Neuroradiology: Imaging and Stroke

Stroke 2017

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Stroke

- Arterial ischemia/infarct accounts for ~85%
- Cerebral venous occlusions - 0.5-1%
- Spontaneous intracranial hemorrhage (ICH)~15%
  - Hypertension (50%)
  - Amyloid angiopathy (20%)
  - Others (tumor, vascular malformation, coagulopathy) (30%)
Arterial Ischemic Stroke
Etiology

- Atherosclerotic vascular disease (ASVD) (40-45%)
  - Most infarcts are embolic, arising from an “at risk” plaque.
  - Carotid bifurcation > cavernous ICA segment.
  - Most frequently occluded intracranial vessel is the MCA.

- “Small vessel disease” (15-30%)
  - Small artery occlusions (lacunar infarcts)
  - Occlusion of perforating branches, most commonly in the basal ganglia, thalami, deep white matter, and brainstem.

- Cardioembolic disease (15-20%)
  - Common risk factors include MI and arrhythmia

- Craniocervical artery dissection (~2%)
  - Important cause of ischemic stroke in young and middle-aged adults
Acute Ischemic Stroke Terminology

- **Cerebral ischemia** - Affected tissue remains viable although blood flow is inadequate to sustain normal function.
- **Cerebral infarction** - Frank cell death occurs with loss of neurons, glia, or both.
- **Infarct core** - Center of affected parenchyma. Typically has a cerebral blood flow (CBF) <6-8 cm³/100g/min.
- **Ischemic penumbra** - Less ischemic brain surrounding the infarct core with CBF 10-20 cm³/100g/min. Present in ~1/2 of patients. “At risk” but potentially salvageable tissue.
Imaging in Acute Stroke

- Clinical presentation is misleading in 15-20% of presumed strokes.
- Imaging has become essential in rapid stroke assessment.
The 4 “Must Know” Acute Stroke Questions

- Is there intracranial hemorrhage (or a stroke “mimic”)?
- Is a large vessel occluded?
- Is part of the brain irreversibly injured?
- Is an ischemic “penumbra” present?
Time is Brain

- ~ 2 million neurons lost each minute when a major vessel such as the MCA is occluded.
- It is essential to initiate a CT scan within 25 minutes of arrival - “Code Stroke”
- Complete interpretation of the CT scan within 45 minutes
The 4 “Must Know” Acute Stroke Questions

- Is there intracranial hemorrhage (or a stroke “mimic”)? → CT
- Is a large vessel occluded? → CTA
- Is part of the brain irreversibly injured? → CT/MRI(DWI)
- Is an ischemic “penumbra” present? → CT or MR Perfusion
Brookwood-Baptist Acute Stroke workflow

Activate Stroke Code
Last seen normal < 6 hours

Stroke protocols initiated per
Originating Site process

CT Head without Labs
Contraindications

Yes

Patient meets criteria for IV tPA

No

Obtain advance imaging
CTA head and neck

If all meet IV tPA

Next obtain CTA head and neck

If large vessel occlusion: call Neurointerventional team to discuss

Transfer Line: 205-877-2727
Neurointerventional (Dr Sharma)
Pager: 205-940-5275
Cell: 716-961-0281

1. Wake up Stroke or last seen normal > 6 hours: Obtain CT head: if CT head shows no signs of stroke: Obtain CTA head and neck: if it shows large vessels occlusion – Call Neurointerventional team to discuss
CT vs MR

CT
- Faster
- Cheaper
- More readily available
- More appropriate in most hospitals in the acute setting.
- Good at detecting acute hemorrhage
- Limited sensitivity in detection of hyperacute/acute infarcts

MR
- Used at some centers as the workhorse in acute stroke workup.
- Far superior to CT in detecting small vessel and brainstem ischemia.
- Slower, but can be expedited with selection of only a few key sequences.
- Contraindications
- More expensive
Imaging in Acute Ischemic Stroke
Relevant Pathophysiology

- As oxygen is depleted, cellular energy production fails, and ion homeostasis is lost.
- ATP dependent Na⁺/K⁺ membrane pumps fail → Na⁺ accumulates in the cell, producing swelling and reduction in extracellular volume.
- “Cytotoxic edema”.
CT in acute infarct

- Initial non-enhanced CT (NECT) are abnormal in 50-60% of acute ischemic strokes.
  - Hyperdense MCA
  - Insular ribbon sign
  - Disappearing basal ganglia sign
  - Wedge shaped hypoattenuation
“Hyperdense MCA”

- Seen in 30% of cases with documented proximal MCA occlusion.
- Most specific but least sensitive sign of early ischemic stroke.

- Less common sites for a hyperdense vessel are the intracranial ICA, basilar artery, and deeper MCA branches in the sylvian fissure (“dot sign”).

