Acute Care Management

GEORGIA STROKE CURRICULUM



Learning Objectives

- Identify:
 - Types of stroke: Ischemic and Hemorrhagic
 - Evidence based stroke treatment
 - Stroke complications
 - Evidence based stroke quality programs

Ischemic Stroke





Defining the Stroke Timeline

• Acute Ischemic Stroke (AIS)

- Hyperacute stroke: first 24 hours
- Acute stroke: first 24 72 hours
- Subacute stroke: after 72 hours
- Chronic stroke: 6 weeks or greater



Priority the first 24 hours is frequent assessment of neurological status

Neurologic Assessment Scales

- **ABCD2** (0-7) TIA score for stroke risk in 2-90 days
- NIHSS (0-42) Neurological function & stroke severity
- GCS (3-15) neurologic scale, not sensitive for focal deficit
- Hunt/Hess (1-5) Severity outcomes for SAH (mortality in 30 days)
- Fisher (1-4) Risk of vasospasms for SAH
- ICH score (0-6) Predicts 30-day mortality
- Modified Rankin Scale (0-6) 90 day follow up for disability

NIH Stroke Scale



- Evidence based, validated, standard tool used to measure neurological function over time (0-42)
- Communication tool between bedside staff and providers
- NIHSS > 25 to 30 indicates long term neurologically impairment and increased morbidity and mortality
- An increase or decrease of 4 points in the stroke scale indicates important neurological changes
- Best practice as initial stroke assessment tool and long-term outcome for AIS
- Providers certified a minimum of every 2 years

Acute Care Diagnostic Testing

CT (non-contrast)

- MRI:MR diffusion-weighted imaging (DWI) most sensitive and specific imaging technique for demonstrating AIS
- Vascular imaging includes MRA and CTA/CTP (venous)
- Angiography evaluates the surface characteristics of the artery
- Carotid Doppler Ultrasound
 - Detects vessel occlusion or stenosis
- Transcranial Doppler (TCD)
 - Detects vasospasms
- Echo Cardiac diagnostics
- Additional lab work



Venous TCDS Transferriporal window

The Ischemic Cascade

- Brain tissue moves from reversible ischemia to irreversible infarction with blood-brain-barrier breakdown and vasogenic edema
- Cerebral Blood Flow drops below 10 ml/100 g/min



Malignancy Profile: A small infarct volume, large area of penumbra and a short time to reperfusion are predictors for good outcomes



Cerebral Edema

Pathophysiology

- Ischemic stroke: primarily cytotoxic edema
 - Cellular breakdown, cell membrane pump failure (Na⁺/K⁺)
 - Retention of *intracellular* Na⁺ and CA⁺⁺, rapid retention of water in gray matter
 - Blood brain barrier remains intact
 - Vasogenic extracellular edema primarily in white matter
- o ICH: perihematomal edema primarily vasogenic edema
 - Aggravated by clot lysis and inflammatory factors
 - Extracellular edema, BBB is disrupted, capillary leakage
- SAH: primarily cytotoxic edema
 - Localized versus global edema

Management

Symptom management for increased ICP or seizures



Intracranial Pressure (ICP) Compliance Principle



Hydrocephalus

Normal Pressure or Communicating

- CSF flow can "communicate" with Superior Sagittal Sinus
- Characterized by gait disorder, cognitive decline, urinary incontinence in the elderly → shunt treatment may improve symptoms



Obstructive or Non-communicating

- CSF flow "can't communicate" with arachnoid villi in Superior Sagittal Sinus
- Arachnoid villi are damaged or
 obstructed by presence of blood
 (SAH) or infection (meningitis)



Hemicraniectomy

- Cerebral Edema: most common in MCA territory
- Allows room for swelling for a large AIS
- Timing is critical and controversial

Complications

- o Contralateral hematoma (Epidural Hematoma)
- Herniation through bone window
- CSF leakage
- Infection
- o Seizure





Bone flap storage

Hemorrhagic Stroke





Hemorrhagic Stroke

- Subarachnoid Hemorrhage (SAH)
 - Aneurysm or AVM
- Intracerebral Hemorrhage (ICH)
 - Uncontrolled Chronic HTN
- Intraparenchymal Hemorrhage (IPH)
 - Uncontrolled Chronic HTN
- Intraventricular Hemorrhage (IVH)
 - Uncontrolled Chronic HTN
- Higher Morbidity and Mortality



