EM CASES SUMMARY

Episode 195 Management of Subarachnoid Hemorrhage

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Management of the undifferentiated crashing brain: Management of the patient with a suspected subarachnoid hemorrhage prior to CT imaging

Clinical features on their own have shown to not be reliable in distinguishing ischemic versus hemorrhagic CNS insult. While patients with head bleeds are more likely to complain of headache, nausea and vomiting compared to patients with ischemic strokes, a significant minority of patients with ischemic strokes do have these symptoms. Definitive management requires neuroimaging however we need to be able to empirically resuscitate the sick neurological patient keeping in mind important factors for the crashing brain.

4 critical priorities in the first 10 minutes:

1. Check and correct the **glucose** or empirically give an amp of D50W
2. Perform a **rapid neurological exam** prioritizing GCS, eyes (reaction to light, discongugate gaze, deviation), and motor response
3. **Avoid hypotension and hypoxia** at all costs considering early airway management/capture if needed
4. **Resuscitate to get to the scanner** so targeted treatment can be initiated after diagnosis is made

Initial imaging for suspected subarachnoid hemorrhage: Non-contrast CT vs CT plus CTA upfront?

In the crashing neurological patient we need more information than a plain CT head can offer to drive definitive management. The delay to definitive management can mean loss of brain viability. If available at your center, consider CT + CTA as the *initial* imaging modality of choice patients who:

- Have neurological deficits: speech or motor deficit, vision loss, decreasing or low GCS.
- Pre-existing intracranial vascular abnormality
- Have a contraindication to LP
- With shared decision-making >6 hours post headache onset (see **SAH Part 1 Episode 194** for details)

Emergency Nontraumatic Subarachnoid Hemorrhage Management

The management of patients with nontraumatic subarachnoid hemorrhage in the early stages of disease predicts outcomes, and attention to details matters.

Goals of management in patients with nontraumatic subarachnoid hemorrhage

- Prevent hematoma expansion
- Prevent re-bleeding
- Prevent ischemia
- Identify and treat raised ICP
- Prepare the patient for definitive management
- Optimize vital signs
- Prevent aspiration

Subarachnoid hemorrhage blood pressure management

- **Target** SBP <160 or MAP <110.
- **First line:** anti-emetics (**ondansetron** 8mg IV) and pain management (**fentanyl** 1microgram/Kg).
- **Second line**: **Labetalol** 10-20mg IV q5 mins and/or **hydralazine** 5-10mg q30 mins (beware hypotension!)
- Avoid nitrates as they may increase ICP
- Avoid hypotension aiming for MAP >80mmHg using early and peri-intubation vasopressors as needed

Management of raised ICP (a clinical determination, rather than a radiographic diagnosis)

1. **Raise the head of the bead** and blunt the pain and emesis response early.
2. **Hyperosmolar therapies**: Administer hypertonic saline 3mL/kg ~ 250mL (preferred) or mannitol at 1g/kg (if hypertonic saline is not available). 1-2 amps of sodium bicarbonate is another option if time is of the essence.
3. **Gentle limited hyperventilation:** Intubate, deeply sedate and paralyze the patient and hyperventilate to target PCO2 of 30-35mmHg (for no longer than 1-2hrs)
4. **Neurosurgical intervention:** Placement of EVD or decompressive hemicraniectomy.

Pearl: *If hypertonic saline or mannitol are not easily available you can also use 1-2 ampules (50cc) of sodium bicarbonate to manage raised ICP in a pinch.*

Pitfall: *Hyperventilation causes vasoconstriction thereby decreasing cerebral edema and ICP, but it should not be done for longer than 1-2 hours or otherwise there is risk of prolonged ischemic damage and blood-brain barrier breakdown.*

The role of nimodipine in prevention of delayed ischemia and vasospasm in subarachnoid hemorrhage

Delayed brain ischemia and intracranial vessel vasospasm is a common, potentially devastating subarachnoid hemorrhage complication that occurs in as many as 1/3 of patients and leads to death or severe disability in about 20% of patients. It usually occurs after 72h following the SAH event, and peaks at 7 days. It is likely secondary to BBB breakdown and inflammation resulting in vascular tone dysregulation. It is more common in high volume bleeds and in high grade subarachnoid hemorrhage.