- Remains hyperdense until the clot is lysed
Early Ischemic Changes by CT

- Blurring and indistinctness of gray-white matter interfaces can be seen in 50-70% of cases within the first 3 hrs.
- “Insular ribbon” sign- Blurring of the insular cortex and adjacent white matter.
- “Disappearing basal ganglia” sign- Edema of the basal ganglia and loss of differentiation with the internal capsule.
- Wedge shaped parenchymal hypodensity and cortical sulcal effacement.
Contraindications to IV-TPA on initial NECT

- Hemorrhage
- Cytotoxic edema involving greater than 1/3 of the MCA territory (~70mL).
- Stroke mimicker (neoplasm).
Alberta Stroke Program Early CT Score (ASPECTS)
Stroke vs. Tumor

Cytotoxic Edema
- Cellular swelling
- Gray-white margin lost

Vasogenic Edema
- Leaky capillaries
- Gray matter is spared
Differentiating acute from chronic infarct
Vascular imaging in acute stroke

- Goal of imaging
  - Is a large vessel occluded?
  - Is there a critical stenosis?
  - Degree of collateral supply?
  - Role for endovascular therapy?
CT Angiography (CTA)

- **Pros**
  - Faster and more readily available
  - Very accurate in detecting thrombus and degree of stenosis
  - Less prone to motion artifact in an uncooperative patient
  - Less contraindications
  - Reveal access limitations for endovascular treatment
  - Assess collaterals

- **Cons**
  - IV contrast
  - Radiation

*NECT should always accompany CTA head + neck*
MR Angiography

**Pros**
- “Time of Flight” allows for dynamic vessel assessment without contrast
- Better at diagnosing dissection
- No radiation

**Cons**
- Time
- Contraindications
- Motion artifact

*can do MRA neck with and without contrast
*MRA head w/o only.
MRI in Acute Stroke

- **Diffusion Weighted Imaging (DWI)**
  - Gold standard in assessing infarct “core”.
  - Positive within minutes of ischemic insult.
  - High signal on DWI. Low signal on ADC.
  - 95-97% sensitive for detection of acute/hyperacute infarcts
MRI in Acute Stroke

- **T1WI**
  - Usually normal in first 3-6hr
  - Subtle gyral swelling/edema (low signal) within 12-24hr.

- **T2/FLAIR**
  - Increased signal and swelling.
  - 30-50% of acute strokes visible on FLAIR within first 4 hrs.
  - Nearly all are positive on FLAIR by 7 hrs.
  - Loss of “flow void” due to slow flow or occlusion.

- **T2**
  - Intra-arterial thrombus “blooming”.
  - Can identify petechial hemorrhage

- **T1C+**
  - Intravascular enhancement due to slow flow.
Perfusion Imaging

- Can be performed with CT or MR
- Rapid dynamic imaging of the brain following contrast administration allows for quantification of cerebral blood volume (CBV), flow (CBF), and mean transit time (MTT)
- **Infarct “core”** - CBV and CBF are decreased. MTT is prolonged.
- **Ischemic “penumbra”** – CBF decreased, CBV normal or increased. MTT prolonged.
- Potentially salvageable brain tissue is equivalent to CBF - CBV
Perfusion Imaging Pros and Cons

- **Pros**
  - Identifies core infarct
  - Identifies penumbra

- **Cons**
  - Time (~25-30min)
  - Limited brain coverage
  - Very susceptible to motion
  - Contrast required
  - High radiation dose
Imaging in subacute stroke

- Generally refers to 2 days-2 weeks
- Edema and mass effect peak at 3-4 days
- Hemorrhagic transformation (HT) occurs in 20-25% of cases
  - Ischemia damaged endothelium becomes “leaky”

- CT
  - Wedge-shaped area more sharply defined
  - Mass effect increases with herniation in some cases
  - HT seen as gyriform or basal ganglia hyperdensity

- MR
  - Increased T2/FLAIR signal and swelling
  - Enhancement typical (2-2-2 rule).
  - DWI remains bright, though ADC gradually becomes less dark
Know when to consult Neurosurgery

- Complications from stroke usually occur in the subacute setting
  - Mass effect, herniation $\rightarrow$ decompressive craniectomy
  - Hydrocephalus $\rightarrow$ ventriculostomy
  - Hemorrhagic transformation larger than 30cc $\rightarrow$ decompression
Imaging in Chronic infarct

- Beyond 2-3 weeks
- Progressive volume loss and gliosis (scar)

CT
- Sharply delineated wedge-shaped hypodense area in a vascular territory.
- Adjacent sulci are ventricle enlarged.