Causes of Hemorrhage

- Stimulant abuse Cocaine, Amphetamines
- Cerebral Cavernous Malformation (CCM)
- Arteriovascular Malformations (AVM)
- Amyloid Angiopathy (lobar ICH)
- Hematologic disorders
- Anticoagulant use
- Neoplasms
- Vasculitis



ICH High-Risk Populations

- Hypertensive patients (especially poorly controlled)
- Patients with multiple comorbid risk factors
- Patients with renal or liver failure
- Patients with dementia
- Anticoagulant users
- o Age > 55 years
- Alcohol abusers
- Smokers
- Certain ethnic populations
 - Asians (especially Japanese)
 - African Americans
 - Hispanics



Hemorrhage Mortality



Only 1/71 patients with ICH volume ≥ 30 mL functions independently at 30 days

ICH Score

ICH Score (Hemphill et al.)

Feature	Finding	Points
GCS	3-4	2
	5-12	1
	13-15	0
Age	>=80	1
	<80	0
Location	Infratentorial	1
	Supratentorial	0
ICH volume	>=30cc	1
	<30cc	0
Intraventricular Blood	Yes	1
	No	0
ICH SCORE		0-6 points

ICH Score	30 Day Mortality
0	0%
1	13%
2	26%
3	72%
4	97%
5	100%
6	100%

Must be documented within 6 hours of admission

Management of Hemorrhage

Goal: Provide general supportive care to manage the primary brain injury and limit secondary brain injury

- Support ABCs
- Seizure management
- Reverse anticoagulation immediately
- Prepare for Intubation/Protect the Airway
- Monitoring (BP, fever, ICP, labs, arrhythmias)
- BP management (IV infusion of vasodilator Cardene)
- VTE prophylaxis (SCD/IPC) (Pharmacological 24-48 hrs)



Hemorrhagic Stroke

Acquired Coagulopathies - Anticoagulation

- Warfarin/anticoagulation treatment for DVT, PE, A-Fib
- Incidence of anticoagulant associated ICH increases with INR 2.5 - 4.5
- Associated with longer duration of ICH expansion
- Doubles ICH mortality





Seizures and Hemorrhage

- Seizure risk 8% after ICH
- More frequent in ICH than AIS
- \circ Onset usually ≤ 24 hours of ICH
- More associated with cerebral amyloid angiopathy ICH than deep ICH
- Seizures in ICH patients and outcomes
 - May contribute to coma
 - Results in neuronal injury and destabilization
 - Associated with deterioration of NIHSS and increase in midline shift



Subarachnoid Hemorrhage (SAH)

Aneurysm Risk factors:

- Genetic Syndromes: ADPKD or Ehlers-Danlos Syndrome, Type IV
- Family history
- Female > male (2.7:1 ratio)
- Japanese or Finnish Ethnicity
- Age <u>></u> 40 years
- Vascular comorbidities: HTN, HLD, DM
- Smoking

Treatment clipping vs. coiling

- SAH mortality is 26-50%
 - 10-15% are fatal before hospital arrival
- Risk of recurrence is 10-25%
 - Highest risk within first 6 months



Clinical Manifestations of SAH

o 80% report "worst headache of my life"

- 10-43% have a 'sentinel' headache prior to event
 - Sentinel headache increases the risk of early re-bleeding
 - Often occurs within 2-8 weeks before overt SAH
- Nausea/Vomiting
- Decreased LOC
- Nuchal Rigidity
- O Photophobia
- Seizure



Diagnosis of SAH

○ NCCT

- Nearly 100% sensitivity within 6 hours
- 87% sensitivity within 72 hours
- 50% sensitivity after 5 days
- Lumbar Puncture can enhance sensitivity
 - Xanthochromia occurs > 12 hours after SAH



- MRI further adds to sensitivity
- Gold Standard is an angiogram



Hunt and Hess Scale for SAH

Grading system to classify the severity of SAH 1-5

Based on clinical picture (symptoms)

Predictor of patient prognosis/outcome

- 1. Asymptomatic/mild headache, slight nuchal rigidity
- 2. Mod/severe headache, nuchal rigidity, no deficit except oculomotor nerve palsy
- 3. Drowsiness/confusion & mild deficit
- 4. Stupor, moderate-severe hemiparesis
- 5. Coma posturing/no motor response

