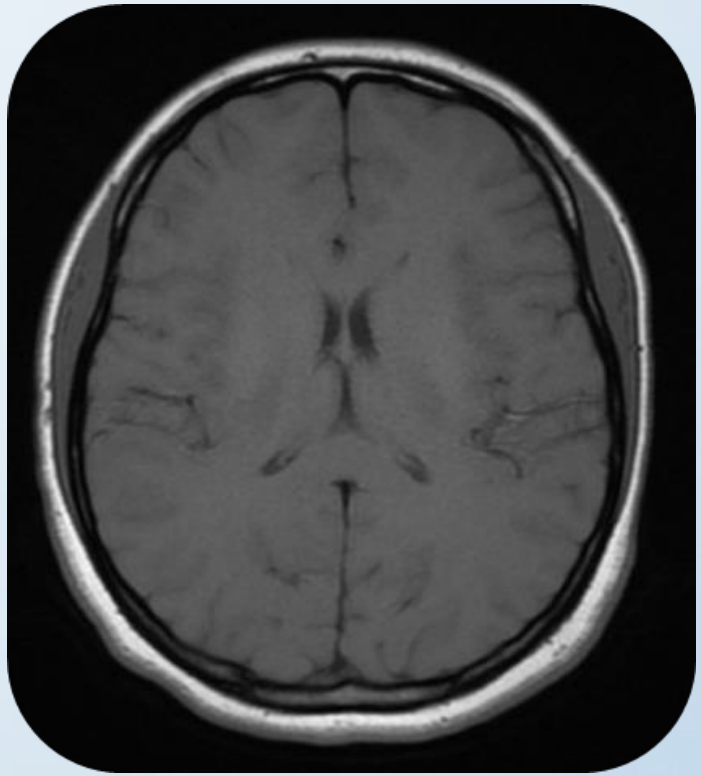
Brain MRI

Maedeh Asna Ashari, MD
Assistant Professor of Emergency Medicine
IUMS

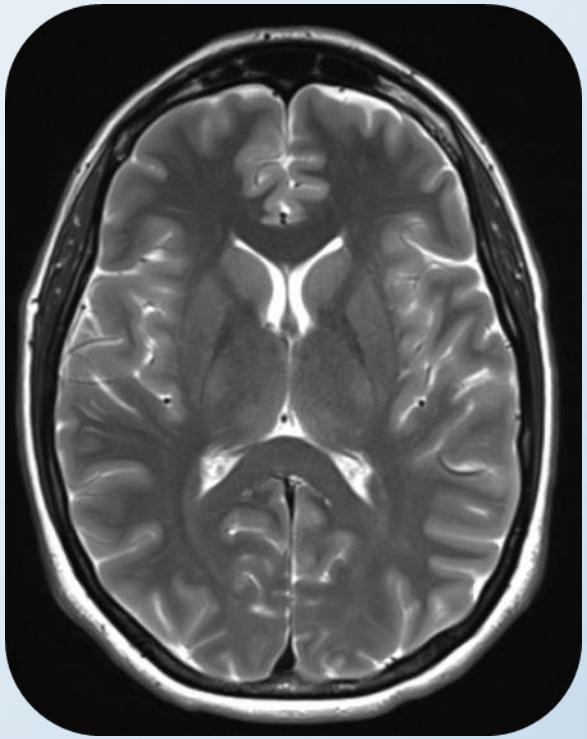
MRI sequences

Tissue	T1-Weighted	T2-Weighted	Flair
CSF	Dark	Bright	Dark
White Matter	Light	Dark Gray	Dark Gray
Cortex	Gray	Light Gray	Light Gray
Fat (within bone marrow)	Bright	Light	Light
Inflammation (infection, demyelination)	Dark	Bright	Bright

Tissue	T1-Weighted
CSF	Dark
White Matter	Light
Cortex	Gray
Fat (within bone marrow)	Bright
Inflammation (infection, demyelination)	Dark



Tissue	T2-Weighted
CSF	Bright
White Matter	Dark Gray
Cortex	Light Gray
Fat (within bone marrow)	Light
Inflammation (infection, demyelination)	Bright



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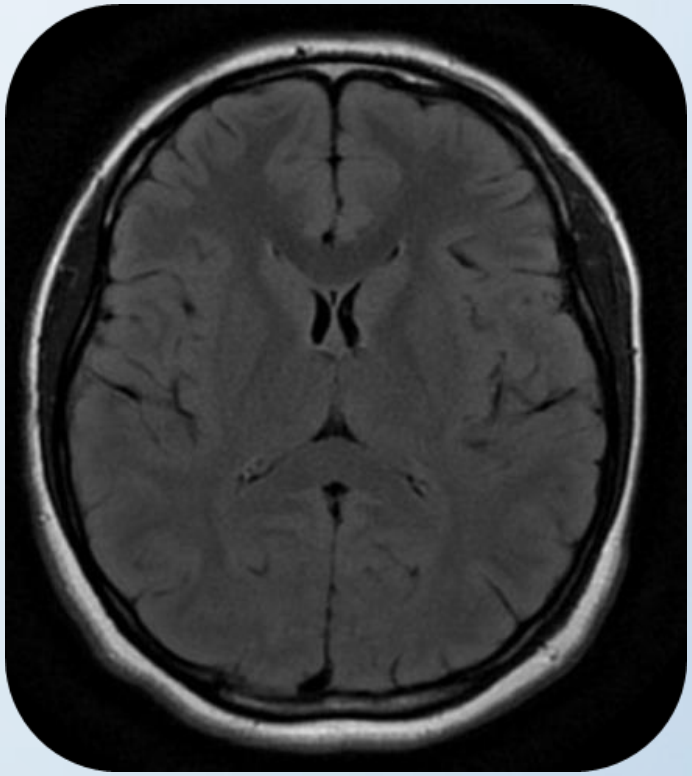
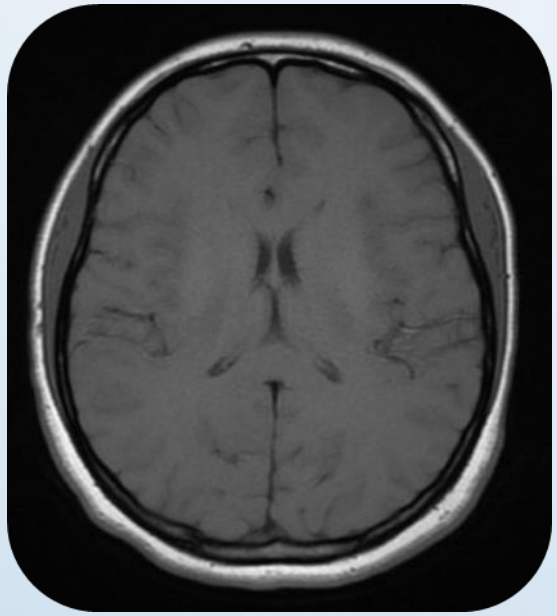
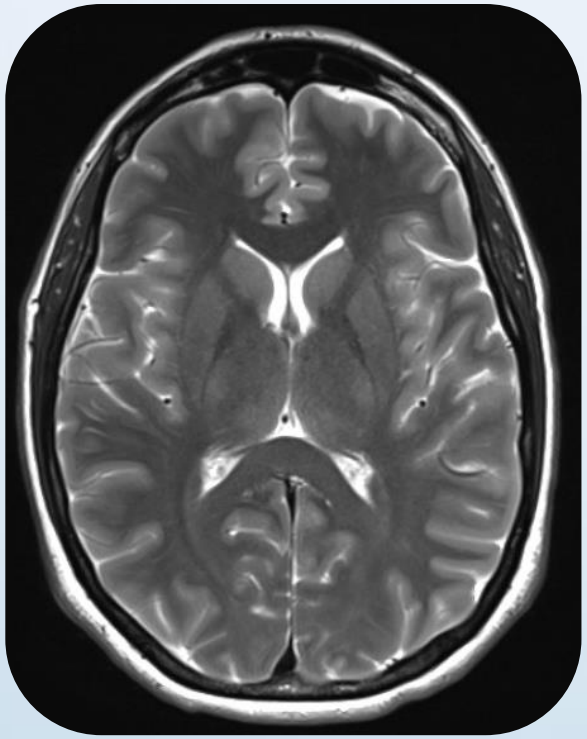




