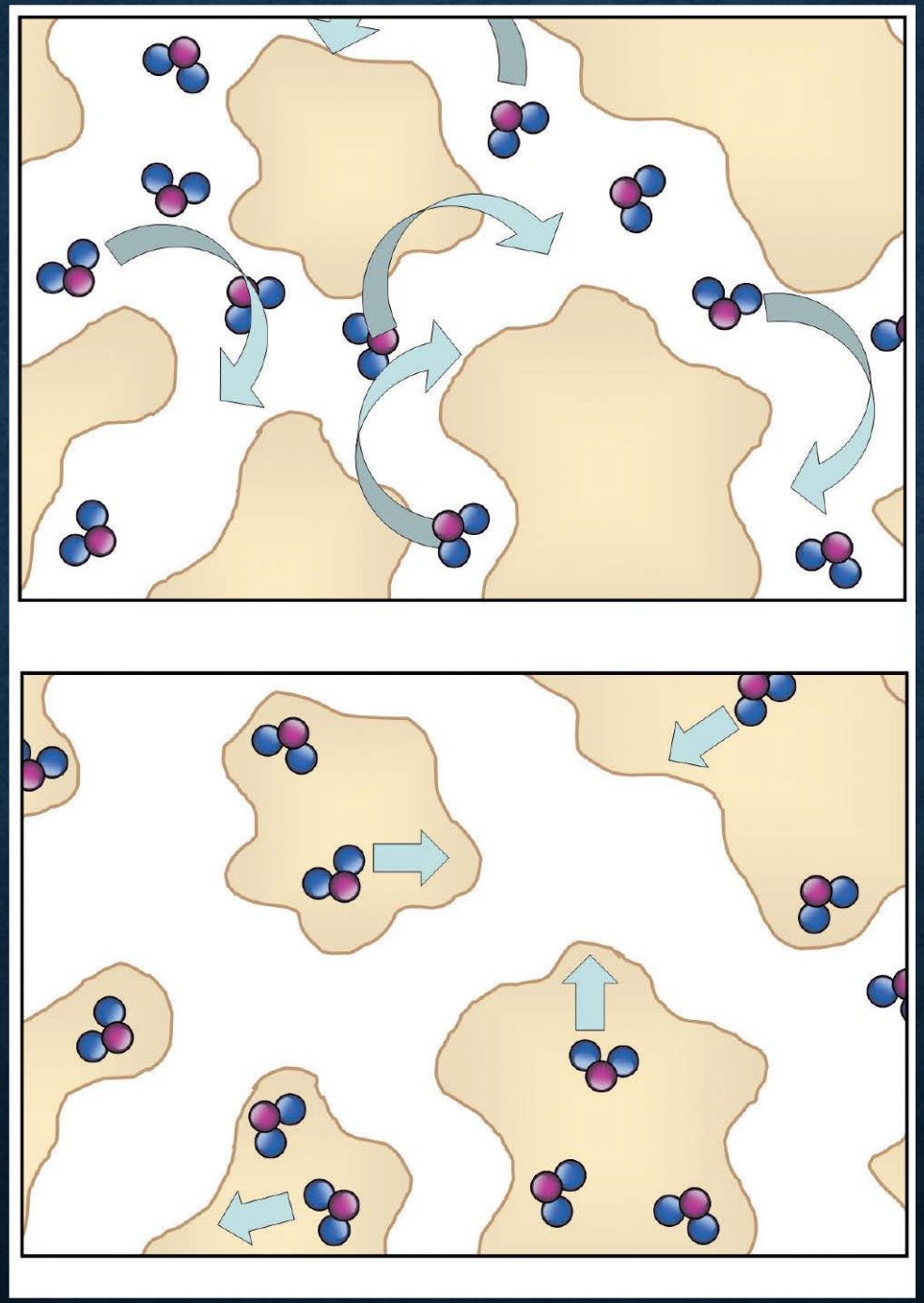
BRAIN MRI

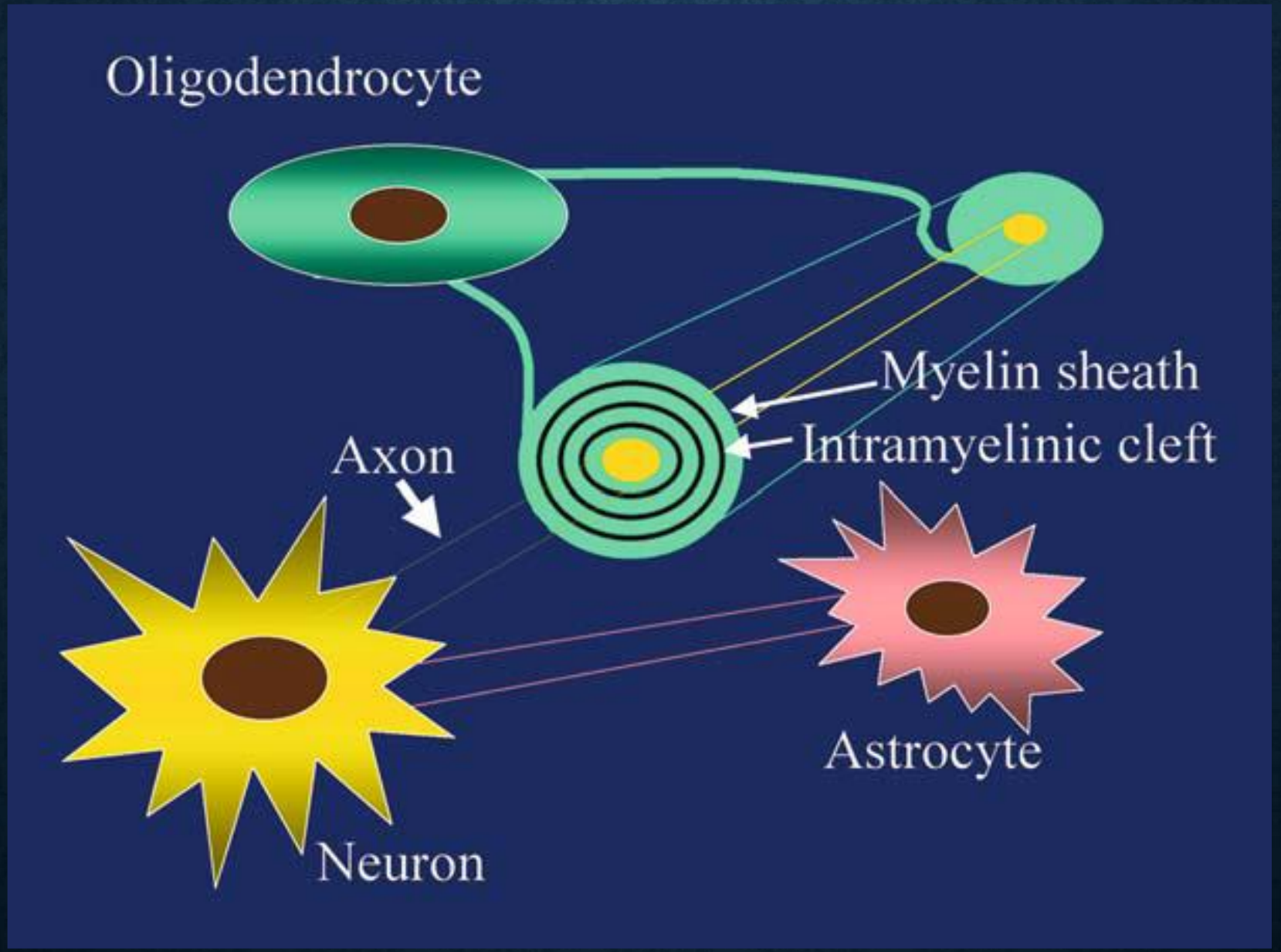
دکتر حسام قدیریان

متخصص جراحی مغز و اعصاب

استادیار دانشگاه علوم پزشکی بابل

DIFFUSION WEIGHTED IMAGING (DWI)




Oligodendrocyte
Myelin sheath
Intramyelinic cleft
Axon
Astrocyte
Neuron

DIFFUSION WEIGHTED IMAGING (DWI)

- Normally water protons have the ability to diffuse extracellularly and loose signal.
  High intensity on DWI indicates restriction of the ability of water protons to diffuse extracellularly.
  Restricted diffusion is seen in
  - Acute Infarction
  - Abscess