Grade	Clinical features	Mortality Outcome*
I.	Asymptomatic or mild headache	11
١١.	Moderate to severe headache, or with oculomotor palsy	26
III.	Confused, drowsy, or mild focal signs	37
IV.	Stupor (localizes pain)	71
V.	Coma (posturing or no motor response)	100

Cerebral Vasospasm & Rebleeding

Vasospasm causing Delayed Cerebral Injury (DCI)

- Risk for vasospasm day 3-21, most common day 7-15
- Oxyhemoglobin (blood breakdown) and Calcium channels trigger vasoconstriction
 Management:
 - Nimodipine Calcium Channel Blocker
 - Angiography with Angioplasty or direct Verapamil
 - Induced Hypertension and Hypervolemia (HH)

Rebleeding

- \circ Increased mortality, especially within first 2-12 hours after initial bleed
 - \circ Risk igtharpoint with initial sentinel headache, large aneurysm, elevated SBP

Management/Prevention:

- Early treatment
- BP management Pre treatment: 140-160 mmHG
- Post treatment: BP parameters liberalized to permissive hypertension



Fisher Scale for SAH

Predictor of Vasospasms

Based on blood volume on initial CT

Grades

- 1. No hemorrhage evident
- 2. SAH < 1ml thick
- 3. SAH > 1ml thick
- 4. SAH any thickness with IVH or parenchymal extension



Subarachnoid Hemorrhage

Endovascular Coiling

- Endovascular (Angio) approach
 - \circ Primary Coil
 - Balloon Assisted Coil
 - Stent Assisted Coil
 - \circ Flow Diversion
 - o Stent
 - o Intrasacular
- Antiplatelet therapy post procedure
- Post angio care



Microsurgical Clipping

- Involves a craniotomy
- Clips are placed across the neck of the aneurysm blocking it from circulation
- Favorable option for patients with an IPH volume > 50ml
- May involve intra-operative angiogram & post-angio care
- General anesthesia
- Titanium clips are MRI compatible







Additional Stroke Disorders

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Dissections and CVT

Cervical Cerebral Arterial Dissection

- Boxers, sports, wrestling, sneezing
- Carotid artery dissection: Horner's syndrome, ipsilateral deficits.
- Vertebral artery dissection: pressure headache, occipital throbbing, ipsilateral deficits
- Most Common Cause: Trauma to vessel wall or connective tissue disease
- Treatment: anticoagulants or antiplatelet

Cerebral Venous Thrombosis (CVT)

- Rare, young age
- Clot in dural sinus (vein)
- Inflammatory bowel disease, birth control, substance abuse, head trauma or hypercoagulable state
- Treatment: anticoagulation therapy (heparin infusion to oral anticoagulation for several months)





Patent Foramen Ovale (PFO)

Anatomical variant present in 25% of healthy adults

Emboli to left heart from abnormal blood flow

- Contributes to stroke < 55 years of age
- Diagnostic: TEE
- Transesophageal Echocardiogram





Management

- Medical Therapy: antiplatelet or anticoagulation
- Surgical Therapy: Percutaneous Closure Device
- Surgical closure may be superior to medical management, depend on PFO size



Carotid Endarterectomy (CEA) Carotid Artery Stenting (CAS)

- Risk factors: HTN, smoking, alcohol consumption, high LDL/cholesterol
- Mild < 50% occlusion
- Moderate 50-69% occlusion
- Higher Grade \ge 70%-99%

Medical Management

Medical Management

Surgery or Medical Management

- Intervention for symptomatic patients with carotid-territory TIA or stroke
- \circ 100% occlusion can now be treated with angioplasty
- Post procedure: ICU overnight
- Discharge on Plavix for 30 days, statin
- No lifting 5lbs or greater, no driving for 7 days



Post-Endovascular Care

$\,\circ\,$ Assess for complications after Vascular Access

- Recognition of Reperfusion Syndrome
- Hematoma and suture line evulsion
- Arterial Dissection/Thrombosis
- Retroperitoneal Hemorrhage
- o Pseudo Aneurysm
- MI and Stroke
- Cranial nerve and neuro assessment
- Site and distal extremity assessment
- Fluid management (euvolemic)
- Blood pressure management
- Arterial sheath management