T1 Sequence

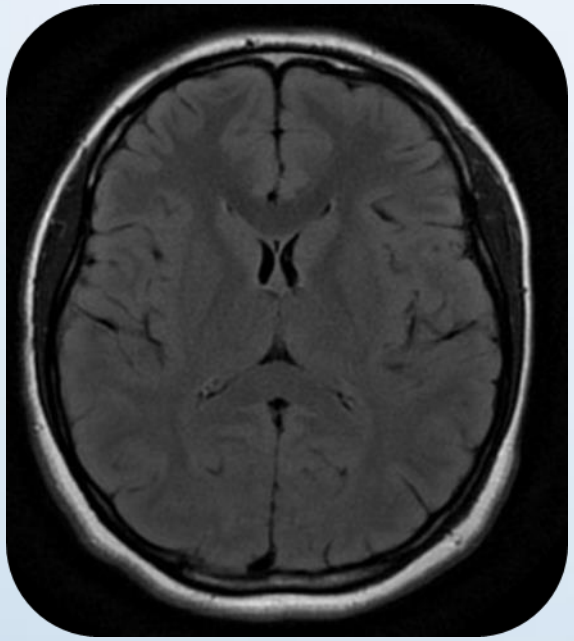
Provides the most anatomically-relevant images



T2 Sequence

Pathologic conditions that can be depicted with T2 sequences include:

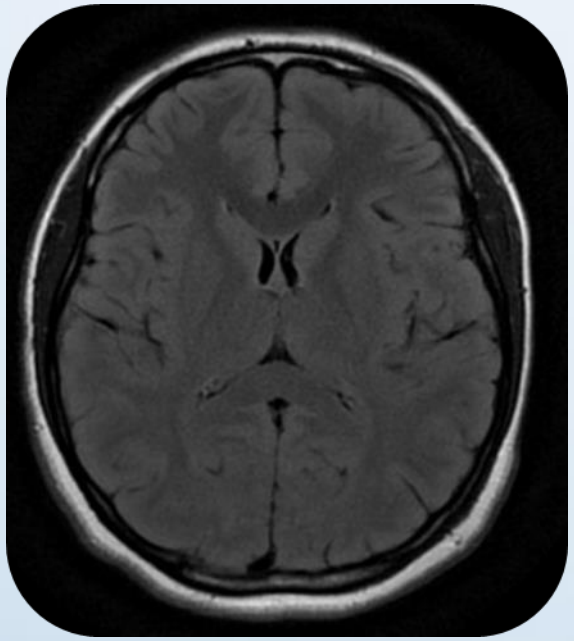
cerebral hemorrhage arteriovenous malformation, cavernoma, hemorrhage in tumor, punctate foci of hemorrhage in diffuse axonal injury



Flair sequence

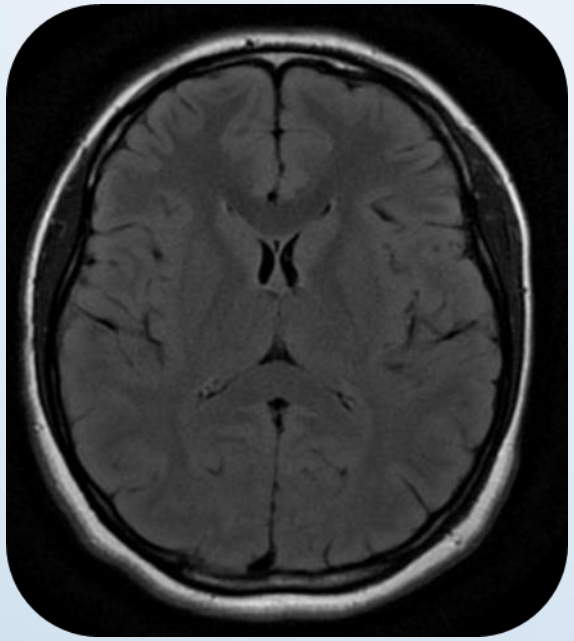
Commonly used sequence similar to T2, but the fluid is darker or "suppressed“

useful for areas of **edema** or **inflammation**
used to identify **plaques in multiple sclerosis**



