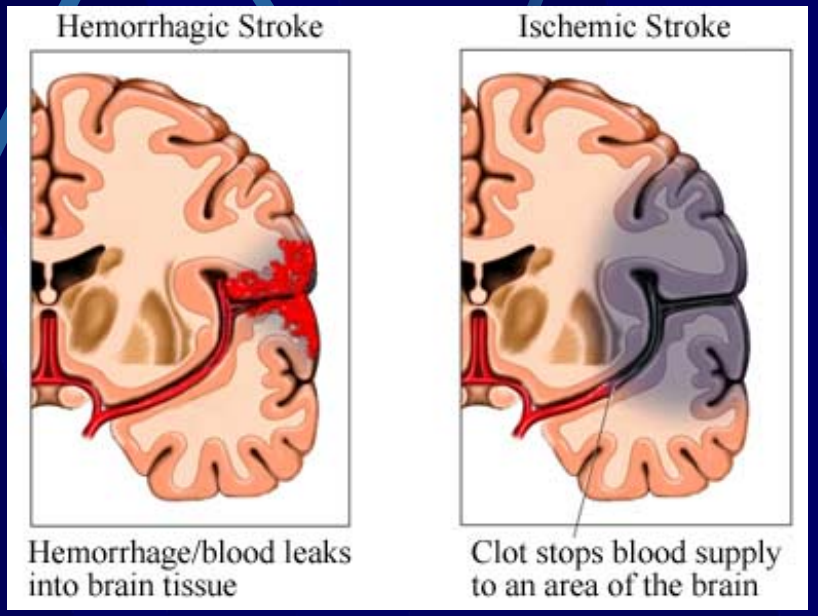
Cerebrovascular Accident

- Discussion primarily involves Ischemic CVA
- Radiology imaging discussion will cover evolution of lesions and their appearance over time with various modalities



- Fawzi Elsagga

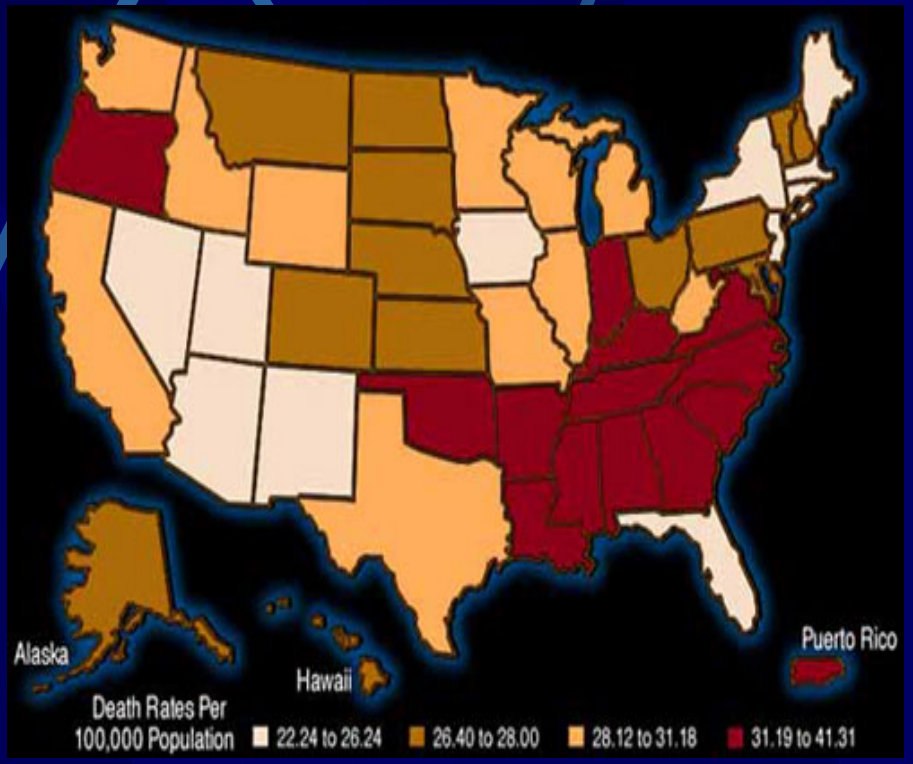
OVERVIEW

EPIDEMIOLOGY

Stroke is #3 cause of mortality

“The Stroke Belt”

