Foundations Frameworks

Approach to Stroke Management

Author: Quentin Reuter, MD
Editors: Jonathan Edlow, MD; Andrew Ketterer, MD, MA; Kristen Grabow Moore, MD, MEd

Here is a step-wise approach to managing patients with a suspected stroke (those with altered mental status and/or focal neurologic deficits):

1. **Initial Stabilization**
    a. ABCs
        i. Evaluate the patient's ability to protect their airway and consider intubation. Recognize that ischemic strokes usually do not require intubation unless they have a large ischemic territory or a stroke affecting centers responsible for maintaining mental status (i.e. the brainstem)
        ii. Hypertension can be an early physiologic response and a sign of stroke; if the patient is hypotensive, you must investigate the cause
        iii. Beware of Cushing response (indicative of imminent herniation): irregular respirations, hypertension, bradycardia
            1. Emergently lower intracranial pressure (ICP) with hypertonic saline/mannitol and surgical decompression
    b. Evaluate pupils; consider ordering early mannitol (1 g/kg over 15 min) or hypertonic saline (3% 250 mL bolus) for suspected increased ICP
    c. Check a glucose and rule-out hypoglycemia

2. **CT Brain**
    a. Obtain an emergent CT brain to determine if the neurologic deficit is from hemorrhagic vs. (suspected) ischemic stroke. If there is concern for large vessel occlusion (LVO) stroke, obtain a CT angiogram.
    b. Hemorrhagic stroke
        i. Hypertensive
            1. Patients usually have chronic HTN, vascular disease
            2. Blood seen in parenchyma, usual locations: Basal ganglia, thalamus, pons, cerebellum
        ii. SAH
            1. Spontaneous: aneurysmal, arteriovenous malformations (blood typically in the basal cisterns)
                a. 12% initial mortality for aneurysmal SAH. Patients can be severely obtunded, have associated cardiogenic shock/arrhythmias, neurogenic pulmonary edema. Provide supportive care as indicated.
        iii. "Convexal": blood is high on convexities, outside of brain (seen in amyloid angiopathy, reversible cerebral vasoconstriction, or hypertensive encephalopathy)
        iv. Hemorrhagic transformation: blood seen in areas outside of the deep structures (e.g., a hemorrhagic stroke seen in an MCA territory)
    c. Ischemic Stroke
        i. Large vs. small vessel ischemic stroke: suspicion based on neurologic exam
        ii. Large vessel = Cortical (large territory)
            1. Anterior Cerebral Artery (ACA): strength/sensation of leg
            2. Middle Cerebral Artery (MCA): strength/sensation of arm and face, aphasias if left-sided, neglect if right-sided
            3. Posterior Cerebral Artery (PCA): contralateral visual field deficit
            4. Basilar artery: motor and sensory deficit + cranial nerve findings, possible AMS

5. Superior Cerebellar Artery (SCA), Posterior Inferior Cerebellar Artery (PICA), Vertebral Arteries: ataxia, dysarthria, vertigo, cerebellar findings
6. Diagnostic pearls: (think large vessel IF)
    a. Decreased consciousness
    b. Motor AND sensory involvement
    c. Higher level "thinking" processes affected
    d. Aphasia, right sided neglect ("take your left hand and point at the ceiling"), eye deviation (frontal eye fields affected)
7. Complications: cerebral edema leading to herniation and death

iii. Small vessel = Lacunar syndrome (damage to the white matter pathways [a.k.a. the wiring], not the higher level "thinking" processes)
1. No change in consciousness
2. No aphasia or neglect
3. Motor OR sensory involvement, not both

3. Treatment Approach

a. All strokes:
i. ABC stabilization
ii. Check glucose
iii. Head of bed up (if concerned for aspiration or ICP), NPO, bedrails up to prevent fall
iv. CBC, chemistry, coags, troponin, EKG

b. Hemorrhagic:
i. BP control:
1. Uncontrolled hypertension can worsen amount of hemorrhage, but hypotension will worsen ischemia to surrounding areas experiencing mass effect
2. Aim for a systolic blood pressure of 140-180 using a nicardipine drip (CCB also reduces risk of cerebral vasospasm causing secondary ischemic stroke)

ii. Anticoagulation reversal – check platelets, coags, take a good medication reconciliation
1. Anti-platelet medications- consider reversal with platelet transfusion, ddAVP (new data showing *potential harm* in platelet transfusion for spontaneous ICH)
2. Warfarin- reverse with vitamin K, Fresh Frozen Plasma (FFP), or Prothrombin Complex Concentrate (PCC)
3. Dabigatran- reversal agent (idarucizumab, i.e. Praxbind) or dialysis
4. Anti-Xa NOACs- specific reversal agents are under investigation, consider PCC

iii. Treat all seizures with benzodiazepines. Consider seizure prophylaxis with levetiracetam (indicated for those who have had a seizure)

iv. Increased ICP
1. Signs of increased ICP:
    a. Worsening mental status, CN 6 palsy, fixed & dilated pupil, decorticate/decerebrate posturing
    b. EKG with QT prolongation and diffuse, deep T wave inversions (cerebral T waves)
    c. US – dilated optic nerve (> 5 mm)
    d. CT showing midline shift, effacement of sulci
2. Treatment:
    a. Surgical decompression
    b. Elevate head of bed
    c. Optimize venous drainage - remove cervical collar, don't place internal jugular central lines
    d. Hyperventilate to a pCO_2 of 35-40 – only a temporizing measure to be performed if patient begins to actively herniate
    e. Osmotic agents
        i. Hypertonic (3%) saline 250 mL bolus
        ii. Mannitol (1 gram/kg over 15 minutes)

iii. 23% Hypertonic saline "bullet"

c. Ischemic Strokes
    i. Treatment options:
        1. tPA: patient presenting within 4.5 hours of symptom onset, no contraindications (SBP > 180, elevated INR on anticoagulation, previous ICH, recent surgery, > 3hr in pt > 80 yo, recent spinal injection/LP)
        2. Clot retrieval: emerging data shows benefit of clot retrieval in patients with LVO and viable brain tissue on CT perfusion/MR up to 24 hours post onset of symptoms
d. Disposition: neuro ICU, +/- neurosurgical or neuro-interventionalist consult

4. Consider Stroke Mimics

a. Check a blood sugar
b. Evaluate for the following stroke mimics:
    i. Hypoglycemia
    ii. Seizure (e.g. postictal Todd's paralysis)
    iii. Metabolic encephalopathy
    iv. Illicit drug use or EtOH intoxication
    v. Complex migraines
    vi. Peripheral nerve compression
    vii. Peripheral causes of vertigo
    viii. PRES or hypertensive encephalopathy
    ix. Conversion disorder
    x. Recrudescence (re-emergence of symptoms of an old stroke from a toxicologic, metabolic, infectious, or cardiac cause)

References:

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