



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 pCO₂ 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:

- Filho, JO, Mullen, MT. Initial assessment and management of acute stroke. last update Sept 2017. uptodate.com
- Adams, J et al. Emergency Medicine: Clinical Essentials, Second Edition. Transient Ischemic Attack and Acute Ischemic Stroke. pp. 870-880. 2013