Hemorrhagic Stroke Management in the Hospital Setting

Definition and Epidemiology

- Hemorrhagic stroke involves bleeding into or around the brain, classified as intracerebral hemorrhage (ICH) or subarachnoid hemorrhage (SAH), causing neurologic deficits and high mortality
- This guide details inpatient management, including causes, diagnostics, treatment, and complications
- Prevalence Hemorrhagic stroke accounts for ~10-15% of strokes, with ICH ~10% and SAH ~5% of all strokes
- Annual incidence ~12-15 per 100,000 for ICH, ~6-9 per 100,000 for SAH
- Risk Factors Hypertension (HTN), anticoagulation, amyloid angiopathy, aneurysm, arteriovenous malformation (AVM)
- Rare Demographics Pediatric ICH (trauma), pregnancy-related SAH, cocaine-induced hemorrhage

Pathophysiology

- **Mechanisms:**
  - ICH results from vessel rupture (e.g., HTN, amyloid), causing hematoma, mass effect, and edema
  - SAH involves bleeding into the subarachnoid space, often from aneurysm rupture, leading to vasospasm and hydrocephalus
  - Increased intracranial pressure (ICP) reduces cerebral perfusion, causing ischemia
- **Effects**
  - Hematoma expansion (30-40% within 6h), cerebral edema, and vasospasm (SAH, 20-30%) lead to neurologic deterioration
- **Mortality** ~40% for ICH, ~30% for SAH at 30 days
- **Molecular Pathways**
  - **HTN:** Chronic shear stress weakens arterioles
  - **Amyloid:** Aβ deposition disrupts vessel integrity
  - **Vasospasm:** Endothelin-1, reduced nitric oxide
  - **Key Pathway** Vessel rupture → Bleeding, mass effect → ICP elevation → Ischemia, neurologic deficits

Causes and Differential Diagnosis

Causes

- ICH Chronic HTN, amyloid angiopathy, anticoagulation, AVM, tumor, trauma
- SAH Saccular aneurysm, trauma, AVM, cocaine, vasculitis
- Rare Moya-moya disease, cerebral venous sinus thrombosis, reversible cerebral vasoconstriction syndrome (RCVS)

Differential Diagnosis

- Ischemic Stroke Sudden deficits, no hemorrhage on CT
- Seizure Post-ictal deficits, EEG epileptiform activity
- Tumor Gradual onset, enhancing lesion on MRI
- Migraine Aura, no imaging abnormalities

Conditions to Rule Out

- Infection (meningitis, abscess) CSF analysis, blood cultures
- Coagulopathy INR, aPTT, platelet count
- Hypertension BP >180/110, check history
- Drug Use (cocaine) Toxicology screen

Clinical Presentation

Symptoms

- Sudden headache, focal weakness, aphasia
- Nausea, vomiting, altered consciousness
- Seizures, visual changes (SAH)
- Rare Coma, neck stiffness (SAH), vertigo (cerebellar ICH)

Exam

- ICH: Focal deficits (hemiparesis, dysarthria), GCS <13, pupil asymmetry
- SAH: Meningismus, photophobia, Hunt-Hess grade 2-5
- Both: Hypertension (BP >180/110), lethargy
- Red Flags: GCS <8, BP >200/120, pupil dilation, seizure, midline shift >5 mm

Labs and Studies

Labs

- CBC: Platelets <100K (bleeding risk), leukocytosis (infection)
- Coagulation: INR >1.5, aPTT (anticoagulation), D-dimer (DIC)
- CMP: Cr (AKI), glucose (hyperglycemia worsens outcome)
- Troponin: MI trigger, cardiac strain
- Advanced: Toxicology (cocaine), anti-Xa (DOACs), TEG (coagulopathy)

Imaging

- CT Head Non-contrast (NCCT) Gold standard; ICH (hyperdense), SAH (cistern blood)
- CTA Head/Neck Aneurysm, AVM, vasospasm
- MRI Brain Gradient echo for microbleeds, amyloid
- CXR Pulmonary edema (HF, neurogenic)
- Advanced DSA (digital subtraction angiography) for aneurysm, MRA for AVM

Other

- EEG Seizures (10-20% of ICH/SAH)
- ICP Monitoring EVD for hydrocephalus (SAH, 20%)
- Advanced Lumbar puncture (SAH if CT negative), cerebral oximetry (ICU)

Diagnosis

Criteria

- ICH NCCT showing hyperdense parenchymal bleed, focal deficits
- SAH NCCT showing subarachnoid blood, meningismus, or xanthochromia on LP
- Differential Ischemic stroke, tumor, seizure, migraine

Flowsheet

Step 1 History/Exam Sudden deficits, headache, assess BP, GCS

Step 2 Labs Coagulation, troponin, glucose, toxicology

Step 3 Imaging NCCT (hemorrhage), CTA (aneurysm, AVM)

Step 4 Studies EEG (seizures), LP (SAH if CT negative)

Step 5 Risk Stratify ICH score, Hunt-Hess, plan intervention

Management Strategies

General Principles Reverse coagulopathy, control BP, manage ICP, prevent complications