Flair sequence

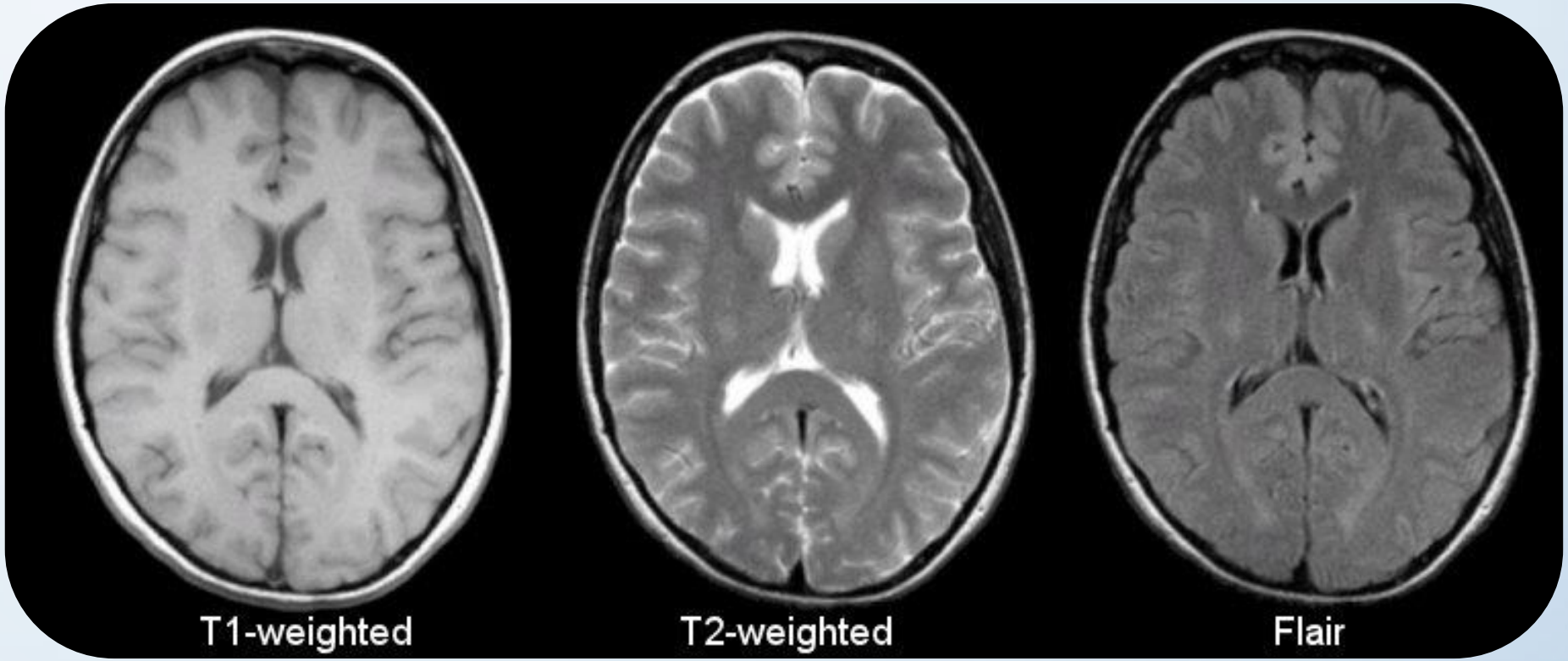
The FLAIR sequence is part of almost all protocols for imaging the brain, particularly useful in the detection of **subtle changes at the periphery of the hemispheres** and in the **periventricular region close to CSF**

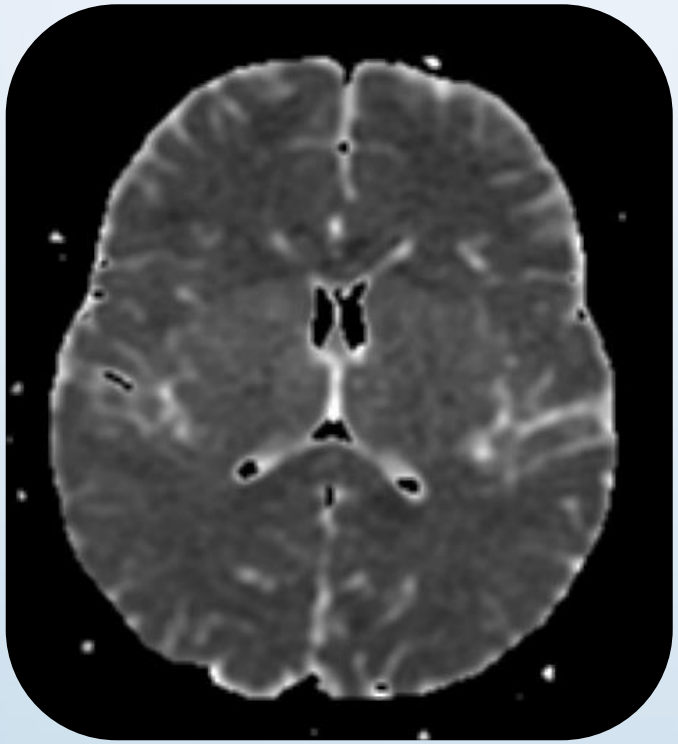


Flair sequence

The usefulness of FLAIR sequences has been evaluated in many diseases of the central nervous system such as :

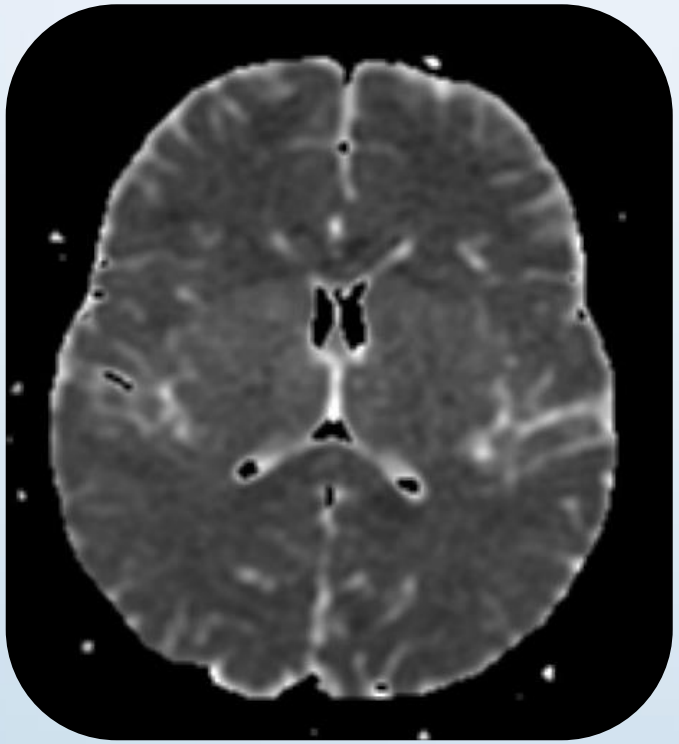
- infarction
- multiple sclerosis
- subarachnoid hemorrhage
- head injuries, and others


T1-weighted
T2-weighted
Flair



DWI

These "blocky" images show how easily water moves around
restricted diffusion occurs in **stroke**, **abscesses** and **cellular tumors**