Nimodipine 60mg PO/NG q4h initiated within 48h of event is recommended in the 2023 AHA guidelines and supported by a Cochrane review of 16 studies for the prevention of delayed ischemia and vasospasm in patients with subarachnoid . DO NOT use IV formulation (evidence of adverse events).

Prevention of hypovolemia related to cerebral diuresis in the management of subarachnoid hemorrhage

Irritation from blood on brain tissue can cause cerebral salt wasting which draws water out and causes significant diuresis. This may result in hypovolemia and hypotension. In turn, hypotension can increase the risk for vasospasm and cerebral ischemia.

Steps to prevent hypovolemia and resultant hypotension as a result of cerebral diuresis:

1. Insert a foley catheter and closely monitor urine output
2. Monitor volume status
3. Closely monitor sodium level
4. Match output with input to maintain *euvolemia* and *eunatremia*

Seizure prophylaxis in the management of subarachnoid hemorrhage

Not all patients with subarachnoid hemorrhage should receive seizure prophylaxis.
Seizure prophylaxis is indicated in the subarachnoid hemorrhage patients with:

- Ruptured MCA aneurysm
- Intraparenchymal hemorrhage associated with SAH
- High grade or high volume aneurysmal SAH
- Hydrocephalus
- Cortical infarction
- Recurrent seizure or non-immediate seizures
- Patient is intubated
- GCS <10-13
- Ongoing bleeding (especially if aneurysm >5mm)

Isolated seizure at time of event may not warrant seizure prophylaxis
Consultation with on call neurosurgeon may be warranted in the decision to start seizure prophylaxis.

Seizure prophylaxis medication of choice in subarachnoid hemorrhage: levetiracetam loading dose 20-60mg/kg infused over 5-15 minutes. If levetiracetam is not available phenytoin 20mg/kg is another option.

Prevention of re-bleeding and definitive management of nontraumatic subarachnoid hemorrhage

Neuroprotective critical care

- Target neuroprotective vital signs:
  - Normothermia: Treat temperature >37C with Tylenol and physical cooling if needed.
  - Normocarbia
  - Euvolemia: Monitor urine output
  - Euglycemia: 6-10mmol/L
  - Normoxia – target saturation >94%
  - Eunatremia – monitor electrolytes closely and consider hypertonic saline if hyponatremia is developing
  - Normotension – target SBP <160 or MAP <110. and avoid significant swings in blood pressure
- Monitor and manage increased ICP
- Careful neuromonitoring with frequent neurovital checks and re-imaging if any signs of worsening
- Monitoring for and prevention of delayed vasospasm and cerebral salt wasting by maintaining euvolemia and eunatremia.

TXA in *not* recommended for nontraumatic spontaneous subarachnoid haemorrhage. TXA may have a role in traumatic subarachnoid hemorrhage.

Neurosurgical intervention options

- For the aneurysm: endovascular coiling or embolization, neurosurgical clipping, flow diversion, or resection
- For obstructive hydrocephalus secondary to hematoma decompressing into ventricles: external ventricular drain (EVD)

Prognostication in nontraumatic subarachnoid hemorrhage

The Hunt and Hess Score is the mostly widely used and simplest prognostication tool for spontaneous subarachnoid hemorrhage which can help guide discussions with families and patients.