ICU care for GCS <13, hydrocephalus, or vasospasm

Supportive Care

- Airway Intubation for GCS <8, respiratory failure
- Monitoring BP q15min, neuro checks q1h, ICP if EVD
- Seizure Prophylaxis Levetiracetam 500 mg IV q12h (ICH, SAH)

Specific Therapies

- BP Control:
  - Nicardipine 5-15 mg/h IV, labetalol 10-20 mg IV q10min
  - Target SBP 140-160 mmHg (ICH), <140 mmHg (SAH pre-aneurysm secure)
- Coagulopathy Reversal
  - Warfarin Vitamin K 10 mg IV, PCC 25-50 units/kg
  - DOACs Idarucizumab 5 g IV (dabigatran), andexanet alfa (apixaban)
  - Platelets >100K for surgery, FFP for INR >1.5
- ICP Management
  - Mannitol 0.5-1 g/kg IV q6h, hypertonic saline 3% 250 mL IV
  - EVD for hydrocephalus (SAH, 20%), elevate head 30°
- Surgical Intervention
  - ICH Craniotomy for hematoma >30 mL, cerebellar bleed >3 cm
  - SAH Aneurysm clipping/coiling, EVD for hydrocephalus
  - Vasospasm (SAH) Nimodipine 60 mg PO q4h, milrinone 0.5-1.5 mcg/kg/min IV
- Complication Management
  - Seizures Levetiracetam 1000 mg IV load, lorazepam 2 mg IV PRN
  - Rebleed Reverse anticoagulation, secure aneurysm (SAH)
  - Hydrocephalus EVD placement, serial CT
- Monitoring
  - NCCT q6-12h (hematoma expansion), TCD (vasospasm)
  - CMP, CBC q12h (AKI, anemia), INR q6h (coagulopathy)

Complications

Acute

- Hematoma Expansion 30-40% in ICH within 6h, worsens outcome
- Vasospasm 20-30% in SAH, peaks day 7, causes ischemia
- Seizures 10-20% in ICH/SAH, increase ICP
- Hydrocephalus 20% in SAH, 10% in ICH, causes coma

Long-Term

- Neurologic Deficits 50-70% of survivors, hemiparesis, aphasia
- Cognitive Impairment 30% post-SAH, memory deficits
- Rare Rebleed (SAH, 5% pre-aneurysm secure), epilepsy, depression

Clinical Scenarios

Case 1 Intracerebral Hemorrhage

- Presentation: 65 y/o M with HTN, sudden weakness, BP 200/110
- Vitals :HR 90, SpO2 96%, RR 18, GCS 12
- Exam: Left hemiparesis, pupil symmetry
- Labs/Studies: INR 1.2, platelets 200K, Cr 1 mg/dL, CT-H non con: Right basal ganglia ICH, 20 mL
- Interpretation Hypertensive ICH, stable hematoma
- Management: Nicardipine 5 mg/h IV (SBP 140-160), levetiracetam 500 mg IV q12h, Neuro checks q1h, repeat CT 6h. Stable, transfer to rehab by day 7

Case 2 Subarachnoid Hemorrhage

- Presentation: 50 y/o F with thunderclap headache, BP 180/100
- Vitals: HR 100, SpO2 94%, RR 20, GCS 14
- Exam: Meningismus, Hunt-Hess 2
- Labs/Studies: INR 1.0, Cr 0.8 mg/dL, CT-H non con: SAH in basal cisterns, CTA 5 mm ACA aneurysm
- Interpretation Aneurysmal SAH
- Management Nimodipine 60 mg PO q4h, labetalol 20 mg IV, aneurysm coiling, EVD for hydrocephalus, TCD q24h, No vasospasm, discharge day 10

Case 3 ICH with Anticoagulation

- Presentation; 75 y/o M on apixaban, fall, coma, BP 160/90
- Vitals; HR 80, SpO2 92%, RR 16, GCS 7
- Exam; Right pupil dilated, decerebrate posturing
- Labs/Studies; INR 1.4, platelets 150K, Cr 1.5 mg/dL, CT-H non con: Left frontal ICH, 40 mL, midline shift
- Interpretation; Anticoagulant-related ICH, surgical candidate
- Management; Andexanet alfa, nicardipine 5 mg/h IV, craniotomy, Levetiracetam 1000 mg IV, ICU care, GCS 10 by day 5, prolonged recovery

Expert Tips

Lower SBP to 140-160 mmHg in ICH; avoid <140 mmHg to maintain perfusion
Use nimodipine in SAH to prevent vasospasm; start within 96h
Reverse anticoagulation within 1h; PCC faster than FFP for warfarin
Prophylactic levetiracetam for 7 days in ICH/SAH; avoid phenytoin
Monitor TCD daily in SAH; milrinone for refractory vasospasm
Pitfall Delayed coagulopathy reversal; increases hematoma expansion
Advanced Minimally invasive ICH evacuation (MISTIE); cerebral oximetry for ICP

Key Pearls

ICH: BP 140-160 mmHg, reverse coagulopathy, surgery for large hematomas
SAH: Nimodipine, aneurysm clipping/coiling, EVD for hydrocephalus
NCCT gold standard; CTA for aneurysm, AVM
Seizures, vasospasm, hydrocephalus are major risks; monitor closely
Recent data emphasize early BP control, coagulopathy reversal

References

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