DWI

It is a relatively low resolution image with the following appearance:

Grey matter: intermediate signal intensity (grey)

White matter: slightly hypointense compared to grey matter

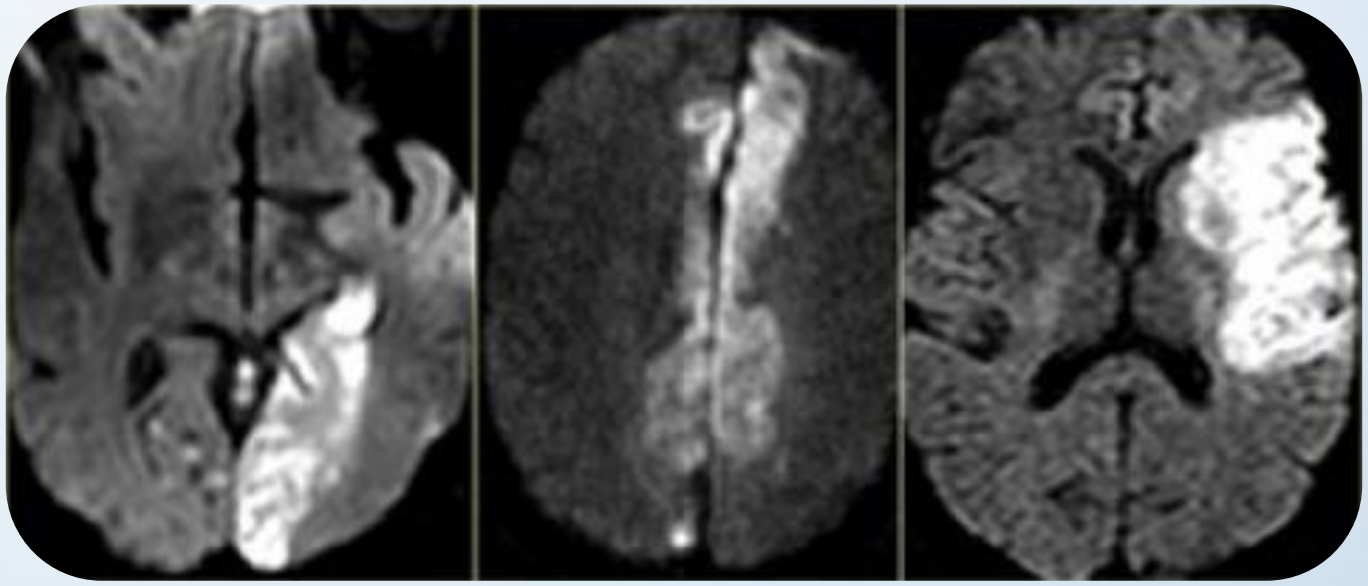
CSF: low signal (black)

Fat: little signal due to paucity of water

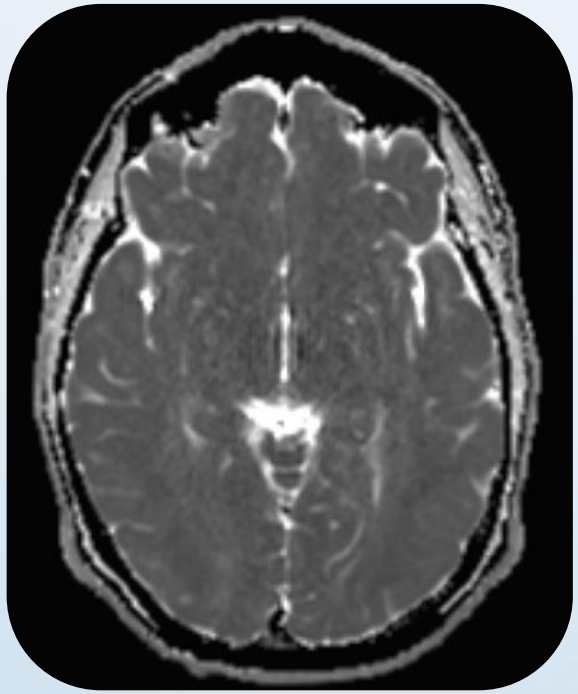
other soft tissues: intermediate signal intensity (grey)

Note

DWI is the most sensitive sequence for stroke imaging

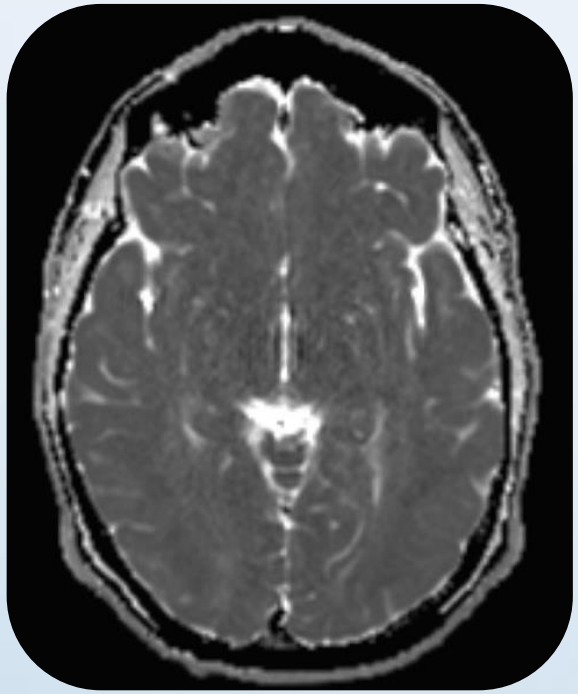


DWI in posterior, anterior and middle cerebral infarction



ADC
Apparent diffusion coefficient

ADC values are calculated automatically by the software and then displayed as a **parametric map** that reflects the degree of diffusion of water molecules through different tissues



ADC

Apparent diffusion coefficient

They are relatively low resolution images with the following appearances:

Grey matter: intermediate signal intensity (grey)

White matter: slightly hyperintense compared to grey matter

CSF: high signal (white)

