# CVA

- Best diagnostic clue: Diffusion restriction with correlating ADC map
- Location: One or more vascular territories, or at border-zones ("watershed")
- Morphology: Wedge shaped when gray matter involved, variable white matter involvement
- DWI/PWI "mismatch" = "penumbra" or "at risk" tissue
- Conventional MR sequences positive in 70-80%
- Restricted diffusion improves accuracy to 95%
- Best imaging tool: MR + T2*, DWI; NECT, perfusion CT, CTA if MR not available
- DSA with thrombolysis in selected patients

# CVA - CT

- Hyperdense vessel on NECT (high specificity, low sensitivity)
  - Caused by acute thrombus in cerebral vessel(s)
  - Hyperdense M1 MCA in 35-50%
  - "Dot sign" = occluded MCA branches in sylvian fissure (16-17%)
- Loss of gray-white matter distinction in first 3 hrs seen in 50-70%
  - Obscuration of deep nuclei
  - Loss of insular "ribbon"
- Parenchymal hypodensity on NECT
  - If > 1/3 MCA territory initially, large lesion later
  - Temporary transition to isodensity (up to 54%) at 2-3 weeks post-ictus = CT "fogging"
- Gyral swelling, sulcal effacement 12-24 hrs
- "Hemorrhagic transformation" in 15-45%
  - Delayed onset (24-48 hrs) is most typical
  - Can be gross (parenchymal) or petechial

# CVA - MRI

- T1WI: Early cortical swelling & hypointensity, loss of gray-white borders
- T2WI
  - Early cortical swelling, hyperintensity in affected distribution
  - May normalize 2-3 weeks post-ictus = MR "fogging"
- PD/Intermediate: Loss of flow voids = slow flow vs occlusion
- FLAIR
  - May be positive (hyperintense) when other sequences normal (as early as 6 hrs post-ictus)
  - MR intra-arterial signal on FLAIR = early specific sign of major vessel occlusion
- T2* GRE
  - Sensitive for detection of acute blood products
  - Shows thrombosed vessel as arterial "blooming" from clot susceptibility

# CVA – MRI

- DWI
  - Hyperintense restriction from cytotoxic edema
    - DWI improves hyperacute stroke detection to 95%
    - Usually correlates to "ischemic core" (final infarct size); some diffusion abnormalities reversible
    - May have reduced sensitivity in brainstem and medulla in first 24 hours
    - High signal can persist up to 57 days post-ictus, (after 10 days, T2 effect may predominate over low ADC = "T2 shine-through")
  - Corresponding low signal on ADC maps
    - May normalize after tissue reperfusion
    - Note: Hyperintensity on ADC map (T2 "shine-through") may mimic diffusion restriction
  - Distinguishes cytotoxic from vasogenic edema in complicated cases; especially helpful for evaluation of new deficits following tumor-resection
- T1 C+
  - Variable enhancement patterns evolve over time
    - Immediate: Intravascular enhancement (stasis from slow antegrade or retrograde collateral flow)
    - Early: Meningeal enhancement (pial collateral flow appears in first 24-48 hrs, then resolves over 3-4 days)
    - Late acute: Parenchymal enhancement (appears after 24-48 hrs, can persist for weeks/months)
- MRA: Demonstrates major vessel occlusions, stenoses, collateral status

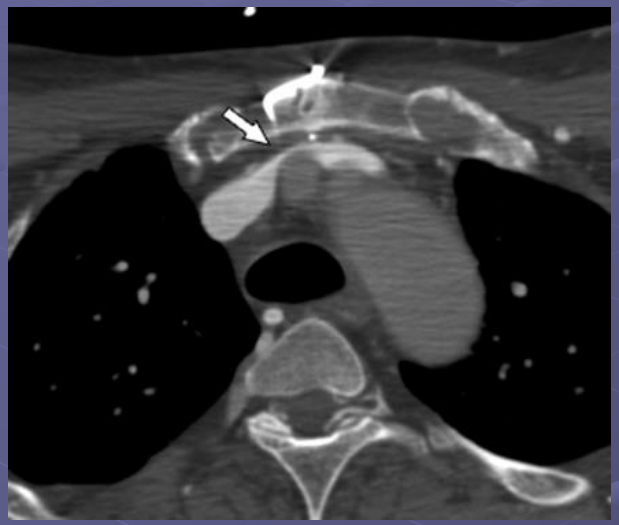
# CVA - Ddx

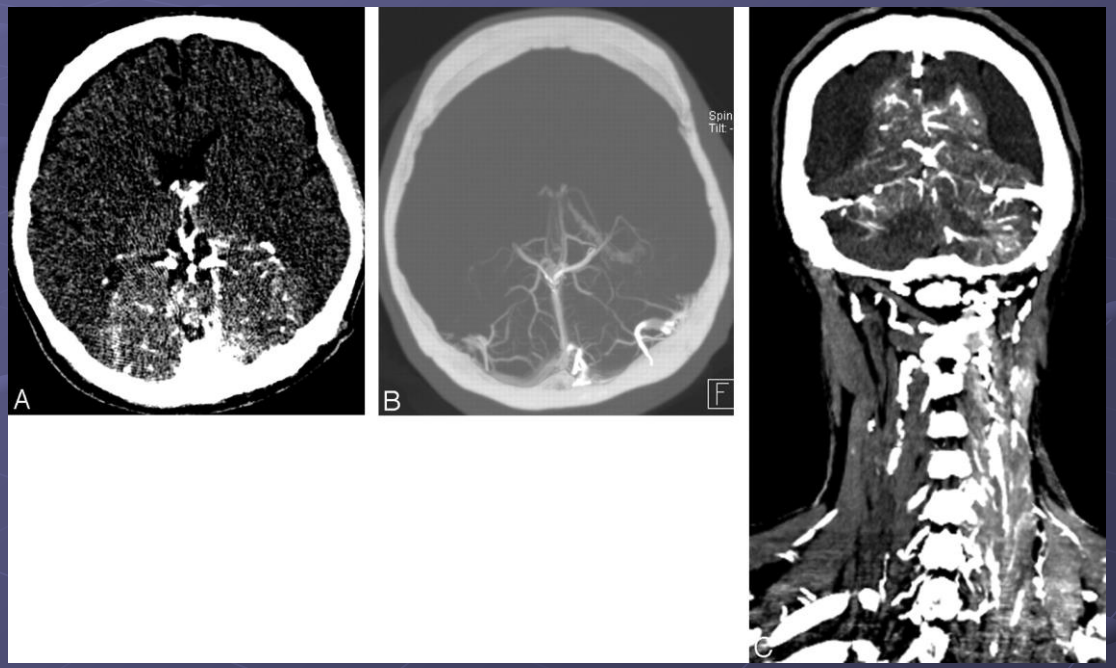
- **Parenchymal hypodensity (nonvascular causes)**
  - Infiltrating neoplasm (e.g., astrocytoma)
  - Cerebral contusion
  - Inflammation (e.g., cerebritis or encephalitis)
  - Evolving encephalomalacia