The incidence and prevalence of stroke are equal among women and men, but greater mortality in women and African Americans



RISK FACTORS

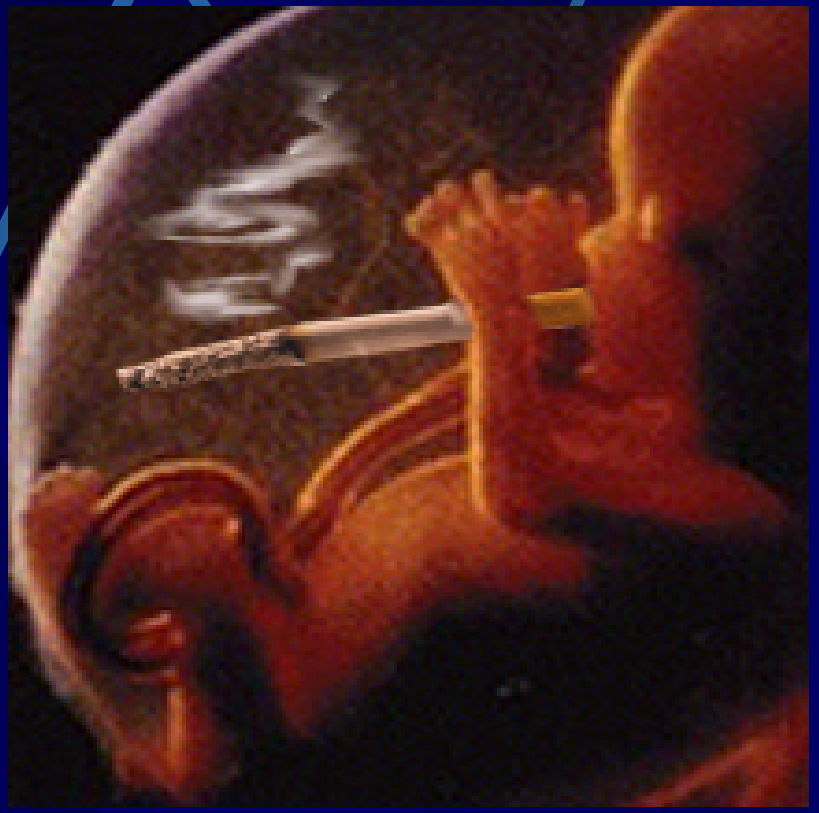
- Factors that cannot be changed:

  Male race, Increased age, Personal hx of CVA or TIA, Family hx of CVA, TIDM, current Asymptomatic carotid bruits

- Treatable Factors

Major: Hypertension, Heart disease, esp. atrial fibrillation, Cigarette smoking Transient ischemic attacks

Secondary: Increased cholesterol /lipids Physical inactivity, Obesity



STROKE TYPES

Stroke Definition=clinical term for sudden, focal neurological deficit lasting more than 24h (TIA lasts<30minutes)

2 Main Types: Ischaemic or Hemorrhagic:

1.Ischaemic—comprise 80% of all strokes

2. Hemorrhagic—comprise 20%

Ischaemic Strokes

Ischaemic Stroke--accounts for 80% of all strokes

1. 50% of these strokes are caused by cerebral thrombosis
   a.) 30% are Large-vessel thrombosis
   b.) 20% involve small, deeply penetrating arteries (lacunar stroke)
2. Approximately 30% of Ischaemic strokes are caused by cerebral embolism. Embolic strokes, which are more common in younger patients, develop rapidly, with maximum deficit usually present within seconds to minutes

Hemorrhagic Stroke

- Account for 20% of all strokes
- Primarily the result of:

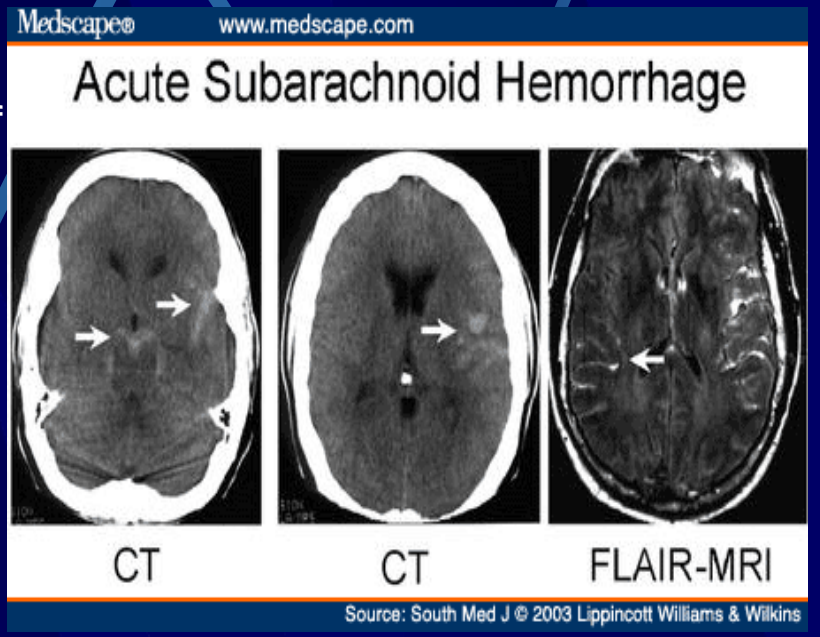
1. intracerebral hemorrhage— HTN
2. subarachnoid hemorrhage—rupture of aneurysm or AVM

Hyperacute Infarct on CT

In cases of hyperacute stroke (0-6h), CT is usually not sensitive for cerebral infarction.

It is sensitive in identifying various forms of acute intracranial hemorrhage and other gross lesions that would preclude the use of thrombolytic therapy

- Contraindications to tPA: hemorrhage, early clues that infarction is very large(relative contraindication) changes occupying >1/3 of vessel territory
- hyperdense MCA sign
- The CT shows hyperdensities (arrows) in the basal cisterns and left sylvian and frontal cortical sulci, consistent with acute subarachnoid hemorrhage.

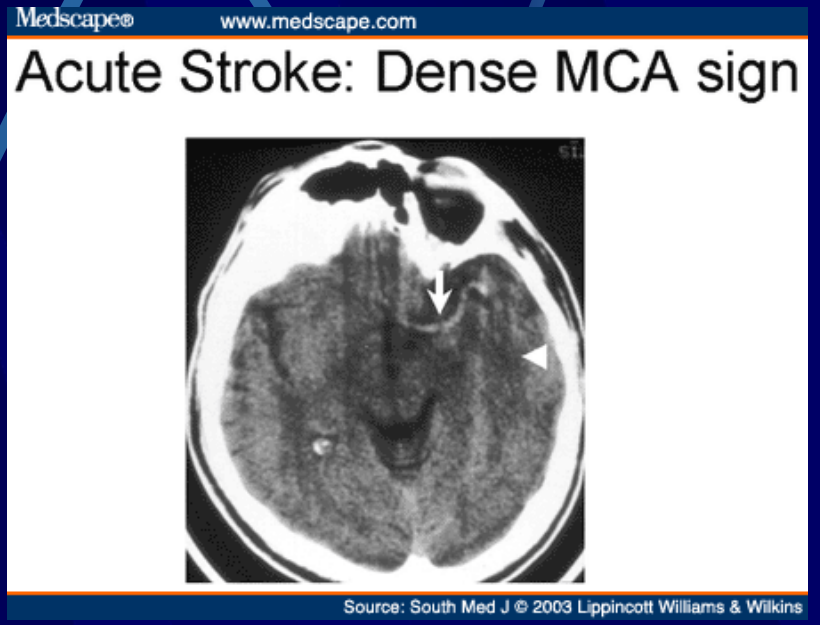


CT Angio

- **CT angiography (CTA) is a 3-dimensional reconstruction of the cerebral vasculature**