Fat: little signal due to paucity of water

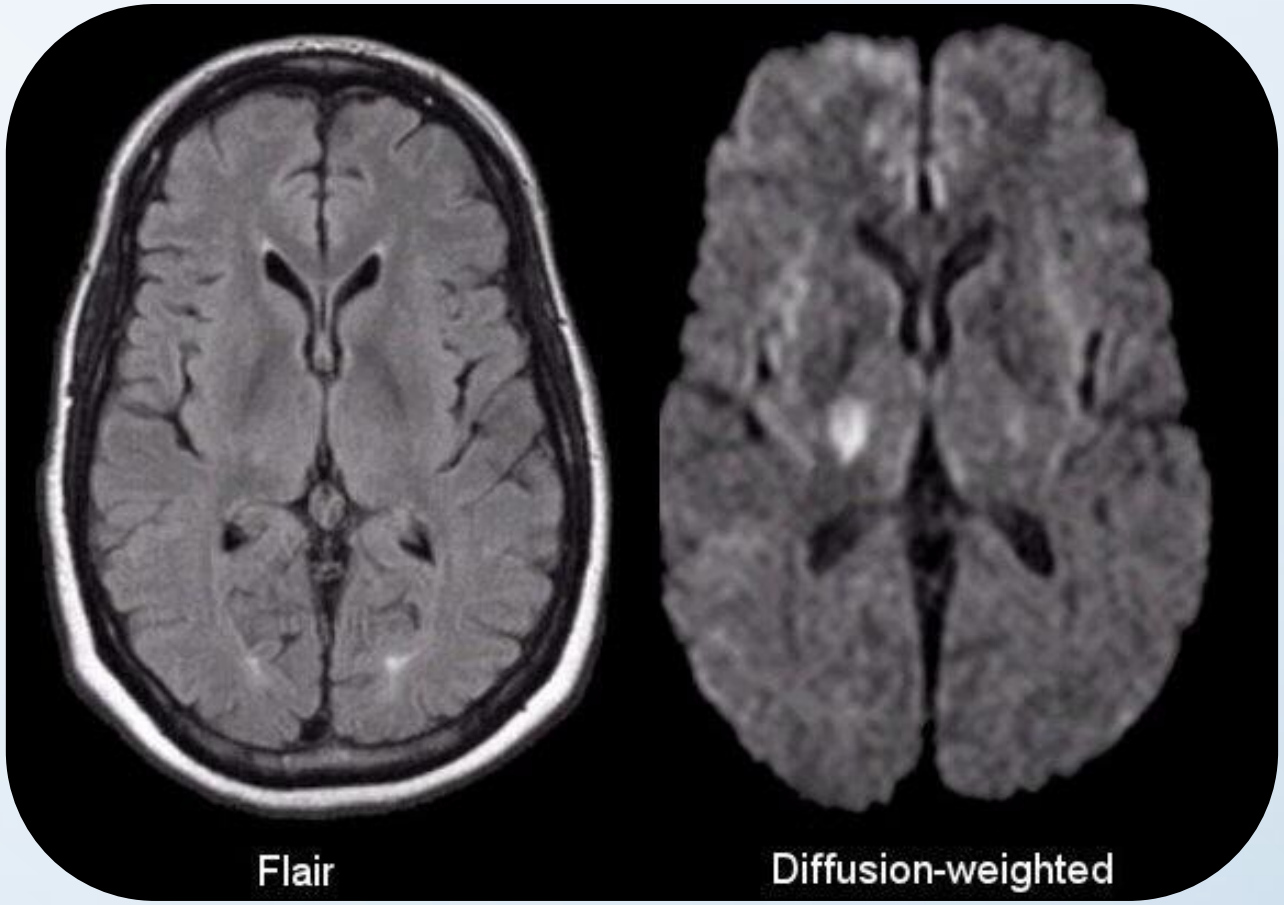
other soft tissues: intermediate signal intensity (grey)

Ischemic CVA

Hyperacute CVA

Within **minutes** of arterial occlusion, **diffusion-weighted imaging** demonstrates increased DWI signal and reduced ADC value

At this stage, the affected parenchyma appears normal on other sequences



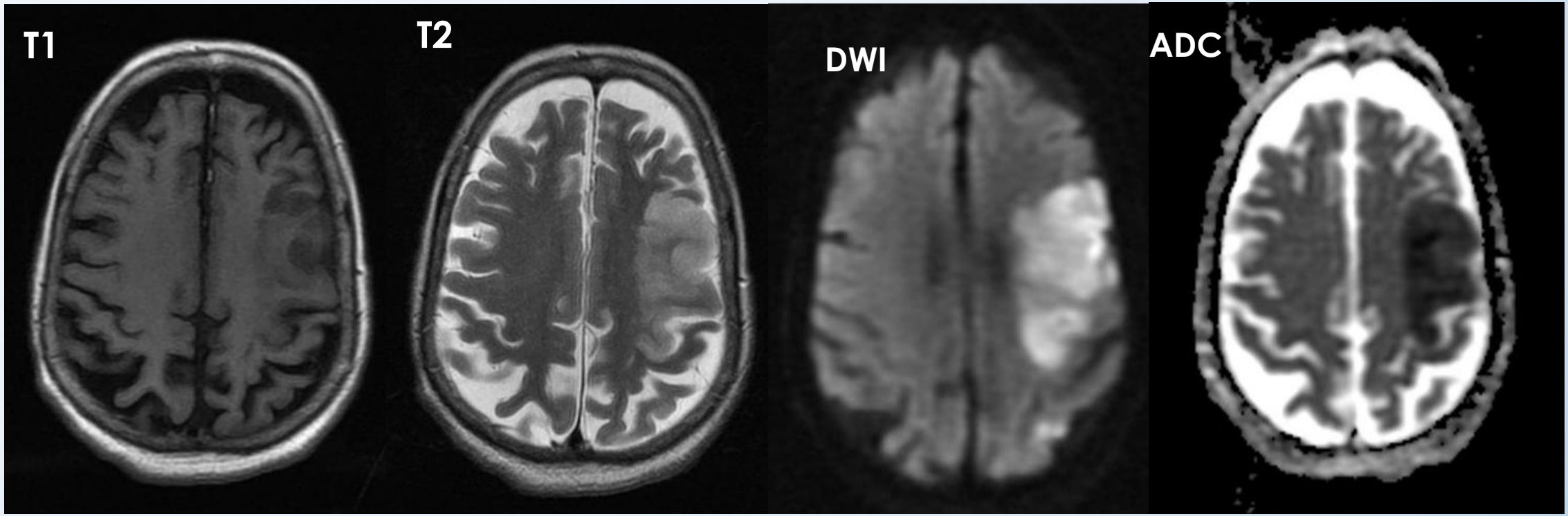
Note the Acute Infarction Only Seen on DWI

Acute CVA

Generally, **after 6 hours**, high T2 signal will be detected, initially more easily seen on FLAIR than conventional fast spin-echo T2

