Imaging of Stroke and Stroke Mimickers
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Outline

• Review imaging characteristics and cases of stroke on CT and MR
• Review lesions that can mimic stroke on imaging

Stroke

• In the United States, stroke is a leading cause of death, killing nearly 130,000 people each year, and a leading cause of serious, long-term adult disability.

• There are an estimated 7,000,000 stroke survivors in the U.S. over the age of 20.

• Approximately 795,000 strokes will occur this year, one occurring every 40 seconds, and taking a life approximately every 4 minutes.
Major Clinical Stroke Subtypes

- Large Artery
- Cardioembolic
- Small Vessel

**Major Clinical Stroke Subtypes (Large Artery)**

- Large artery atherosclerotic strokes represent ~40% of strokes and can arise from thrombosis at the site of a plaque or from emboli produced from a plaque within a more proximal vessel.

- Most common site of atherosclerotic plaque is at the carotid bifurcation with involvement of the distal common carotid artery and the 1st 2 cm of the ICA.

- Other common locations include: carotid siphon and proximal MCA and ACA.

**Major Clinical Stroke Subtypes (Cardioembolic)**

- Cardioembolic disease accounts for 15-25% of ischemic strokes.

- Risk factors include: myocardial infarction, ventricular aneurysm, atrial fibrillation or flutter, cardiomyopathy, and valvular heart disease.
Major Clinical Stroke Subtypes (Small vessel)

- Lacunar infarcts are small in size (<15mm), typically involve the basal ganglia and thalamus, and account for 15-30% of all strokes.

- Lacunar infarcts are often multiple and are due to embolic, atheromatous, or thrombotic lesions in the single penetrating end arterioles that supply deep gray nuclei, including the lenticulostriate and thalamoperforating arteries.

Acute Stroke Imaging

- Initial Non-contrast CT head

- Role:
  1. Exclude intracranial hemorrhage
  2. Evaluate for early signs of ischemia involving more than one-third of the middle cerebral artery territory
  3. Exclude a large mass resulting in the patient's symptoms

Acute Stroke on CT

- Hyperdense vessel (high specificity, low sensitivity)
  - Represents acute thrombus in cerebral vessel

- Loss of GM-WM distinction in 1st 3 hours:
  - Obscuration of deep gray nuclei
  - Subtle loss of normal cortical high density
Acute Stroke Imaging

- CT angiogram
  - Role:
  - Evaluate for large vessel occlusion involving the ICA, proximal MCA, or basilar artery.
  - If a large vessel is occluded the patient may be a candidate for intraarterial therapy.

Acute Stroke Imaging

- MR Brain
  - Role:
  1. Evaluate for the presence and size of the acute infarct.

Acute Stroke on MR

- Restricted diffusion develops shortly after the time of the ischemic event.
- Parenchymal FLAIR hyperintensity develops 6 hours after the infarct.
- Cortical swelling and T2 hyperintensity develops 12-24 hours after the infarct.
- GRE is helpful to evaluate for acute blood products within the infarct bed.
Subacute Infarct on MR

• Subacute infarct is defined as approximately 2-14 days following initial ischemic infarct.

• DWI - Restricted diffusion initially, low ADC values gradually increase as infarct proceeds through subacute stage.

• T2 - Hyperintense edema with mass effect. “Fogging effect” T2 signal can temporally normalize 1-2 weeks following infarct.

• T1C+ - Gyriform enhancement can be seen as early as 2 days post infarct and peaks at 2 weeks post infarct. Enhancement should resolve by 2 months.
Chronic Infarct on MR

- Chronic infarct results in volume lose with gliosis along affected margins.
- DWI: No diffusion restriction
- T2: Volume loss and hyperintense signal
- FLAIR: Low signal in encephalomalacic area with hyperintense gliotic white matter at margins of the infarct
- T1C+: No enhancement
Stroke Mimickers

- Pathology that can mimic an acute arterial infarct on CT and/or MR

- Examples include:
  - 1. Venous Infarct
  - 2. Hypoxic-Ishemic Injury (HII)
  - 3. Creutzfeldt-Jakob Disease (Prion Disease)
  - 4. Multiple Sclerosis (MS) and Acute Disseminated Encephalomyelitis (ADEM)
  - 5. Posterior reversible encephalopathy syndrome (PRES)
  - 6. Hypoglycemia
  - 7. Carbon monoxide poisoning
  - 8. Septicemia
  - 9. Hematoma
  - 10. Osmotic Demyelination Syndrome
  - 11. Tumors

Venous Infarct

Venous infarct is usually the result of central venous thrombosis, complicating both dural venous sinus thrombosis and deep cerebral venous thrombosis. Any other cause of venous occlusion can also lead to venous infarction, including trauma and surgical ligature.