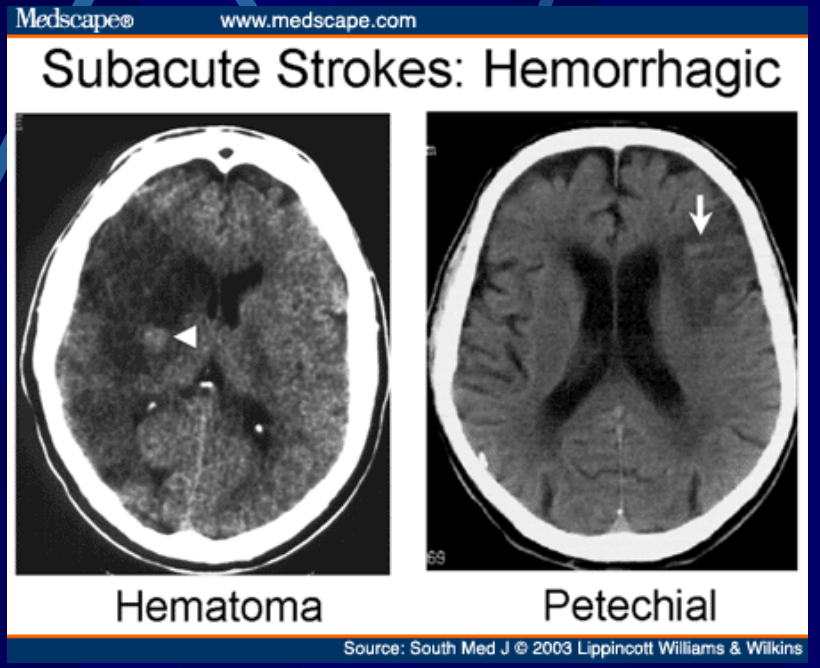
Acute Infarct on CT

- In the acute period (6-24 h), the changes of ischemia become more apparent on the noncontrast CT scan. The loss of gray-white interface, sulcal effacement, hypodensity of basal ganglia, and hypodensity of the insular cortex become prominent.
- In severe cases, edema and mass effect can appear at this stage
- Ex: 8 hours after onset of left-sided weakness and aphasia.



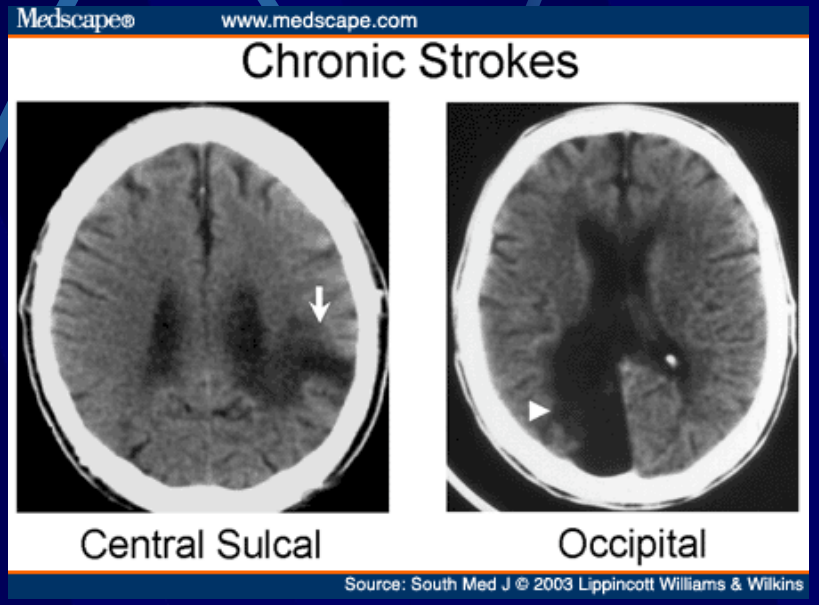
Subacute Infarcts on CT

- During the subacute period (1-7 d), there is increasing edema and mass effect.
- Edema and mass effect peak at 1 to 2 days and then decline
- The first 1 to 2 days are also when risk of hemorrhagic transformation peaks



Chronic Infarcts on CT

- **Chronic infarctions are characterized by marked hypodensity and lack of mass effect on CT scans; the density is similar to CSF**
- **Ex: MCA and PCA strokes**



Role of Advanced MRI Techniques in Hyperacute Stroke

- **Traditional MRI is Not initial study for Hyperacute Stroke, because want to efficiently r/o bleed for emergent management**
- **But, Advanced techniques can detect very early changes**
- **1. Diffusion Weighted Images--In the first few minutes after vascular occlusion, cytotoxic edema develops in the ischemic tissue, resulting in a net shift of water molecules into the intracellular compartment. This shift decreases the diffusion characteristics of the hydrogen ions in the ischemic brain and results in a precipitous drop in the apparent diffusion coefficient (ADC). The Construction of Diffusion Weighted Images (DWI) is based on this concept**
- **DWI is highly sensitive in identifying hyperacute and acute cerebral infarction, showing ischemia within minutes of vascular occlusion, while conventional MRI typically does not show ischemic changes until several hours after ictus**