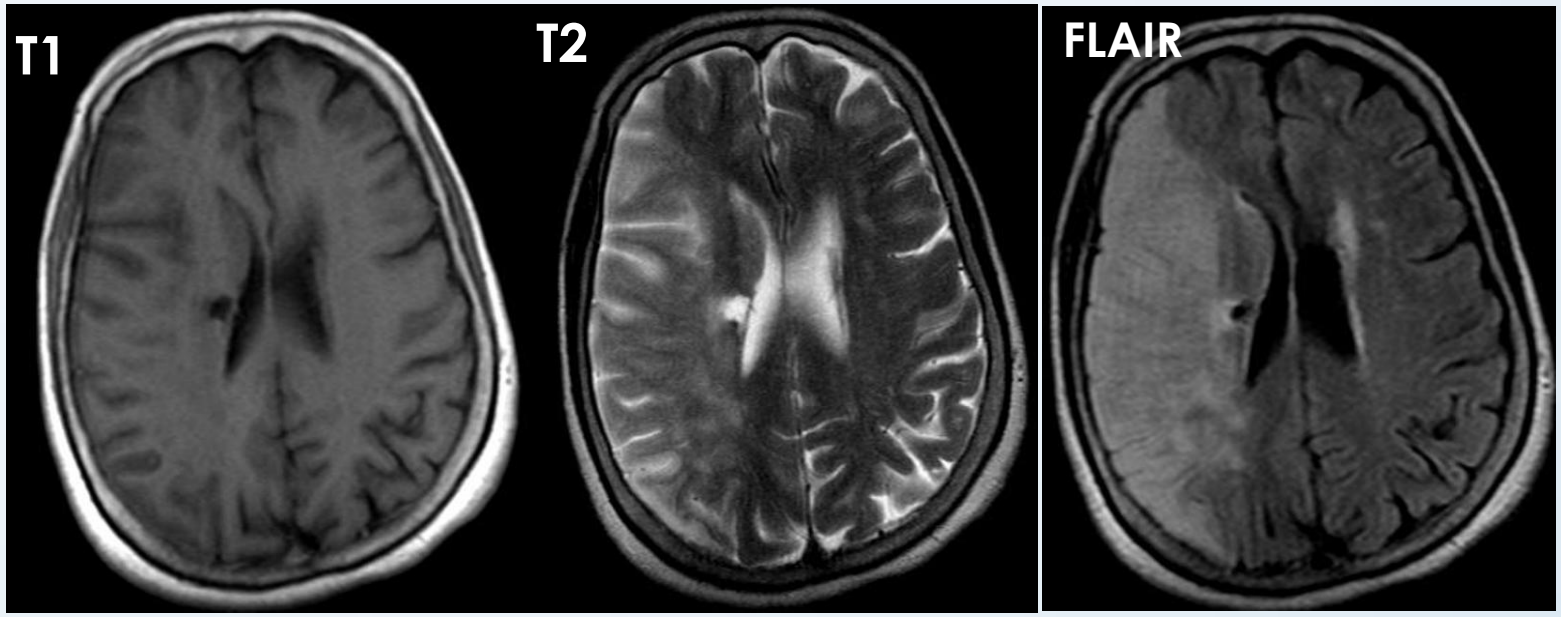
This change continues to increase over the next day or two

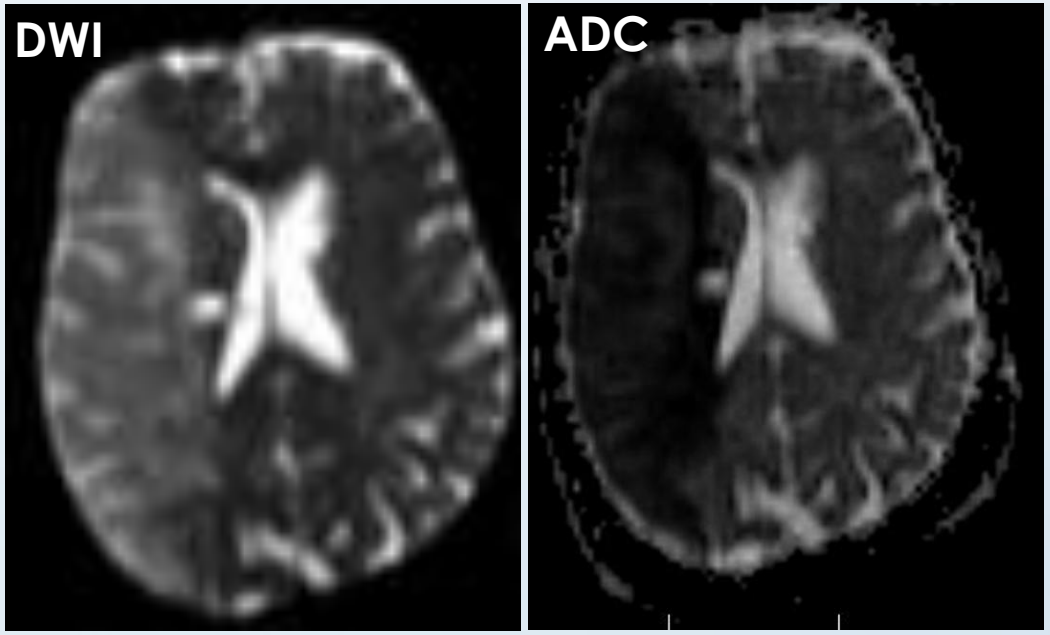
T1 Hypointensity is only seen after 16 hours and persists



Hypointensity Hyperintensity Hyperintensity Hypointensity

>6 hrs Ischemic Stroke





Acute Ischemic Stroke

Subacute CVA

During the **first week**, the infarcted parenchyma continues to demonstrate high DWI signal and low ADC signal, although by the end of the first week ADC values have started to increase

The infarct remains hyperintense on T2 and FLAIR, with T2 signal progressively increasing during the first 4 days