Post Thrombectomy Care

- Vital signs include extremity pulses, puncture site checks and neurological checks per protocol
- If patient receives IV thrombolytic follow the IV thrombolytic protocol
- Maintain BP < 180/105 during and for 24 hours after procedure or per protocol
- Notify provider for any decline in neuro status





FIG 1. (a) Angiogram showing acute occlusion of the right middle cerebral artery (arrow). (b) Post-thrombectomy angiogram showing revascularisation of the right middle cerebral artery territory. (c) Thrombus removed by endovascular thrombectomy

Hyperperfusion/Reperfusion Syndrome

Hyperperfusion – increased blood flow to ischemic tissue

Reperfusion – normalization of flow in setting of fragile/damaged tissue

Reperfusion Syndrome – injury after revascularization

- Carotid endarterectomy and/or stenting, IV fibrinolytic
- Endovascular mechanical thrombectomy
- Large territory ischemic stroke with revascularization

Pathophysiology

- Inflammatory response, cytokine release, leukocyte adhesion, damage to the blood brain barrier
- Ipsilateral headache, contralateral neurological deficits, seizure (cortical area)

Management and Prevention

- Diligent blood pressure control
- Order NCCT head



CSF Diversion Devices

External Ventricular Device (EVD)



- Removes CSF or Blood
- Reduces increased ICP
- Acute Ischemic Stroke (AIS)
 - Edema may prevent the flow of CSF causing hydrocephalus
- Intracerebral Hemorrhage (ICH)
 - For GCS < 8, IVH, hydrocephalus or herniation
- Subarachnoid Hemorrhage (SAH)
 - Acute hydrocephalous
 - Blood in the ventricles

Ventricular Peritoneal (VP) Shunt





Nursing Care Plan

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Blood Pressure Management

- Acute Ischemic Stroke (AIS) without IV thrombolytic ≤ 220/120 mmHg
 - Permissive Hypertension first 24 hours
 - Avoid rapid drop in blood pressure
- Acute Ischemic Stroke (AIS) with IV thrombolytic \leq 180/105 mmHg
- Intracerebral Hemorrhage (ICH) ≤ range 130-150 with a goal of 140 mmHg
- Subarachnoid Hemorrhage (SAH) (ruptured) ≤ 160 mmHg SBP
- Aneurysm (unruptured) ≤ 140 mmHg SBP



Blood Glucose Management

• Treat if less than 60 mg/dl

- IV Fluid: Normal Saline, no dextrose
- Hyperglycemia present in 30-40% of AIS patients
 - Correlated with hemorrhagic conversion and poor outcomes
 - Increases anaerobic metabolism and lactic acidosis causing decreased tissue oxygenation
 - Greater than 200 mg/dl is an independent indicator of volume expansion in AIS and poor outcome
- Goal: 60-180 mg/dl
 - Treatment in the first 24 hours is recommended



Temperature Management

Normothermia 37°C or 98.6°F **Hyperthermia** > 38°C or >100.4°F

Antipyretics

- Tylenol: monitor liver function
- Advil: monitor renal function and platelets

Infection Surveillance

o UTI, aspiration pneumonia

External and intravascular cooling

- Skin surveillance
- Shivering medical prevention
- Skin warming (bear hugger)
- Sedation/paralysis



Stroke Management-Head of Bed

- Best Practice HOB elevated no more than 30°
- HOB lowering
 - Not indicated for majority of strokes
 - Increases cerebral blood flow
 - Increases risk of aspiration
 - Consider if blood pressure & HOB position correlate with improved neurologic exam in first 24 hours
 - Consider for posterior circulation strokes in first 24 hours
 - May recommend <u>HOB 15 degrees or flat</u>





Dysphagia Screening

- Dysphagia (difficulty swallowing)
- Standard of care: NPO until bedside screening
 - SLP consult for failed screening
- Dysphagia present in 50% of patients within three days
 - Higher risk for aspiration and silent aspiration





Increased mortality risk due to aspiration pneumonia

Management of Atrial Fibrillation

Telemetry during admission

Oral anticoagulation

Goal INR 2-3 which reduces mortality of AIS

- Timing: controversial generally wait 4-14 days after AIS or hemorrhagic stroke (size matters)
- CHA₂DS₂-VASc Score is a risk assessment tool to determine benefit of treatment
- HAS-BLED determines risk of bleeding

