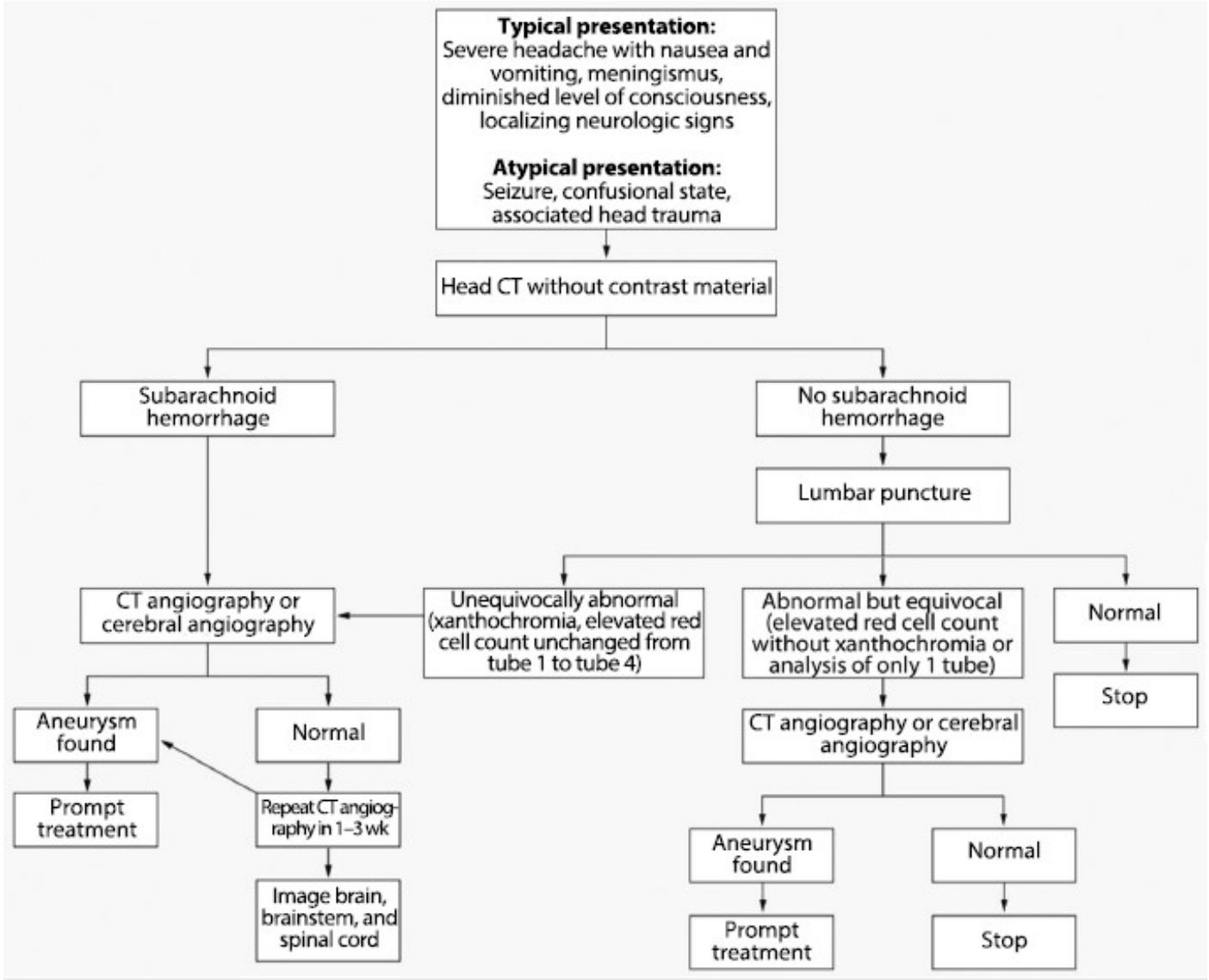


Diagnosis

- Non-contrast CT : 98-100% sensitivity to detect subarachnoid blood at 24 hrs, 93% @ 24 hrs and 50% @ 7 days
- Lumbar puncture
- CT Angiography/ DSA