T1 signal remains low

Chronic CVA

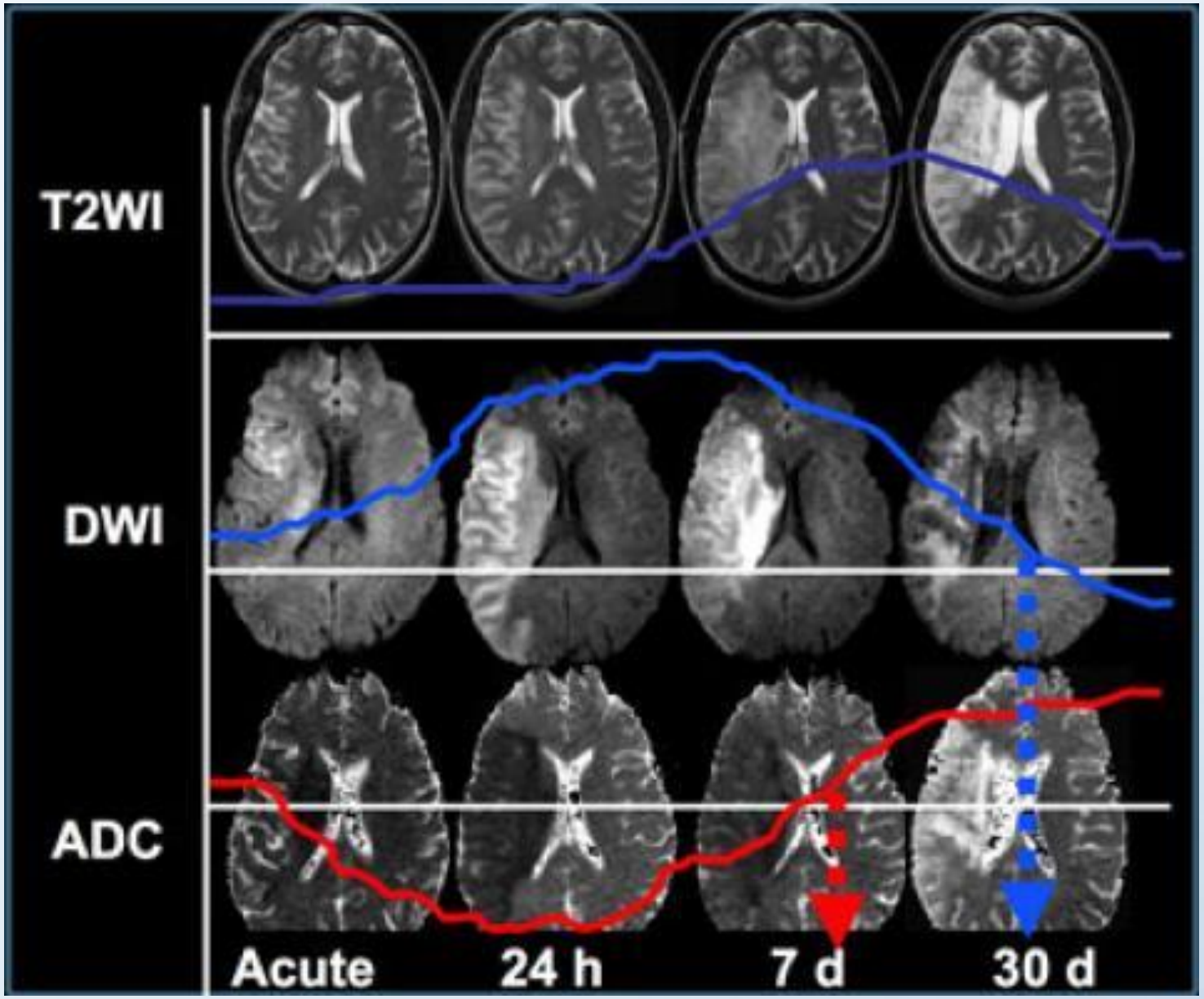
ADC demonstrates pseudonormalization typically occurring between 10-15 days

As ADC values continue to rise, infarcted tissue progressively gets brighter than normal parenchyma

In contrast, **DWI** remains elevated due to persistent high T2/FLAIR signal

T2 fogging is also encountered typically between 1 and 5 weeks, most commonly around week 2

	Hyper Acute 0-6 hr	Acute 6-24hr	Subacute 24hr-1W	chronic
T 1	Isointensity	Hypointensity After 16 hr	Hypointensity	Hypointensity
T 2	Isointensity	Hyperintensity	Hyperintensity	Hyperintensity
FLAIR	Isointensity	Hyperintensity	Hyperintensity	Hyperintensity
DWI	Hyperintensity	Hyperintensity	Hyperintensity	Hyperintensity
ADC	Hypointensity	Hypointensity	Hypointensity	Hypointensity


T2WI
DWI
ADC
Acute
24 h
7 d
30 d

In the acute phase **T2WI** will be **normal**, but in time the infarcted area will become hyperintense

The hyperintensity on **T2WI** reaches its maximum between **7 and 30** days. After this it starts to fade

DWI is already positive in the acute phase and then becomes more bright with a maximum at **7 days**

DWI in brain infarction will be positive for approximately for 3 weeks after onset

ADC will be of low signal intensity with a maximum at 24 hours and then will increase in signal intensity and finally becomes bright in the chronic stage

Encephilitis

Herpes Simplex Type 1 (HSV-1) Encephalitis

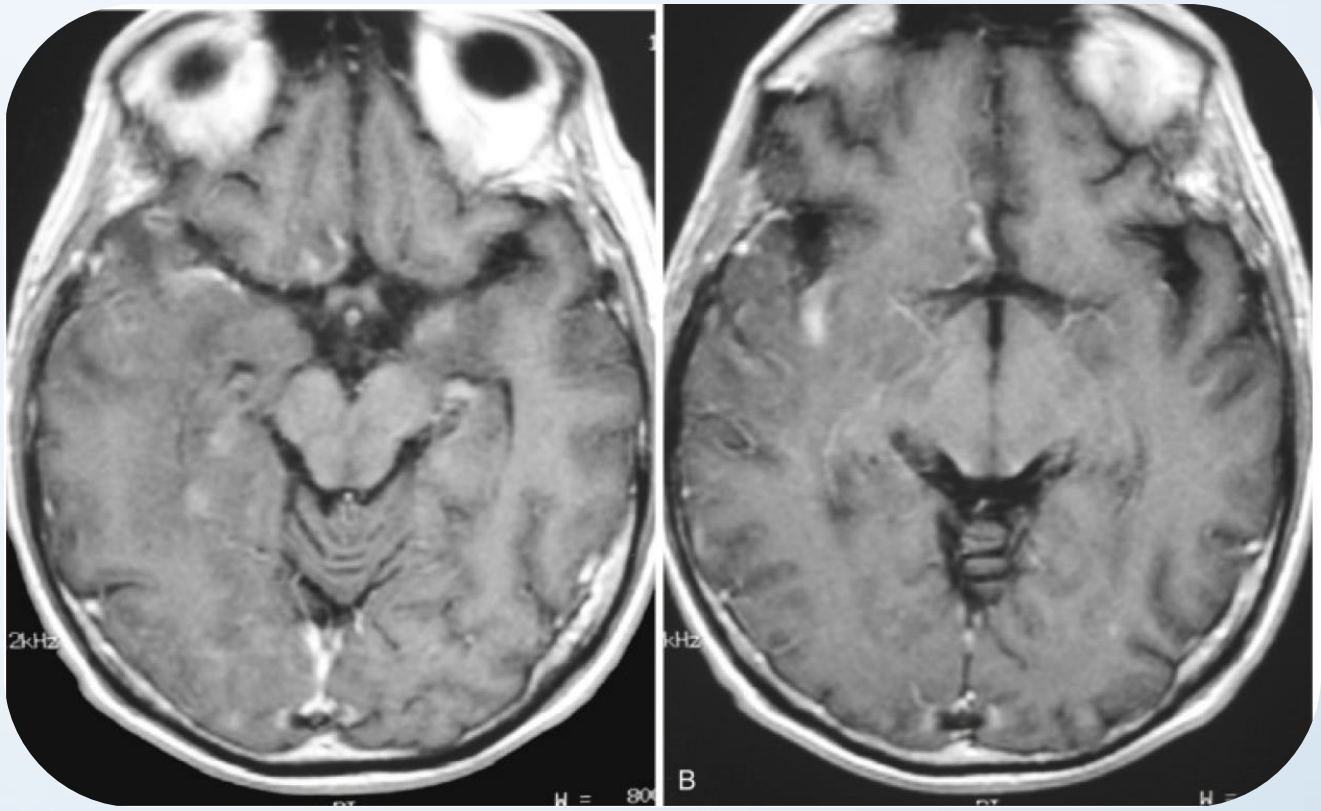
Bilateral asymmetric T2-FLAIR **hyperintensities** in the cortex and subcortical **white matter**, in addition to sulcal effacement involving the hippocampi and mesial temporal lobes