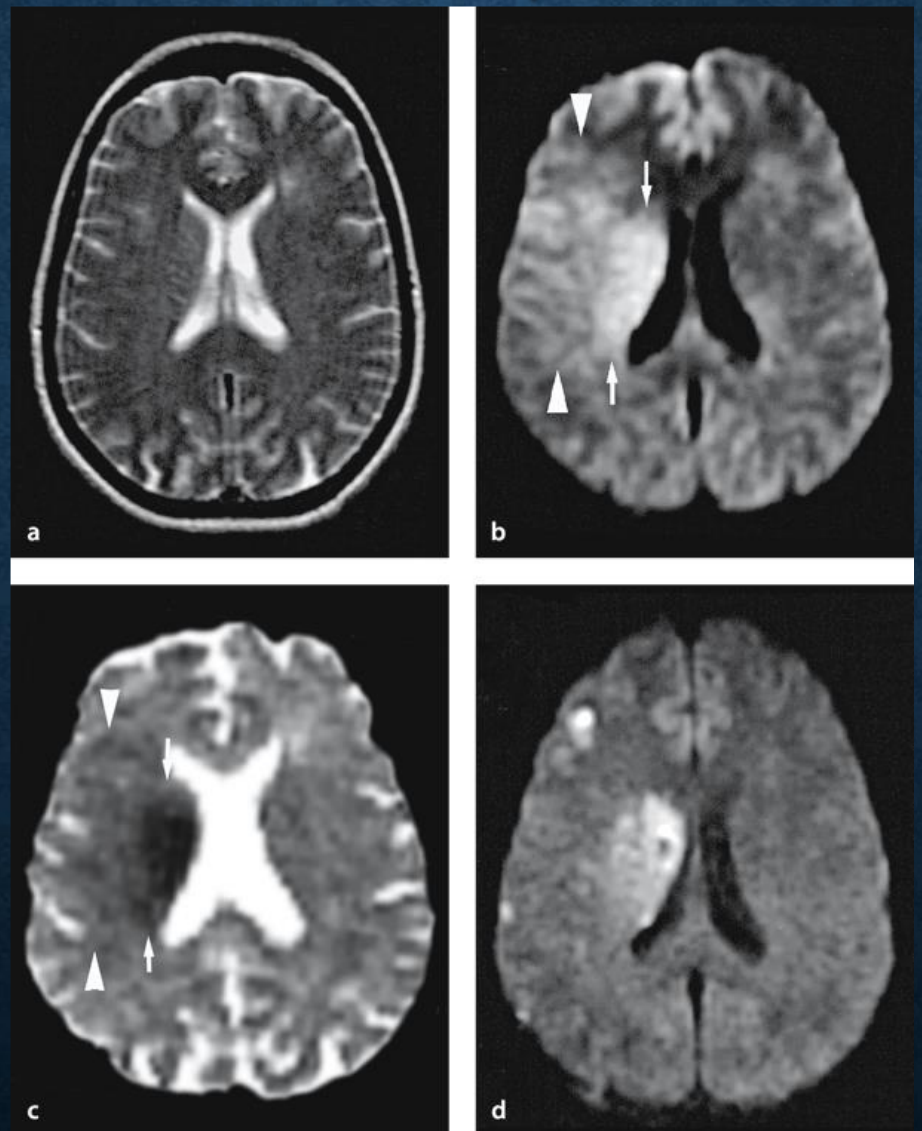
INFARCTION

In recent years, diffusion-weighted (DW) imaging has been proven as the most sensitive MR imaging technique to diagnose hyperacute cerebral infarction. The detection of acute ischemic lesions is based on alterations in motion of water molecules. It is a very sensitive technique,which is not significantly affected by patient motion. DW imaging of the brain can usually be accomplished in less than 2 minutes. The ischemic event results in restricted diffusion of the affected tissue,which can be seen as early as 30 minutes after ictus.