Common Causes:

- Neonates: Shock and dehydration
- Older children: Infection
- Adults: Coagulopathy

Differentiation from Arterial Strokes: Venous infarcts will not follow arterial vascular territories and are more commonly hemorrhagic.

Venous Vascular Territories

http://www.radiologyassistant.nl
Venous Infarct

Venous Infarct

Venous Infarct
Stroke Mimickers
Hypoxic-Ischemic Injury (HII)

- Etiologies include: Cardiac arrest, drowning, asphyxiation.
- Injury patterns are highly variable depending on brain maturity, severity and length of insult.
- Mild to moderate insults commonly will result in watershed zone infarcts.
- Severe insults will commonly result in infarcts of the gray matter structures (basal ganglia, thalamus, cortex, cerebellum, hippocampi).
- Affected structures demonstrate diffusion restriction and hyperintense T2/FLAIR signal.

Differentiation from Arterial Stroke: Hypoxic ischemic injury will often involve multiple vascular territories.
Stroke Mimickers

Creutzfeldt-Jakob Disease (CJD)

- Rapidly progressing, fatal, potentially transmissible dementia caused by a prion.
- MR: Diffusion restriction and hyperintense T2/Flair signal involving cerebral cortex, basal ganglia, and thalamus. No enhancement or mass effect.
- No effective treatment
- Can mimic hypoxic-ischemic injury however clinical picture is much different.
- Differentiation from Arterial Stroke: CJD will often involve multiple vascular territories.
Stroke Mimickers

Multiple Sclerosis

- Probable autoimmune-mediated demyelination in genetically susceptible individuals.
- MRI: Bilateral, asymmetric, linear/ovoid hyperintensities. >85% periventricular / 50-90% callososeptal interface / May also commonly involve subcortical U fibers, brainstem, spinal cord.
- Enhancement and restricted diffusion represent active demyelination.

Differentiating from arterial stroke: MS plaques often will demonstrate rim restricted diffusion rather than the solid restricted diffusion that is seen in white matter infarcts. History and multiple additional MS plaques within the brain also help discriminate from stroke when making the diagnosis.

Multiple Sclerosis with Active Demyelination
Stroke Mimickers

**Acute Disseminated Encephalomyelitis (ADEM)**

- Autoimmune-mediated white matter demyelination of brain and/or spinal cord, usually with remyelination.
- Best clue: Multifocal WM and deep gray lesions 1-2 weeks following infection/vaccination. Mean age 6-8 years, but can occur at any age.
- Can involve brainstem and posterior fossa. Does not usually involve callosomarginal interface.
- Tumefactive, mass-like lesions possible.
- Spinal cord involvement in up to 30%.
- C+ MR: Punctate, ring, incomplete ring, or peripheral enhancement. Diffusion restriction uncommon.

**Differentiation from Arterial Stroke:** ADEM generally presents as multiple lesions predominantly involving white matter territories and cortex, whereas arterial stroke tends to affect specific vascular territories. Clinical presentation also helps differentiate from arterial infarct.
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Posterior Reversible Encephalopathy Syndrome (PRES)

- Cerebrovascular autoregulatory disorder
- Many etiologies with HTN as common component
  - Preeclampsia, eclampsia, chemotherapy, uremic encephalopathies

MR: Most common presentation: Multiple T2/Flair hyperintensities involving cortex and subcortical white matter. Predilection for posterior circulation (parietal, occipital lobes, cerebellum). Often bilateral but asymmetric.

MR: Restricted diffusion is relatively uncommon in PRES.

Differentiation from Arterial Stroke: Regions of abnormal signal in PRES generally will involve multiple vascular territories.
Stroke Mimickers
Adult Hypoglycemia

- Imbalance between glucose supply and utilization leading to brain injury.
- Often presents as coma in an adult diabetic on insulin replacement therapy.
- Location: Common: Cortex of parietal/temporal/occipital/mesial temporal lobes. Usually will spare thalami and white matter.
- MR: Cortical swelling, sulcal effacement, T2/Flair hyperintense signal and diffusion restriction.
- Differentiation from Arterial Stroke: Acute infarcts often are wedge shaped, will follow a vascular territory, and commonly will involve adjacent white matter.