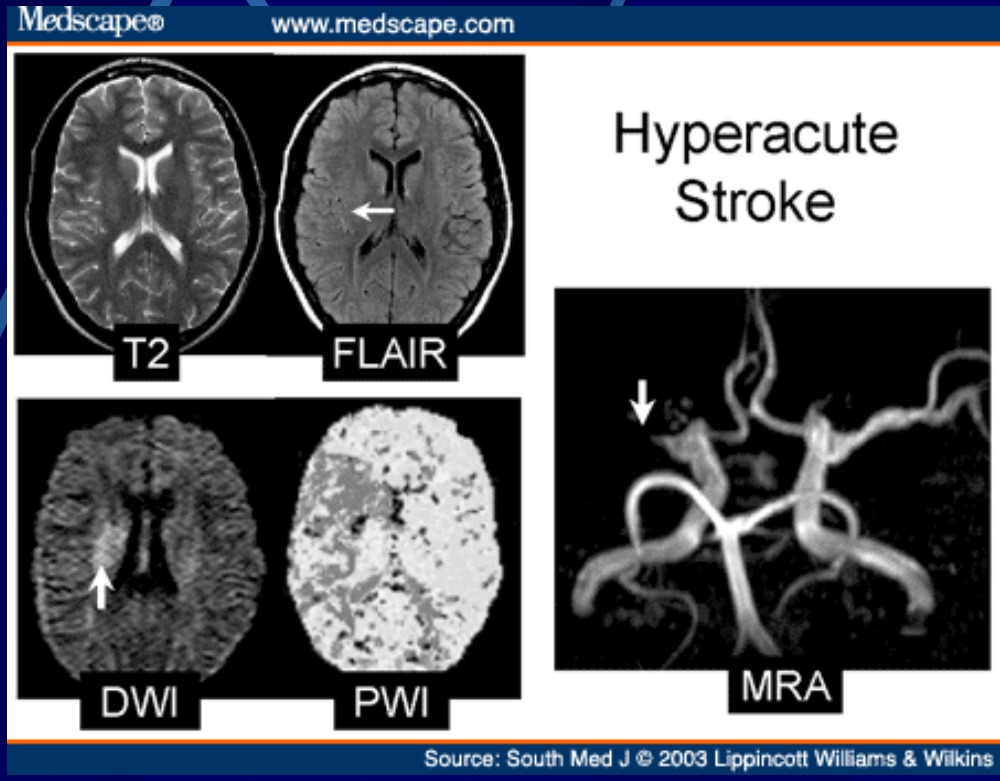
Hyperacute Infarcts on MRI

- **2. Perfusion-Weighted Imaging (PWI)--The imaging principle is to detect the susceptibility effects of a rapidly administered intravenous contrast (gadolinium) bolus as it passes through the cerebral microvasculature.**
- **PWI may show hypoperfusion in a much larger area of tissue than shown by the DWI. This indicates a much larger area of tissue is at risk for infarction, a "diffusion-perfusion mismatch“**

- **3. Fluid-Attenuated Inversion Recovery (FLAIR)--MRI has had important applications in the imaging of stroke patients. FLAIR depicts areas of tissue T2 prolongation while suppressing CSF signal, offering a sensitive method to detect lesions. Hyperintensity of the ischemic brain in acute strokes is seen on FLAIR as early as 4 to 6 hours after ictus at a time when T1-weighted images (T1WI) and T2-weighted images (T2WI) are usually normal**

Hyperacute Infarct on MRI

- Ex: 2 hours postictus. The T2-weighted image is normal. The FLAIR image shows hyperintense vessels in the territory of the (MCA) (arrow), consistent with slow arterial flow, but the FLAIR shows no tissue abnormality. Diffusion-weighted imaging (DWI) shows hyperintensity in the deep middle cerebral artery territory consistent with cytotoxic edema in an acute stroke
- Perfusion-weighted imaging (PWI) shows reduced perfusion in the full (cortical and subcortical) MCA territory. This suggests a much larger area of tissue at risk than shown by DWI or conventional images. The MRA confirms a proximal MCA occlusion or severe stenosis