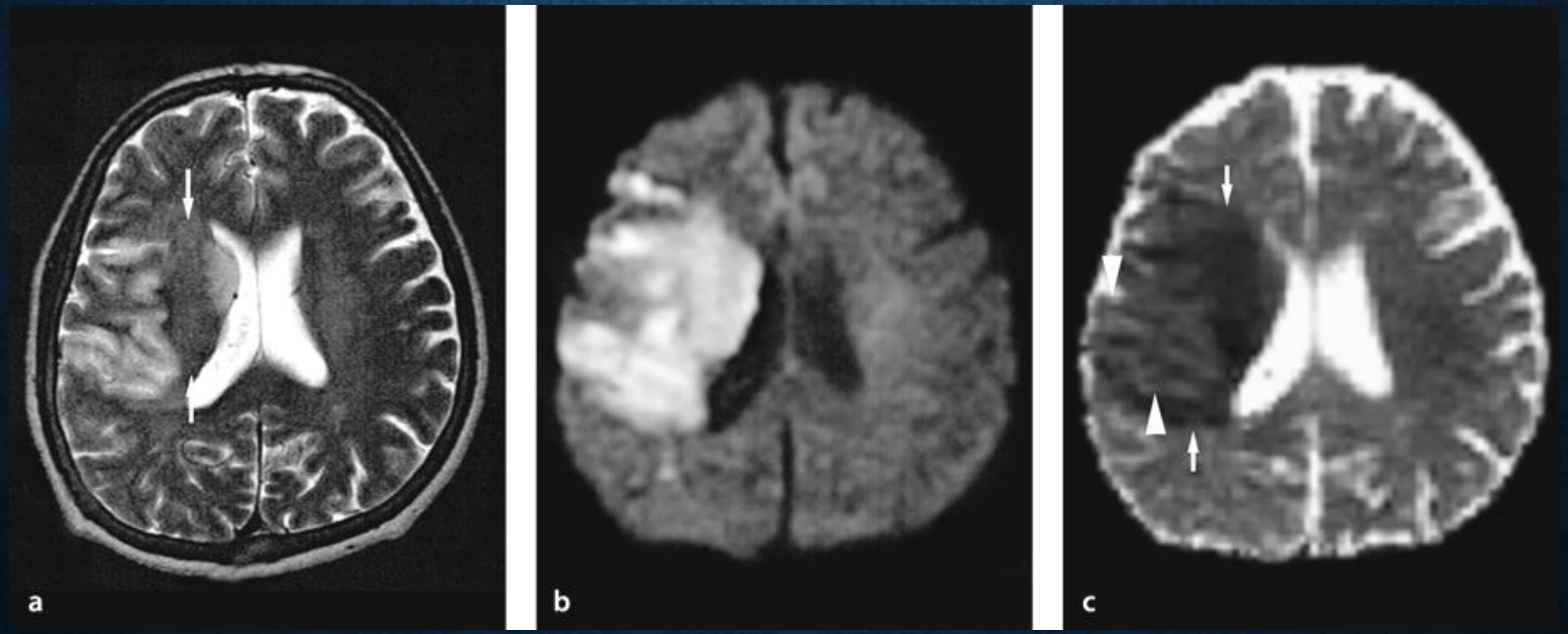
INFARCTION

	< 6 hours	3 days	7 days	30 days
T2	Isointense	Bright	Bright	Bright
DW imaging	Bright	Very bright	Bright	Isointense
ADC	Dark	Very dark	Dark	Bright

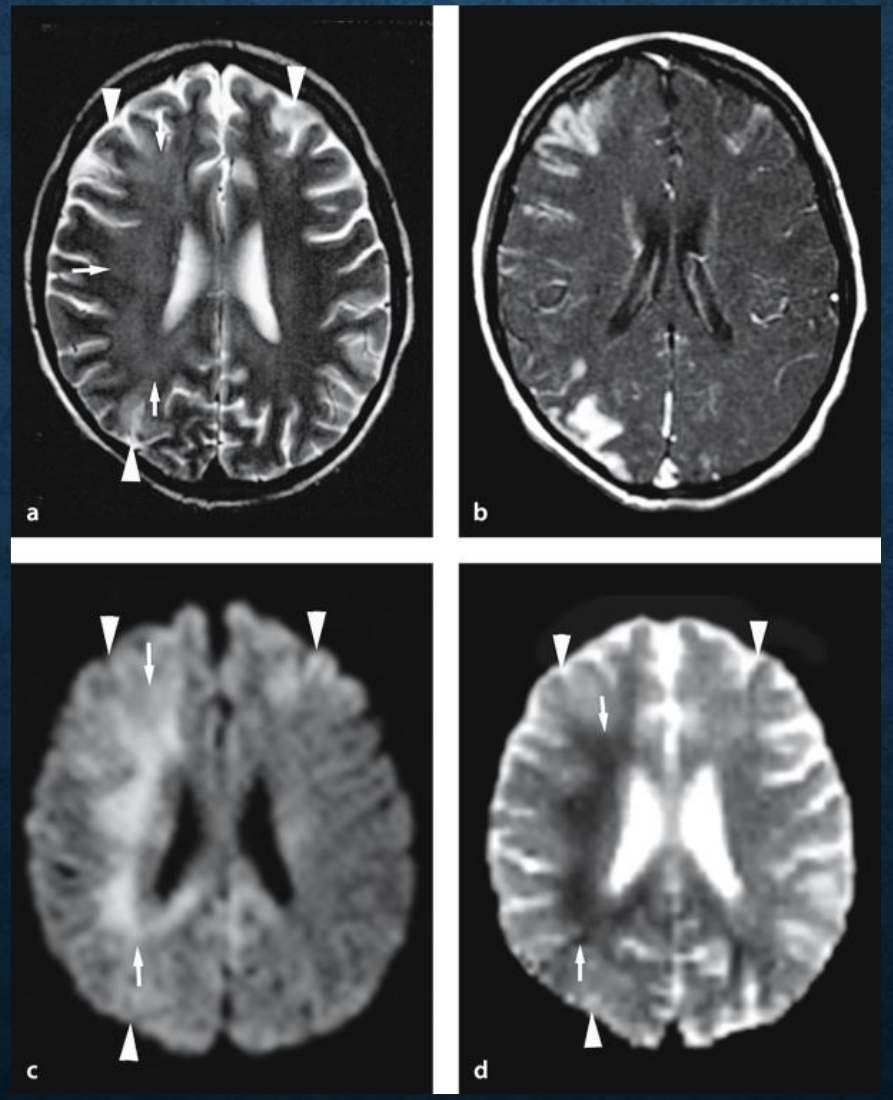
HYPERACUTE INFARCTION (2 HOURS AFTER ONSET)