MR
- Near CSF equivalent signal intensity.
- Peripheral rim of increased FLAIR signal (gliosis)
- Wallerian degeneration with an ipsilateral small cerebral peduncle.
- No restricted diffusion.
Miscellaneous Stroke Subtypes

- Cardiac and atheromatous emboli
- Lacunar infarcts
- Watershed
Cardiac and atheromatous emboli

- Simultaneous small infarcts in multiple different vascular distributions.
- Heart is most common source.
- Ipsilateral hemispheric emboli most commonly due to ICA
- Fat emboli rare but can occur following long bone fracture
- Emboli usually land near the gray/white junction
Lacunar Infarcts

- 25% of all ischemic strokes
- 15mm or less
- Atherosclerotic occlusion of tiny perforating branches
  - Very small end arteries with few collaterals
- May or may not be symptomatic

- CT
  - Usually not visible acutely.
  - Chronic \(\rightarrow\) small “holes” in the deep gray or central white matter
- MR
  - T1 dark and T2 bright “holes”
  - Periphery is bright on FLAIR
  - Restricted diffusion acutely
Watershed Infarct

- Occur at the junction between 2 arterial distributions
- 10-12% of infarcts
- Secondary to hypotension and/or severe proximal stenosis, most commonly the ICA
- Hypoperfusion and microemboli both proposed etiologies
- Imaging
  - **Internal border zone**- Several small infarcts in a linear fashion parallel to the lateral ventricle in the periventricular white matter.
  - **External border zone**- Small or confluent infarcts at the junction of the ACA/MCA or MCA/PCA
Craniocervical Artery Dissection

- Vessel wall tear permitting blood to penetrate into the wall layers, causing luminal narrowing/occlusion.
- Often associated with thrombosis, embolism, and pseudoaneurysm
- Cause 2% of ischemic strokes
- 60% are spontaneous, others post-traumatic
- Underlying collagen vascular disease predisposes
- Extracranial ICA most common followed by vertebral artery
Cerebral Venous Thrombosis

- 0.5-1% of all acute strokes
- OCPs, hypercoagulable state, pregnancy, dehydration, infection
- Transverse sinus most common, followed by SSS
- Most common in young women

Imaging
- Hyperdense vein on NECT
- Filling defect on CTA/CTV
- Loss of “flow void” on MRI
- Infarct in venous, not arterial territory
- More vasogenic, less cytotoxic edema
- More prone to hemorrhage.
Spontaneous Intracranial Hemorrhage (sICH)

- 15% of all strokes
- 70-80,000 /yr
- Common etiologies vary based on age
  - Young adults
    - Vascular malformation
    - Drug abuse
  - Middle aged and elderly
    - Hypertension
    - Amyloid angiopathy
    - Neoplasm
    - Coagulopathy
Role of Imaging in sICH

- Identify presence and location of a clot
- Age the clot
- Identify mass effect and hydrocephalus
- Detect other findings that may be clues to its etiology
- If hemorrhage etiology is not clear by location/morphology, secondary imaging (MRI/CTA) necessary.
- Know when to consult neurosurgery
CT

- Hyperacute (Minutes to Hours) – iso or even hypodense to brain
- Acute (Hours-days) – Hyperdense
- Subacute (Days-weeks) – Gradually becomes less dense. After 10-14 days, the hematoma will become similar in density to brain. Can get “ring enhancement”
- Chronic - Small hemorrhages may become invisible. Larger ones leave a “slit-like” defect.
Hypertensive Hemorrhage

- Accounts for ~50% of sICH in adults
- Accelerated atherosclerosis → formation of small pseudo-aneurysm ("Charcot-Bouchard aneurysms"). Rupture → hemorrhage
- Location
  - Putamen/External capsule (striatocapsular) (50-60%)
  - Thalamus (15-25%)
  - Pons (10%)
  - Cerebellum (dentate nucleus) (10%)
  - Lobar hemorrhage (5%)
- Microbleeds tend to cluster in the basal ganglia and cerebellum
Amyloid Angiopathy

- 20% of hemorrhages in patients >60
- Mean age 73
- The most common of 3 varieties of cerebral amyloid deposition disease
- Amyloid beta accumulates in small vessels

- Imaging
  - Lobar hemorrhage
  - Multiple white matter lesions
  - Multiple punctate microhemorrhage, more peripheral than in hypertension
Vascular Malformations

- Diverse group of lesions, some of which are prone to rupture

- Types associated with hemorrhage
  - Arteriovenous malformation (AVM)
  - Dural AV fistula
  - Cerebral Cavernous malformation
Hemorrhagic Neoplasm

- 10% of sICH
- Usually a high-grade GBM or hemorrhagic mets from an extracranial primary such as renal cell carcinoma.
In Summary

- Time is brain
- Know and utilize the new stroke imaging algorithm
- Identify early signs of stroke
- Know the contraindications to TPA
  - Hemorrhage
  - Large amount of edema
  - Stroke mimicker
- Understand strengths/weaknesses of CT/MR and appropriate ordering practices
- Know when to consult neurosurgery.
References

- Osborn, Anne. *Osborn’s Brain: Imaging, Pathology, and Anatomy* Amirsys Jan 2013