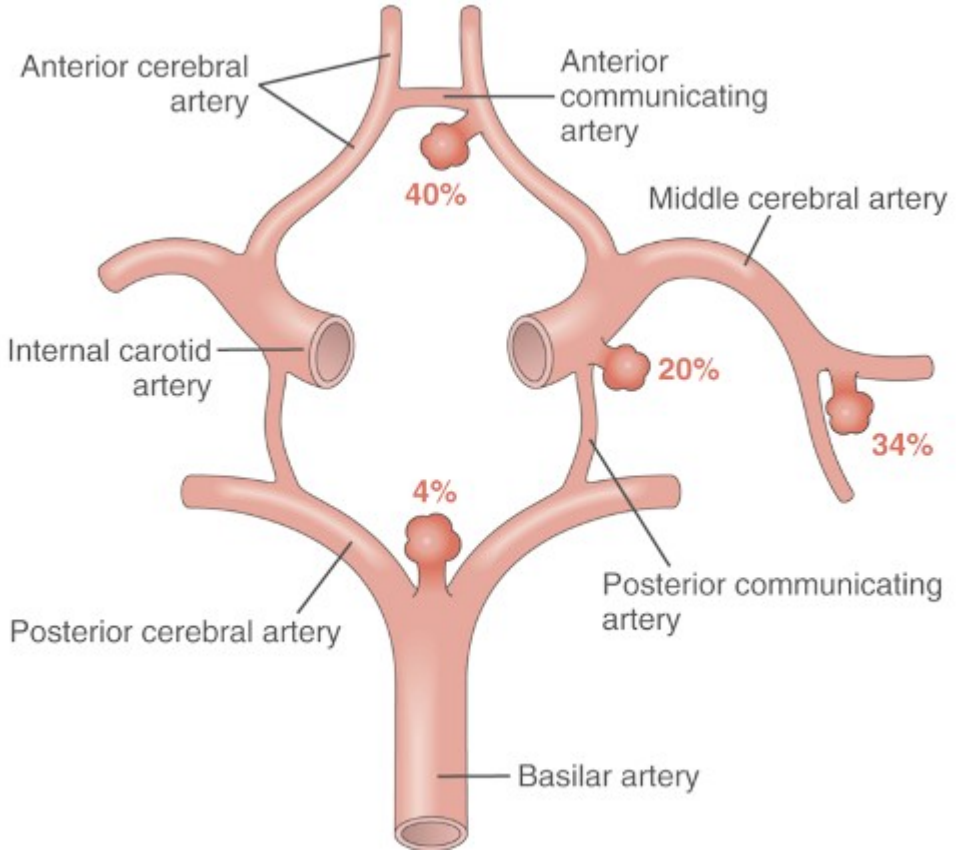


Diagnostic Algorithm

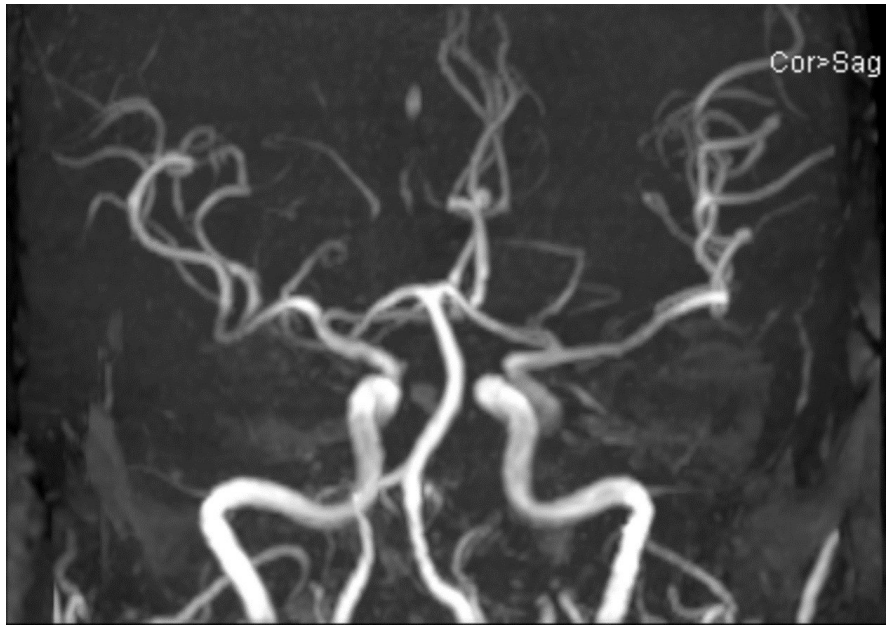


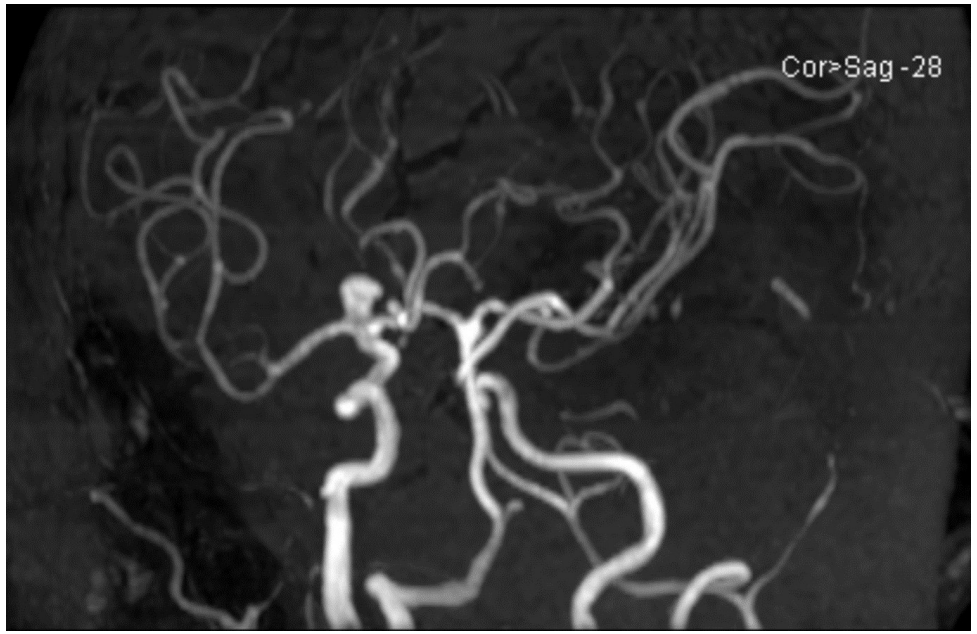


Cerebral arterial anatomy

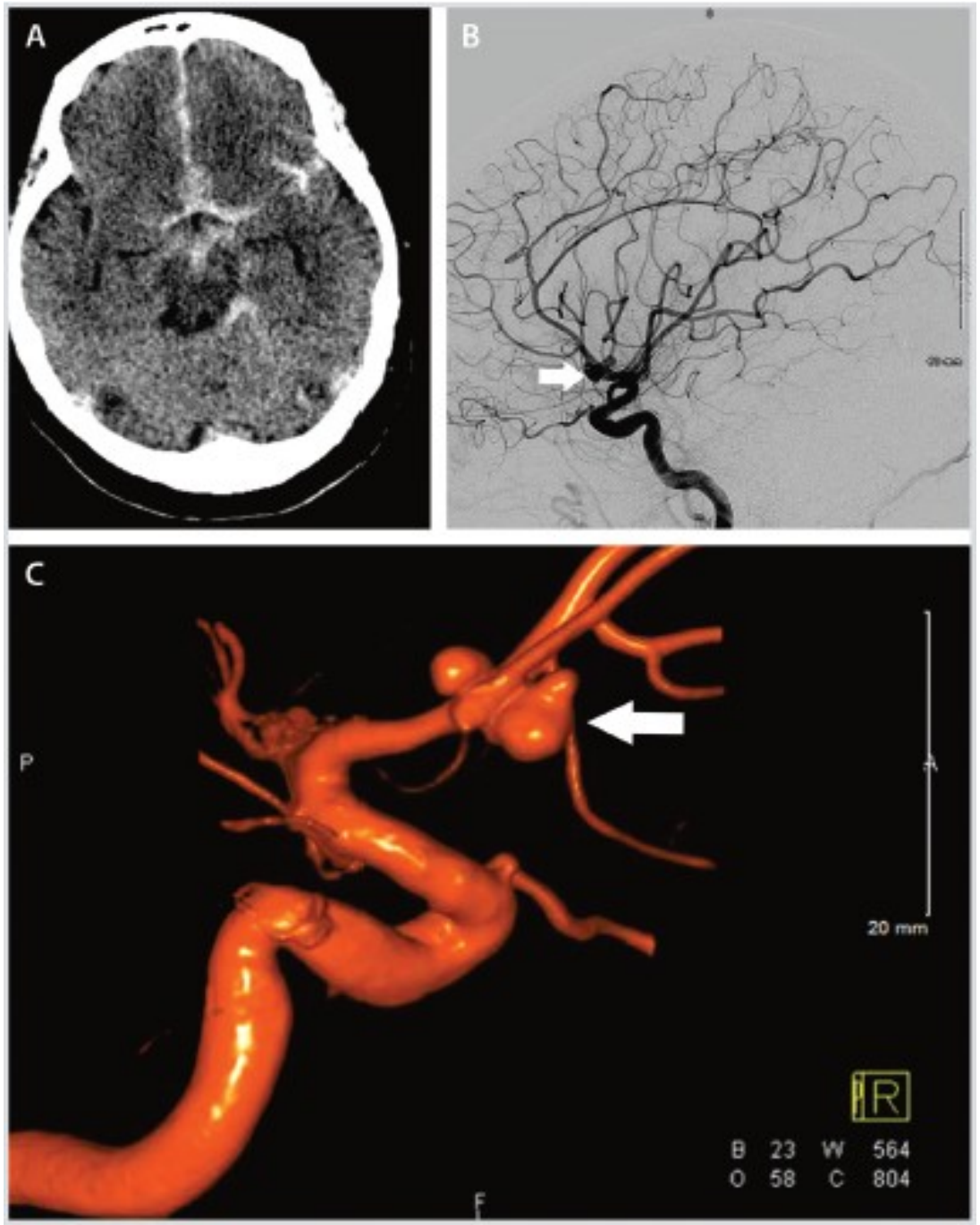





Cor>Sag


Cor>Sag -28




A
B
C
P
A
20 mm
R
B 23 W 564
O 58 C 804
F



Initial evaluation

- ABC
- CT scan
- BP control : SBP< 160 / MAP < 110 mm Hg
  Labetalol /Nicardipine/hydralazine
- Pain control
- Disease Severity Scoring
- Admission/transfer to high volume centre



TABLE 1-4 Clinical and Radiologic Grading Scales for Subarachnoid Hemorrhage[a]

World Federation of Neurological Surgeons Scale[34]			Modified Fisher Scale[35]		
Grade	Glasgow Coma Scale	Neurologic Examination	Grade	Subarachnoid Hemorrhage	Intraventricular Hemorrhage
1	15	No motor deficit	0	Absent	Absent