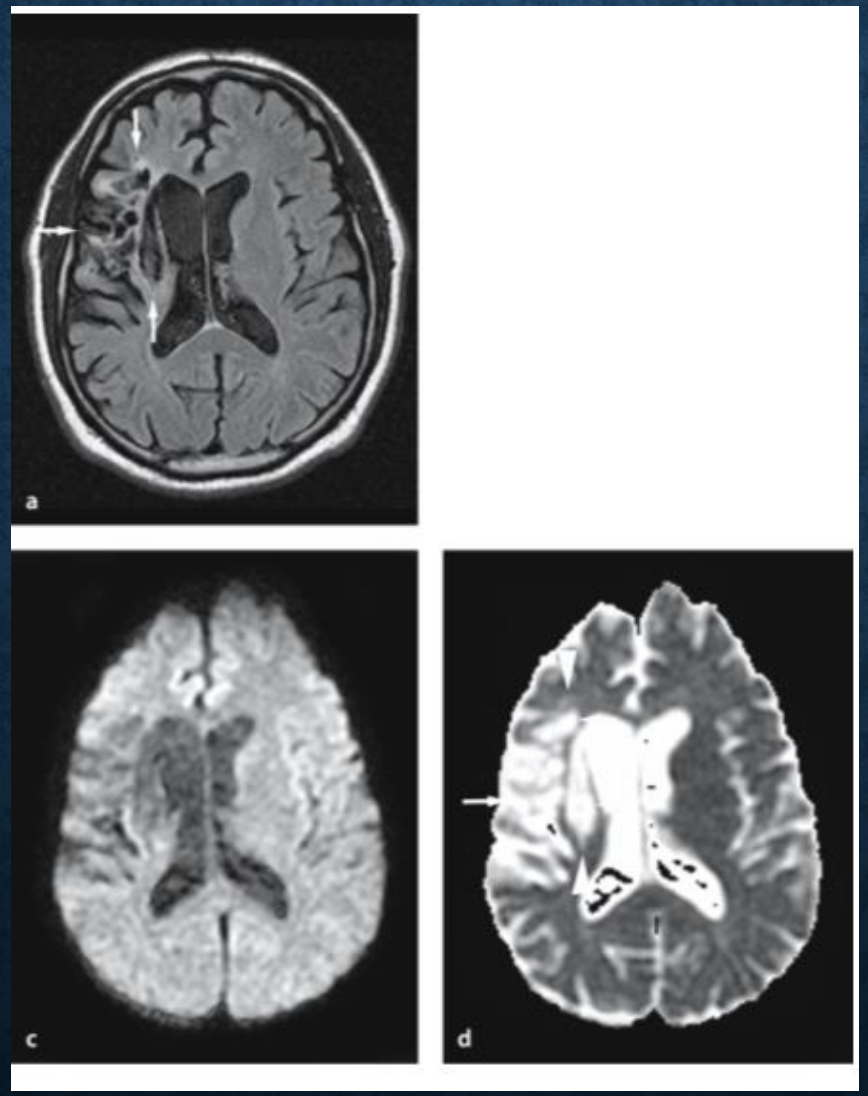
ACUTE INFARCTION (24 HOURS AFTER ONSET)