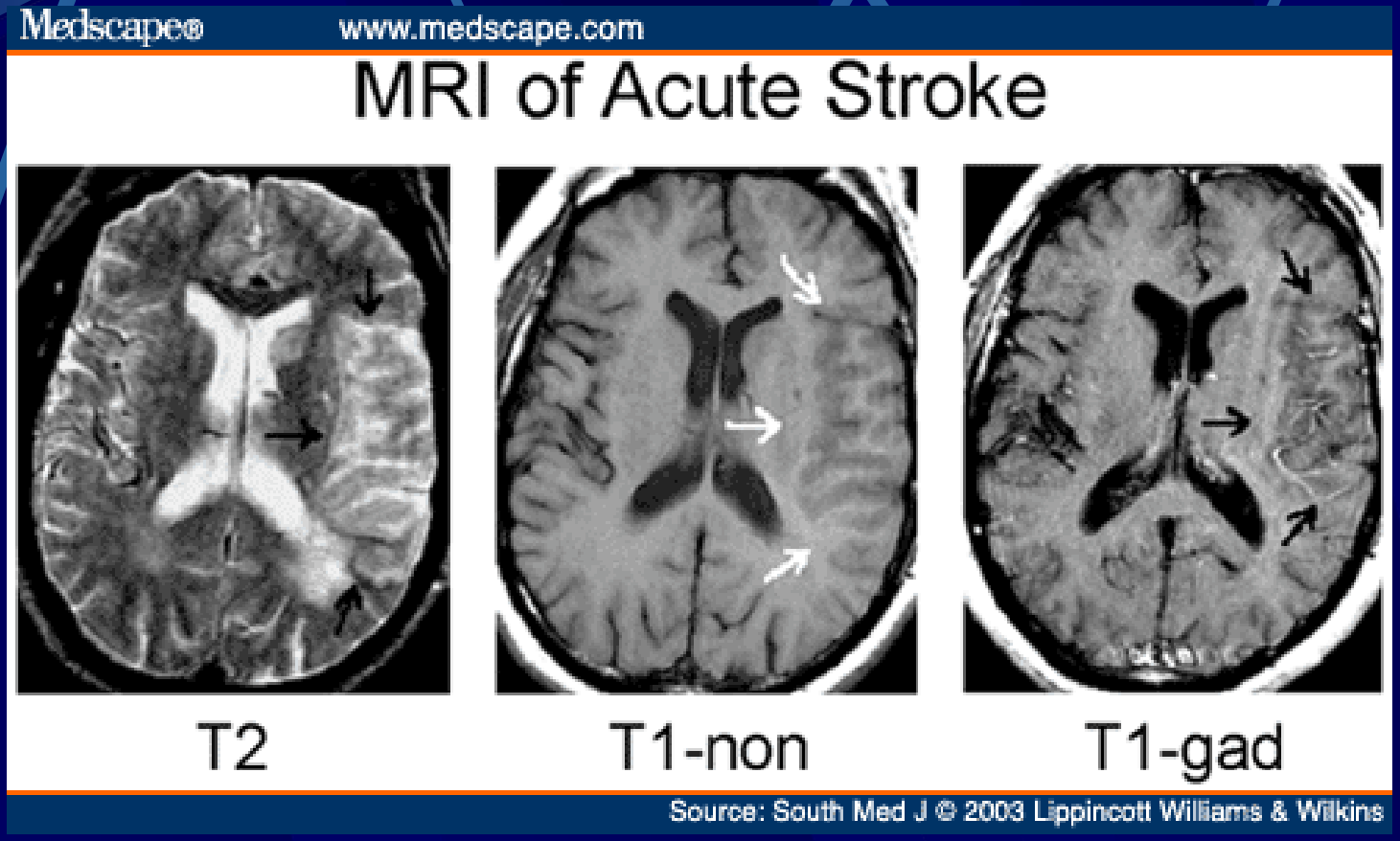
Chronic Infarct on DWI

- In the chronic stage, there are no cells left in the core of the infarct, and the water molecules are almost exclusively extracellular b/c there is no longer flow being created by edema. Hence, chronic infarcts are characterized by high ADC values and appear dark on DWI