2	13–14	No motor deficit	1	Minimal	Absent in both lateral ventricles
3	13–14	Motor deficit	2	Minimal	Present in both lateral ventricles
4	7–12	With or without motor deficit	3	Thick[b]	Absent in both lateral ventricles
5	3–6	With or without motor deficit	4	Thick[b]	Present in both lateral ventricles



Definitive treatment

- Coiling vs Clipping – Patient age, aneurysm morphology and location, comorbidities
- ISAT trial : Disability free survival and less seizures @ 1 yr in coiling group. However, non complete obliteration and rebleed higher. Overall, coiling preferred to clipping when feasible.
- Multidisciplinary discussion – Neurosurgeon/Interventionalist/Neurointensivist
- When – as early as feasible . Rebleeding risk
- Tranexamic acid < 72 hrs



Preferences for treatment of unsecured aneurysms

Characteristics	Preferred Treatment Modality
Advanced age	Endovascular coiling
Poor clinical grade	Endovascular coiling
Multiple underlying systemic conditions	Endovascular coiling
Aneurysms with wide neck-to-body ratio	Surgical clipping
Normal arterial branches arising from dome or body of aneurysm	Surgical clipping
Middle cerebral artery aneurysm	Surgical clipping
Top-of-the-basilar aneurysm	Endovascular coiling
Aneurysm associated with large parenchymal hematoma	Surgical clipping
High surgical risk	Endovascular coiling
Patient preference	Endovascular coiling
Clinical equipoise[a]	Endovascular coiling



ICU Management : Neurologic complications

- Hydrocephalus : 20% of patients in first few days.

  EVD/Lumbar drain – 60% of these, EVD can be removed in initial days.