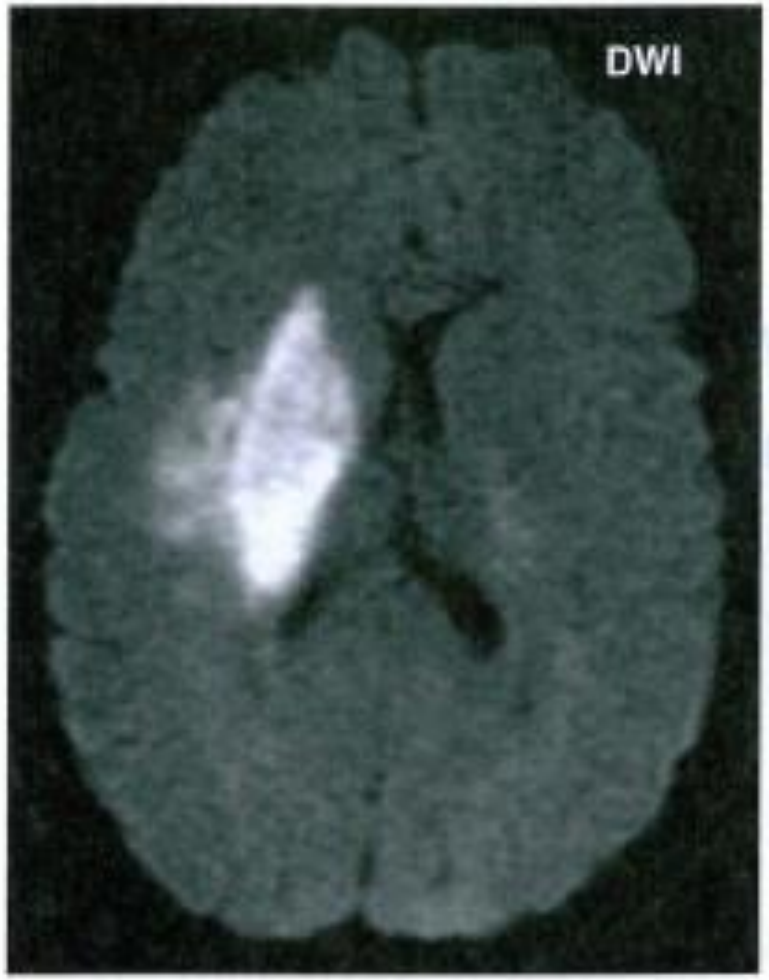


SUBACUTE INFARCTION (10 DAYS AFTER ONSET)

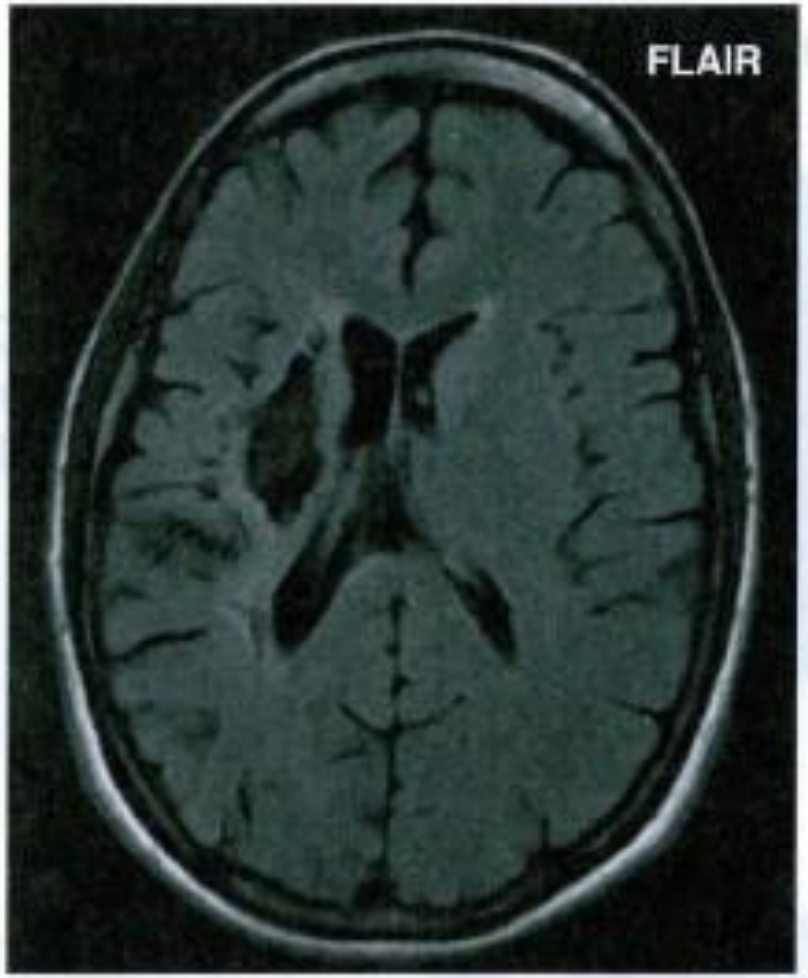


CHRONIC INFARCTION (10 MONTHS AFTER ONSET)