Hunt Hess Grading Scale for Subarachnoid Hemorrhage

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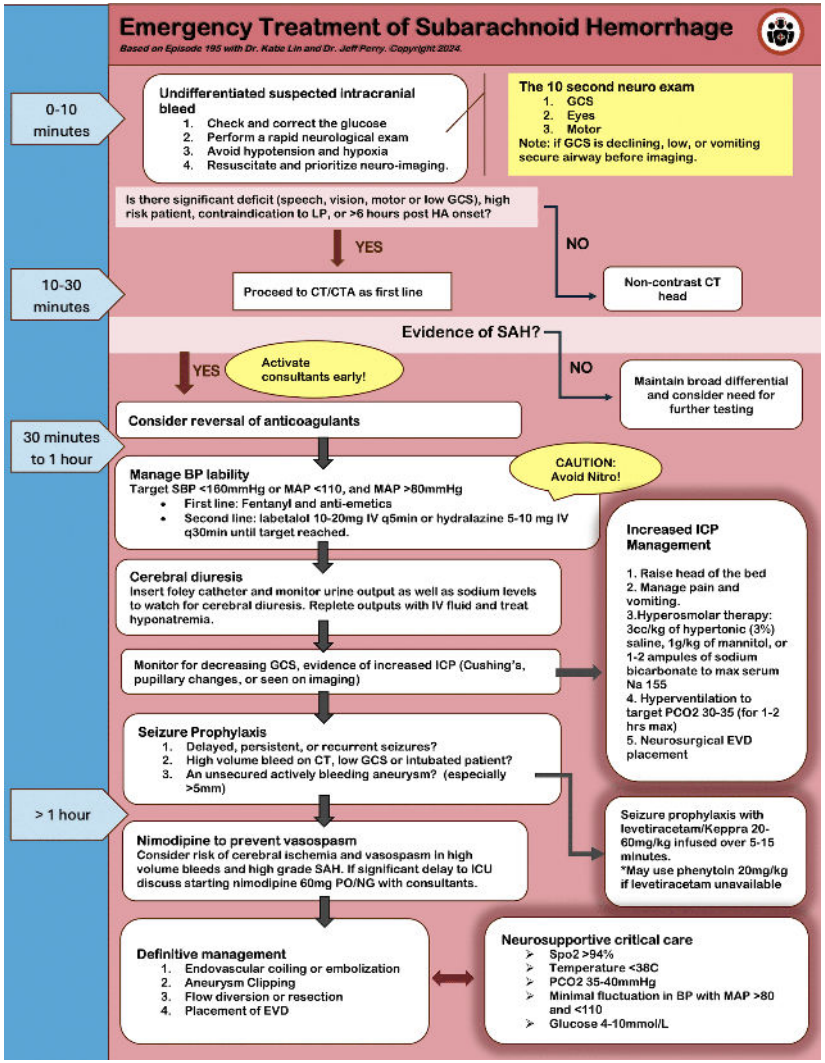
Grade	Clinical Presentation	Survival Rate (%)
I	Mild headache, normal mental status, no neurological deficits, Minimal/slightly nuchal rigidity.	70
II	Severe headache, normal mental status, may have cranial nerve deficit	60
III	Somnolent, confused, may have cranial nerve or mild motor nerve deficit	50
IV	Stupor, moderate to severe motor deficit, may have intermittent reflex posturing	20
V	Coma, decerebrate posturing or flaccid	10

Grade I: GCS 15 with mild symptoms and no deficits (mortality 30%)
Grade II: GCS 15 with moderate to severe symptoms (possible CN deficit) (mortality 40%)
Grade III: GCS <15, mild motor deficits, somnolence (morality 50%)
Grade IV: GCS <8 with severe deficits (mortality 70%)
Grade V: GCS 3-4 with posturing or flaccid (mortality 90%)

Pitfall: *The Hunt and Hess score does not consider if your patient has depressed GCS secondary to obstructive hydrocephalus. It does not take a significant volume of blood in the ventricles to cause obstruction, increased ICP and herniation with decreasing GCS. If this is the main cause of depressed consciousness remember that hydrocephalus can be potentially correctable and survivable with the placement of an EVD. In addition, inter-observer variability in scoring GCS is wide.*



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