Acute Infarct on MRI

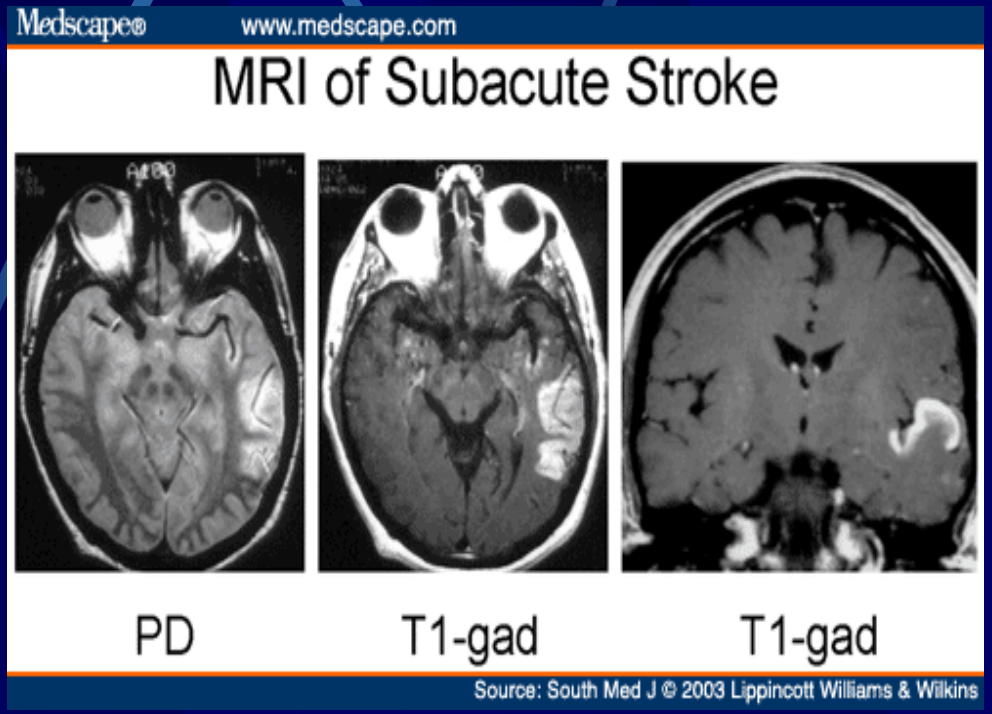
- At this time point (6-24 h), tissue ischemia/infarction is well developed on FLAIR images and begins to show on T2WI (hyperintensity) and T1WI (hypointensity). Hyperintensity develops on T2WI as early as 8 hours after infarction due to cytotoxic and vasogenic edema. Hypointensity on noncontrast T1WI is usually seen 16 to 24 hours after ictus and, again, is related to both cytotoxic and vasogenic edema.
- Ex: MCA stroke 12h post

Acute Infarct on MRI



Subacute Infarct on MRI

- After the first 24 hours, T1WI, T2WI, FLAIR, and contrast-enhanced images are most useful in subacute and chronic stroke, where the focus shifts from identifying the presence and extent of infarct and ischemic penumbra to identifying the underlying pathophysiology
- Infarctions continue to appear as areas of hypointensity on T1WI and hyperintensity on T2W. In general, mass effect is best appreciated on T1WI.
- As the infarct evolves during the first week, the edema and mass effect increases, and both the morphologic and signal changes seen in the magnetic resonance sequences become more prominent and well demarcated



Chronic Infarct on MRI

- By the chronic stage, edema has resolved and volume loss occurs in the area of infarction, beginning 1 month postictus. Tissue loss leads to ipsilateral ex vacuo ventricular enlargement and widening of cortical gyri and fissures in the area of the infarct
- After several months, wallerian degeneration occurs.
- After several years, dystrophic calcification may appear bright on T1WI.

Summary

- Various neuroimaging modalities are playing an increasing role in the evaluation of patients with hyperacute stroke (presenting within 0 to 6 hours) when there is a possibility of clinical benefit from reperfusion.
- primary modality in this setting is a noncontrast CT scan - to rule out cerebral hemorrhage and recent cerebral infarctions
- CT perfusion if available, could help define the tissue at risk for infarction
- In the future, MRI is likely to become the modality of choice in evaluating hyperacute stroke. MRI can help define the acutely ischemic region (DWI), the tissue at risk for further ischemia (PWI), vascular anatomy (MRA),