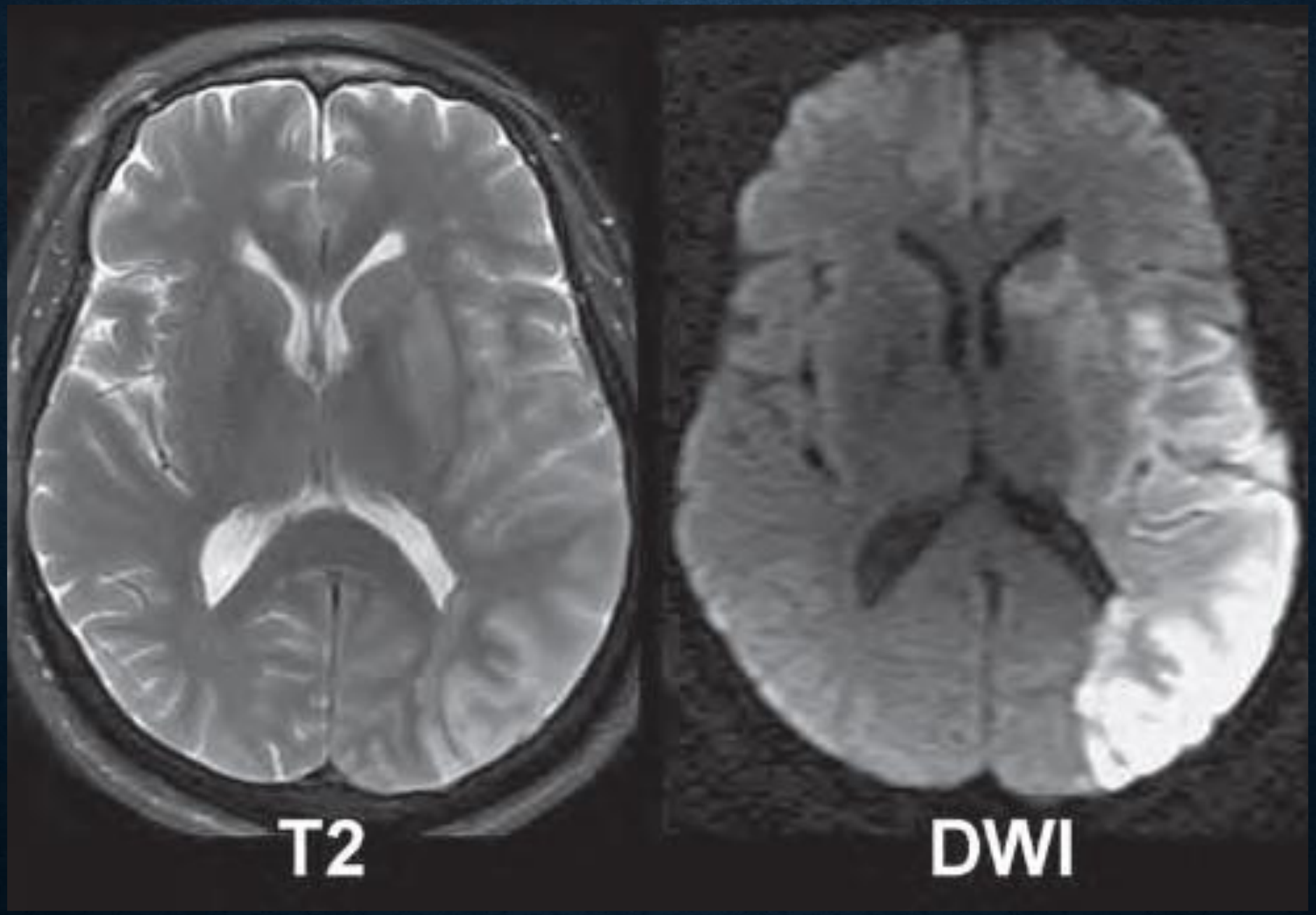


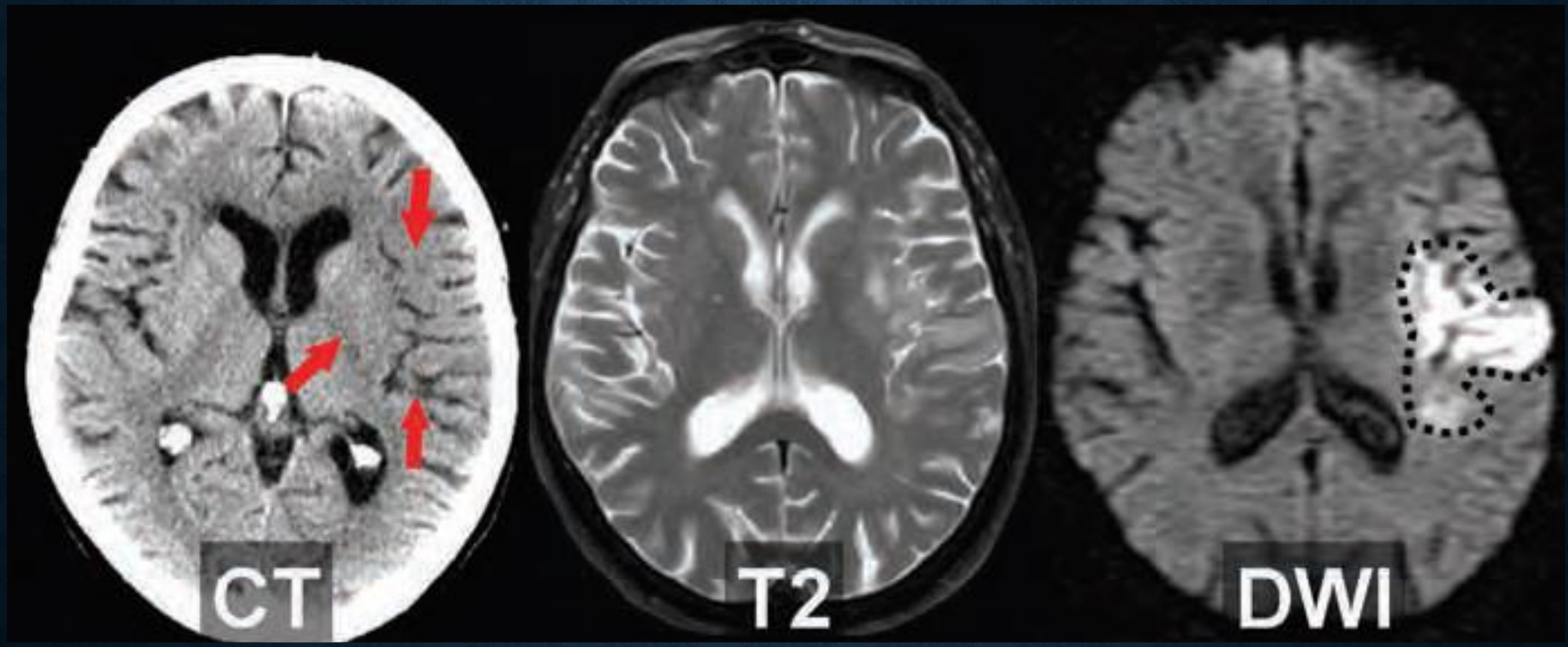


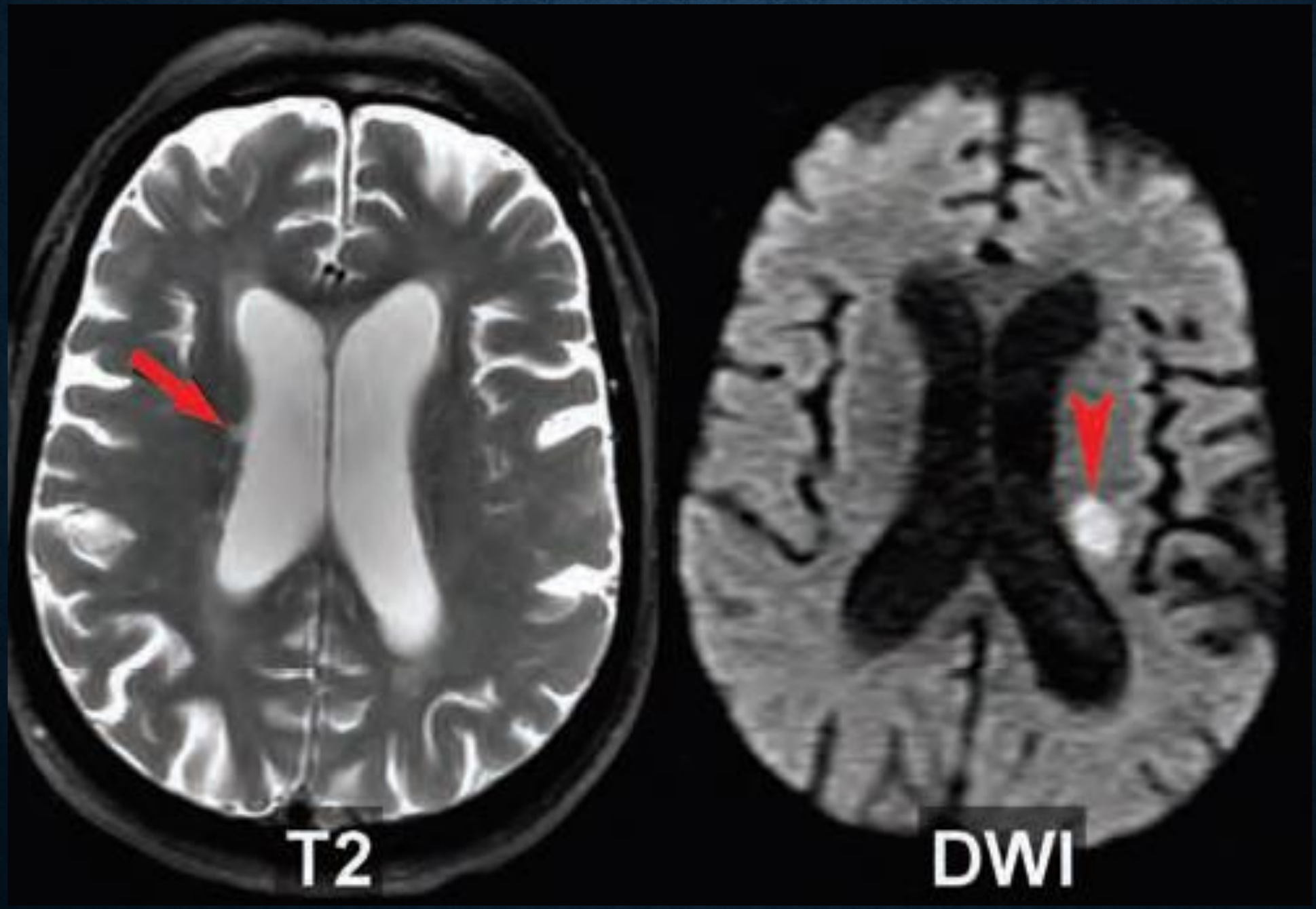
Diffusion weighted imaging (DWI): hyperintensity in the deep right MCA territory signifying acute infarct