# MCA Infarct

- 0-2 Hours
  - Almost 60% of CT scans obtained within the first few hours of cerebral infarction are normal
- 2-6 Hours
  - Dense MCA sign
  - Loss of the insular ribbon sign (gray-white interface loss along the lateral insula )
- 6-12 Hours
  - Effacement of the sulci
- 12-24 Hours
  - Decreased attenuation
- Maximal Swelling at 3-7 days

# Pseudo pathologic Parenchymal Enhancement (left sided injection)

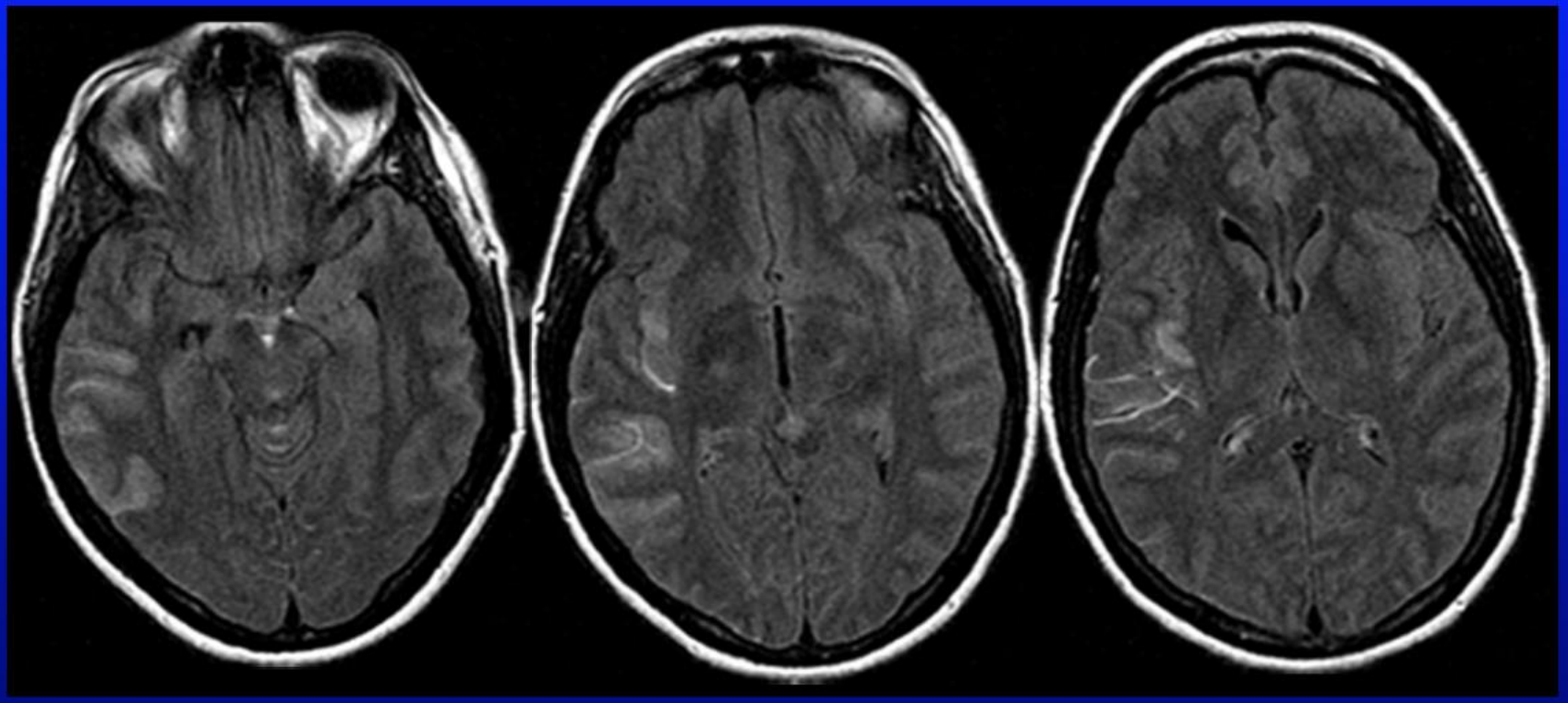





- Pseudopathologic Brain Parenchymal Enhancement due to Venous Reflux from Left-Sided Injection and Brachiocephalic Vein Narrowing

# Hyperintense Vessels on FLAIR

Sluggish flow in poorly developed collaterals



FLAIR