  VP shunt

- Seizures : Role of prophylaxis 3-7 days

  Levetiracetam

  Continuous EEG monitoring



Delayed cerebral ischemia

- Defined as any neurological deterioration (focal or global) presumed secondary to cerebral ischemia that persists for more than an hour and cannot be explained by any local/systemic complication.
- Diagnosis of exclusion – Hydrocephalus/Seizures/Sedation/Metabolic
- ~ 30% of patients
- Pathophysiology – Cerebral Vasospasm, Microcirculatory constriction, microthrombosis, cortical spreading depolarisation, cellular apoptosis
- Risk for vasospasm increases with thickness, density, location and persistence of subarachnoid blood
- Poor clinical grade, h/o LOC, smoking, cocaine, hyperglycemia, SIRS, hydrocephalus increase risk of delayed cerebral ischemia.



DCI - Management

- Prophylaxis : Nimodipine 60 mg q 4 hrly.
  Euvolemia
- Monitoring : Clinical checks q 2hrly
  : CT @ 24 hrs after securing aneurysm, 3-5 and 7-10 days
  : TCD – Mean Cerebral blood flow velocity
  < 120 cm/s – No vasospasm
  > 200 cm/s – Vasospasm
  Rise > 50 cm/s in 24-48 hrs – High risk
  LindeGaard Ratio > 3 –Vasospasm
  : CTA/CTP/DSA
  : EEG/Brain tissue O2 monitoring



- Treatment

  -Fluid bolus

  - Induced hypertension

  - Endovascular intervention: Angioplasty / intra-arterial vasodilator

  - ? Mg, ? Statins , ? Endothelin antagonists



Systemic complications and management

- Cardiopulmonary
- Fever
- Thromboembolism : Sequential compression devices , UFH 24 hrs after securing aneurysm.
- Glucose abnormalities : Target 80-180 mg/dL
- Hyponatremia : SIADH vs Salt wasting .

  Avoid fluid restriction. Hypertonic saline. Fludrocortisone
- Hemoglobin : 8-10 g/dL



Initial treatment
- Early aneurysm repair
- CT/CTA/CTP on admission and on days 3–5, days 7–10
- Oral nimodipine
- Systolic BP <160 mm Hg before **aneurysm repair**
- Euvolemia
- Hemoglobin >8 g/dL

Avoid
- Hypotension
- Increased ICP
- Low CPP
- Fever
- Hypovolemia
- **Hyperglycemia**
- Hypoglycemia
- Hyponatremia
- Hypomagnesemia
- Hypocarbia
- Hypoxia
- Anticonvulsants unless documented seizures
- Low-volume center without access to endovascular specialists and multidisciplinary neurocritical care

Low risk
WFNSS 1, 2, no or thin SAH, no IVH, older age

High risk
WFNSS 1–3, no or thin SAH with IVH or thick SAH without IVH

High risk
Inaccessible due to sedation or WFNSS 3–5, thick SAH with IVH

Neurologic assessment every 4 hours, fluid balance every 2 hours, TCD every second day

Neurologic assessment every 2 hours, fluid balance every 12 hours, TCD every second day

Neurologic assessment hourly, fluid balance every 6 hours, consider fludrocortisone, daily weight, TCD daily, brain tissue oxygen and blood flow monitor, arterial catheter

No change neurologically, normal TCD and CTP

Low risk
Consider transfer to lower-level monitoring 5–10 days after SAH

High risk
Consider transfer to lower-level monitoring 7–10 days after SAH

Elevated TCD velocities (>120 cm/s or Lindegaard ratio >3), increased vigilance for DCI

Elevated TCD velocities (>120 cm/s or Lindegaard ratio >3), abnormal CTP, neurologic deterioration

Elevated TCD velocities (>120 cm/s or Lindegaard ratio >3), abnormal CTP, abnormal brain tissue oxygen or cerebral blood flow, neurologic deterioration

No change **neurologically,** normal TCD, CTP, brain tissue oxygen, cerebral blood flow

Consider transfer to lower-level monitoring 14 days after SAH

Detect causes of neurologic deterioration

Correct identified causes (eg, seizures, increased ICP, hydrocephalus, hyponatremia, infections)

No major identified causes for deterioration: CTA/CTP or catheter angiogram
Consider empiric induced hypertension if aneurysm has been repaired

No major elevation of MTT or angiographic vasospasm; or deficit resolves

CTP elevated MTT or angiographic vasospasm, persisting deficit

Deficit persists >60 minutes or patient cannot tolerate induced hypertension

Continue monitoring

Induced hypertension, fluid boluses as necessary, vigilant monitoring

Consider catheter angiography and balloon and/or pharmacologic angioplasty










QUESTION
EVERYTHING