The basal ganglia are spared, helping in di erentiation from other encephalitides that frequently involve the basal ganglia

Patchy diffusion restriction maybe seen early and may precede T2-FLAIR hyperintensities

Petechial hemorrhages may be demonstrated in areas of involvement

Later in the course of disease, patchy enhancement may also be seen

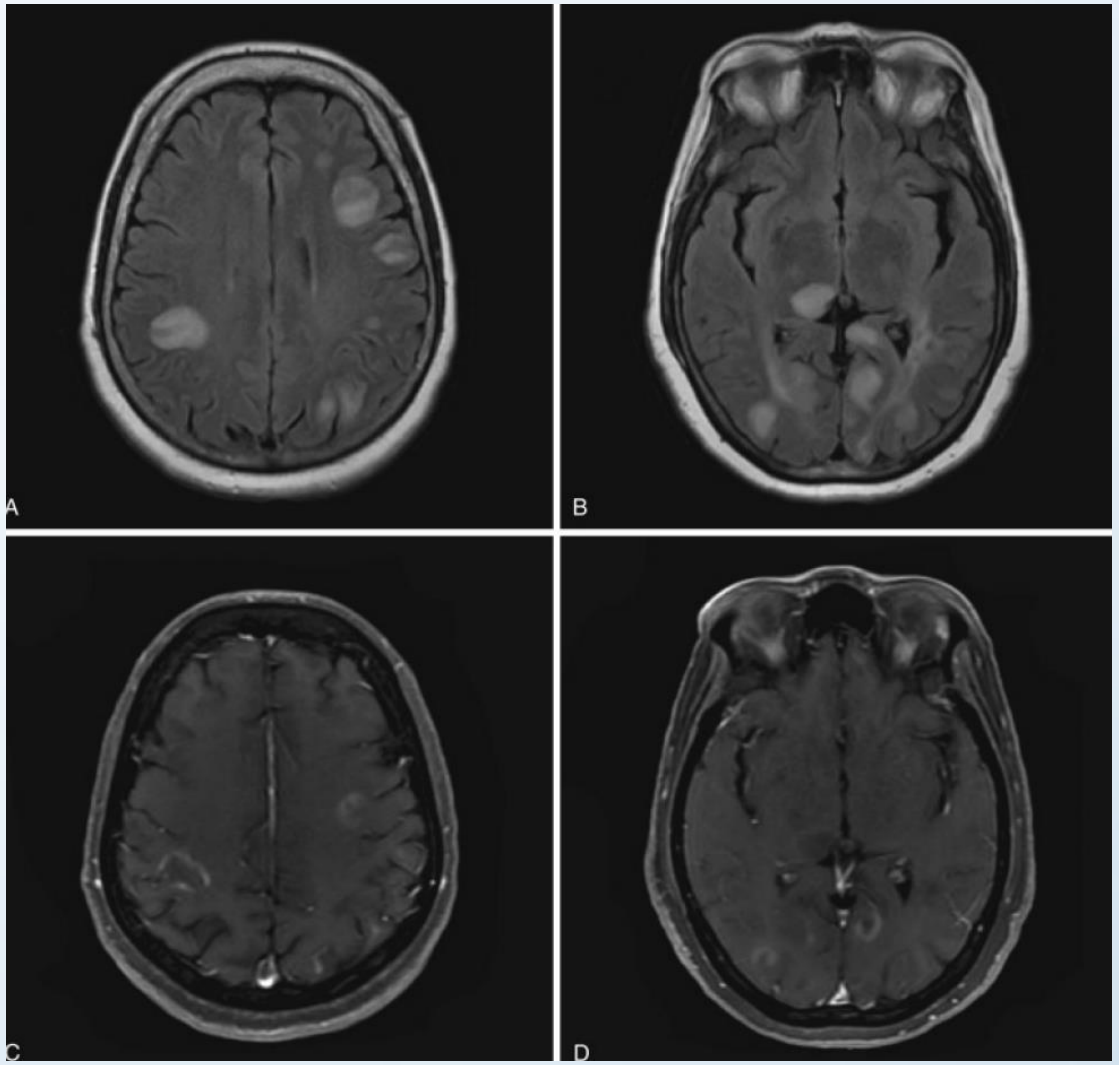


Herpes simplex virus (HSV)-1 encephalitis.
Axial postcontrast T1-weighted image demonstrates **linear streaky enhancement along the sylvian fissure**, insular cortex, and bilateral temporal frontal gyri

Acute disseminated encephalomyelitis (ADEM)

ADEM is an autoimmune demyelinating disease that usually occurs 2 to 31 days following a viral infection or vaccination

Typically multiple bilateral, asymmetric, poorly defined, T2-FLAIR hyperintense, non-enhancing lesions are seen in the subcortical white matter



A and B, T2-FLAIR images show numerous asymmetric rounded hyperintense, predominantly subcortical, white matter lesions. Some lesions involve the cortex. A right pulvinar lesion is also seen
C and D, Postcontrast T1-weighted image demonstrates incomplete ring enhancement associated with these lesions

Thank you For your Attention