Definition of the HAS-BLED score, with point distribution ¹				
Score	Variable	Points		
Н	Hypertension	1 point		
А	Abnormal renal or hepatic function	1–2 points		
S	Prior stroke	1 point		
В	Bleeding	1 point		
L	Labile INR values	1 point		
E	Elderly, i.e., over age 65	1 point		
D	Concomitant use of other drugs or alcohol	1–2 points		

Score	CHA2DS2-VASc
1	CHF + CAD doubles the risk of stroke)
1	HTN
2	Age > 75 years
1	DM
2	Prior Stroke, TIA, or Thromboembolism
1	Vascular Dx: PVD, MI, Aortic plaque
1	Age 65-74
1	Sex category: female

Euvolemia

Most acute ischemic stroke patients are dehydrated on admission

- AIS have a 25% high risk of worsening outcomes
- Monitor I & O's closely (especially post contrast)
- IV solutions: isotonic or hypertonic solutions
- Isotonic solution
 - Normal saline (monitor labs)
 - Long-term use risk for acidosis/hyperchloremia

Hypertonic Solutions

- 3% -24% saline
- Manages increased ICP/cerebral edema

Hypotonic solutions

Avoid ½ NS, ¼ NS, Dextrose containing solutions



Nursing Safety Measures

Personal Care Measures

- Elimination Q 2 hours while awake and Q 4hr @ HS
- Monitor heat/cold for sensory deficits
- Safety during and after treatments
- Positioning and early mobilization
- Divide activities into short steps
- Awareness of surroundings
- Support affected limb
- Frequent orientation
- Protect from injury

Realistic attainable goals

Removal of excessive environmental distractions



Safety Measures

Prioritize patient needs

- Comprehensive Neurological Assessment
- NIHSS, GCS, ICH, Hunt & Hess
- Diagnostic tests
- Discharge planning

Aspiration Precautions

- Stroke Associated Pneumonia (SAP)
- Increased risk: 50% in ICU and 11% in Rehab

VTE Prophylaxis/Immobility

- Mechanical: SCD/IPC
- Pharmacological: LMWH or heparin
- Early mobility and hydration





Safety Measures

Fall & Skin Precautions

Stress Ulcer Prophylaxis

Infection Prevention Protocols/UTI

60% have urinary incontinence or retention

Seizure Precautions

- ICH has increased risk in first 24 hours
- SAH not routinely treated prophylactically

Therapeutic Environment

- Coordinate care
- Quite environment promote sleep hygiene
- Reorientation, alternative means of communication





Stroke Pearls

- Respect culture and beliefs
- Encourage expression of feelings
- Identify positive coping mechanisms
- Include spiritual and psychosocial care
- Assess and manage depression, anxiety, and fatigue



Thank you!



Resources

Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the early management of patients with acute ischemic stroke: 2019 update to the 2018 guidelines for the early management of acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2019;50:e344-e418.

Connolly, E. S., Rabinstein, A. A., Carhuapoma, J. R., Derdeyn, C. P., Dion, J., Higashida, R. T., Hoh, B. L., Kirkness, C. J., Naidech, A. M., Ogilvy, C. S., Patel, A. B., Thompson, B. G., & Vespa, P. (2012). Guidelines for the management of Aneurysmal Subarachnoid hemorrhage. *Stroke*, *43*(6), 1711–1737. https://doi.org/10.1161/str.0b013e3182587839

Keigher, K. M., Livesay, S., & Wessol, J. (2020). *Comprehensive Review for Stroke Nursing*. American Association of Neuroscience Nurses.

Greenberg, S. M., Ziai, W. C., Cordonnier, C., Dowlatshahi, D., Francis, B., Goldstein, J. N., Hemphill, J. C., Johnson, R., Keigher, K. M., Mack, W. J., Mocco, J., Newton, E. J., Ruff, I. M., Sansing, L. H., Schulman, S., Selim, M. H., Sheth, K. N., Sprigg, N., & Sunnerhagen, K. S. (2022). 2022 guideline for the management of patients with spontaneous intracerebral hemorrhage: A guideline from the American Heart Association/American Stroke Association. *Stroke*, *53*(7). https://doi.org/10.1161/str.0000000000000000407