Adult Hypoglycemia

http://radiopaedia.org/

Stroke Mimickers
Carbon Monoxide Poisoning

- Anoxic-ischemic encephalopathy, usually with bilateral lesions, caused by inhalation of CO gas.
- Location: Most common: Bilateral medial basal ganglia (Globus Pallidi). Cerebral white matter around most common location involved.
- MR: Acute stage of COP: T2/Flair signal hyperintensity and diffusion restriction.
- MR: Delayed stage of COP (weeks post exposure): Patients can develop diffusion restriction in white matter and corresponding delayed encephalopathy.
- Differentiation from Arterial Stroke: COP often involves multiple vascular territories.
Carbon Monoxide Poisoning

Stroke Mimickers

Seizure

- Transient seizure-related MR changes which represents reversible post-ictal cerebral edema.
- Location: Supratentorial, related to epileptogenic focus. May focally involve the hippocampus and splenium of the corpus callosum.
- MR: T2/Flair hyperintensity and diffusion restriction in gray matter and/or subcortical white matter with mild mass effect.
- Differentiation from Arterial Stroke: Clinical presentation and signal abnormality will not follow a vascular territory.
Seizure

Stroke Mimickers
Cerebritis

- Focal pyogenic infection of brain parenchyma, typically bacterial; fungal or parasitic less common.
- 4 pathologic stages: Early cerebritis (which can mimic stroke), late cerebritis, early capsule, late capsule.
- Imaging varies with stage of abscess development
- Early cerebritis: ill-defined T2 hyperintense mass and patchy enhancement, restricted diffusion
- Late cerebritis: intense, irregular rim enhancement
- Early capsule: well defined, thin-walled, enhancing rim (early abscess)
- Late capsule: cavity collapses, capsule thickens (late abscess)
- Differentiation from arterial stroke: clinical presentation and signal abnormality will not follow a vascular territory.

Cerebritis
Stroke Mimickers
Herpes Encephalitis (HSV-1)

- Brain parenchyma infection caused by herpes simplex virus type 1 (HSV-1). Typically reactivation in immunocompetent patients.
- MRI: T2/FLAIR hyperintensity of the limbic system (mesial temporal and inferior frontal cortex) with diffusion restriction. Often bilateral but asymmetric.
- T1C+: May see mild, patchy enhancement early. Gyriform enhancement usually seen 1 week after initial symptoms.

Differentiation from Arterial Stroke: Acute infarct should follow vascular territory (MCA, ACA, PCA). Hyperacute symptoms in acute infarct vs 2-3 day history of flu-like illness in HSV-1.
Osmotic Demyelination Syndrome

- Acute demyelination caused by rapid shifts in serum osmolality.
- Location: 50% central pons / 50% extrapontine sites: basal ganglia and cerebral white matter.
- MR: T2/Flair confluent hyperintensity and restricted diffusion in central pons with sparing of the periphery and corticospinal tracts. Similar symmetric signal in basal ganglia and white matter.
- Differentiation from Arterial Stroke: Pontine infarcts are often asymmetric and usually involves both central and peripheral pontine fibers.

Osmotic Demyelination Syndrome

- [Image]

Osmotic Demyelination Syndrome

- [Image]
Stroke Mimickers

Tumor

- Many primary and secondary brain tumors may restrict diffusion secondary to high cellularity.
- Examples: GBM, Anaplastic Astrocytoma, CNS Lymphoma.
- Epidermoid Cysts will also demonstrate restricted diffusion secondary to microstructure of parallel-layered keratin filaments and flakes.
- Differentiation from Arterial Stroke: Acute strokes will not have nearly as much mass effect.

Glioma

CNS Lymphoma
CNS Lymphoma

Epidermoid

Epidermoid
Conclusion

- Stroke is much more common than the "stroke mimickers".
- Acute strokes will restrict diffusion and follow a vascular territory.
- Stroke mimickers often demonstrate restricted diffusion but in an atypical fashion and not within a single vascular territory.

References

Thank You

Questions?