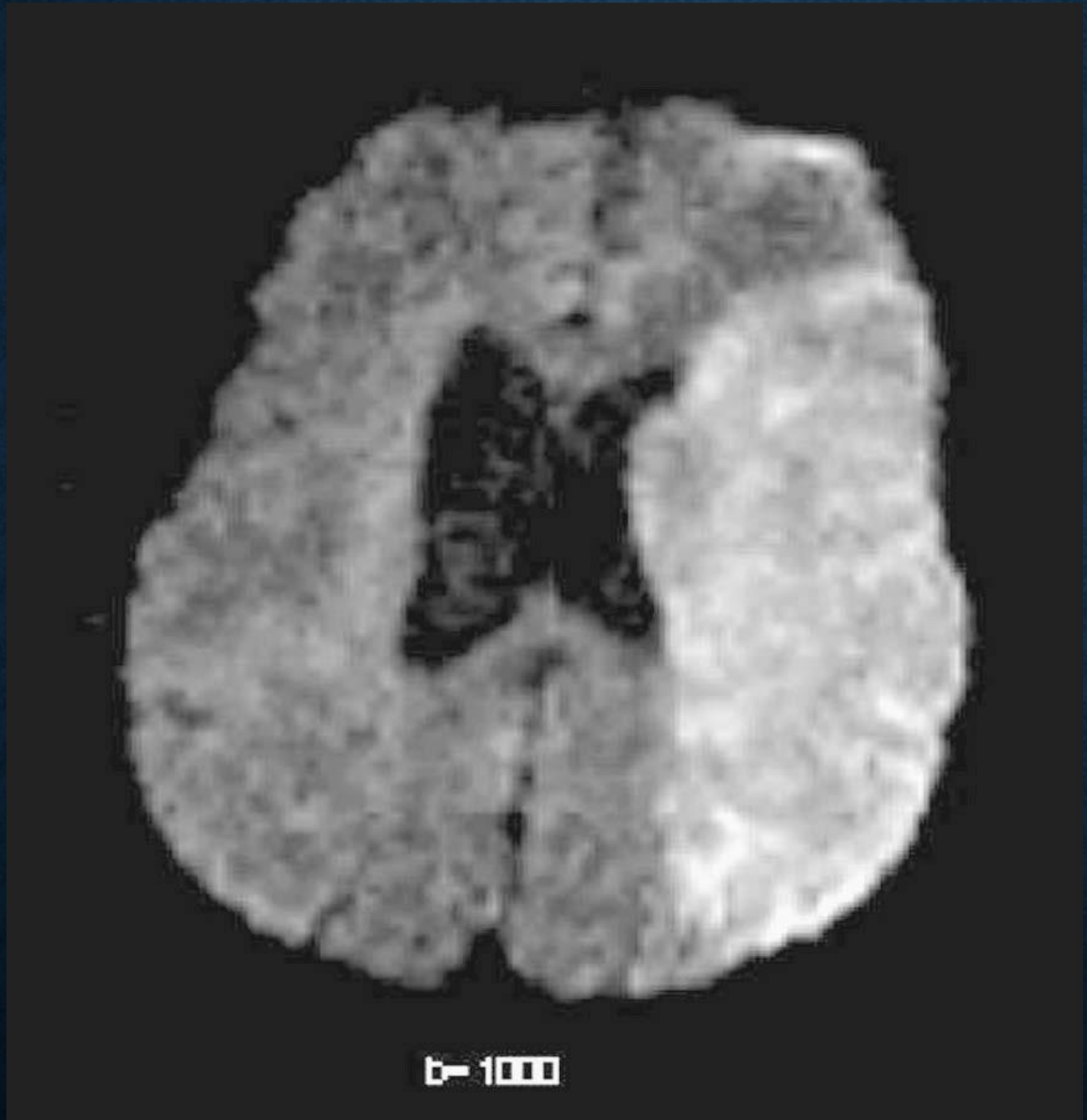


Fluid attenuated inversion recovery (FLAIR): one year follow up showing chronic infarct in the deep structures with relative preservation of the cortex


T2
DWI


CT
T2
DWI


T2
DWI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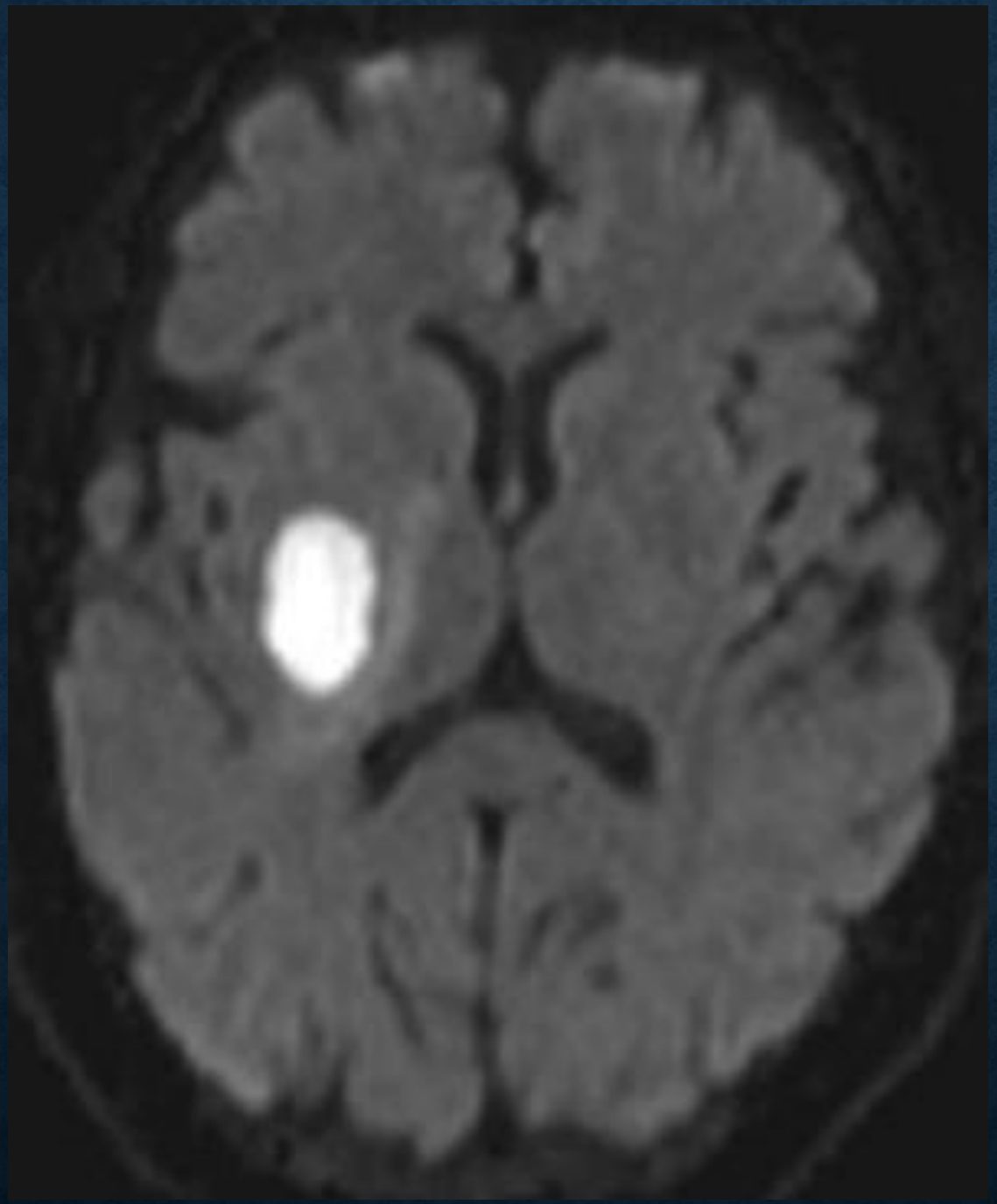
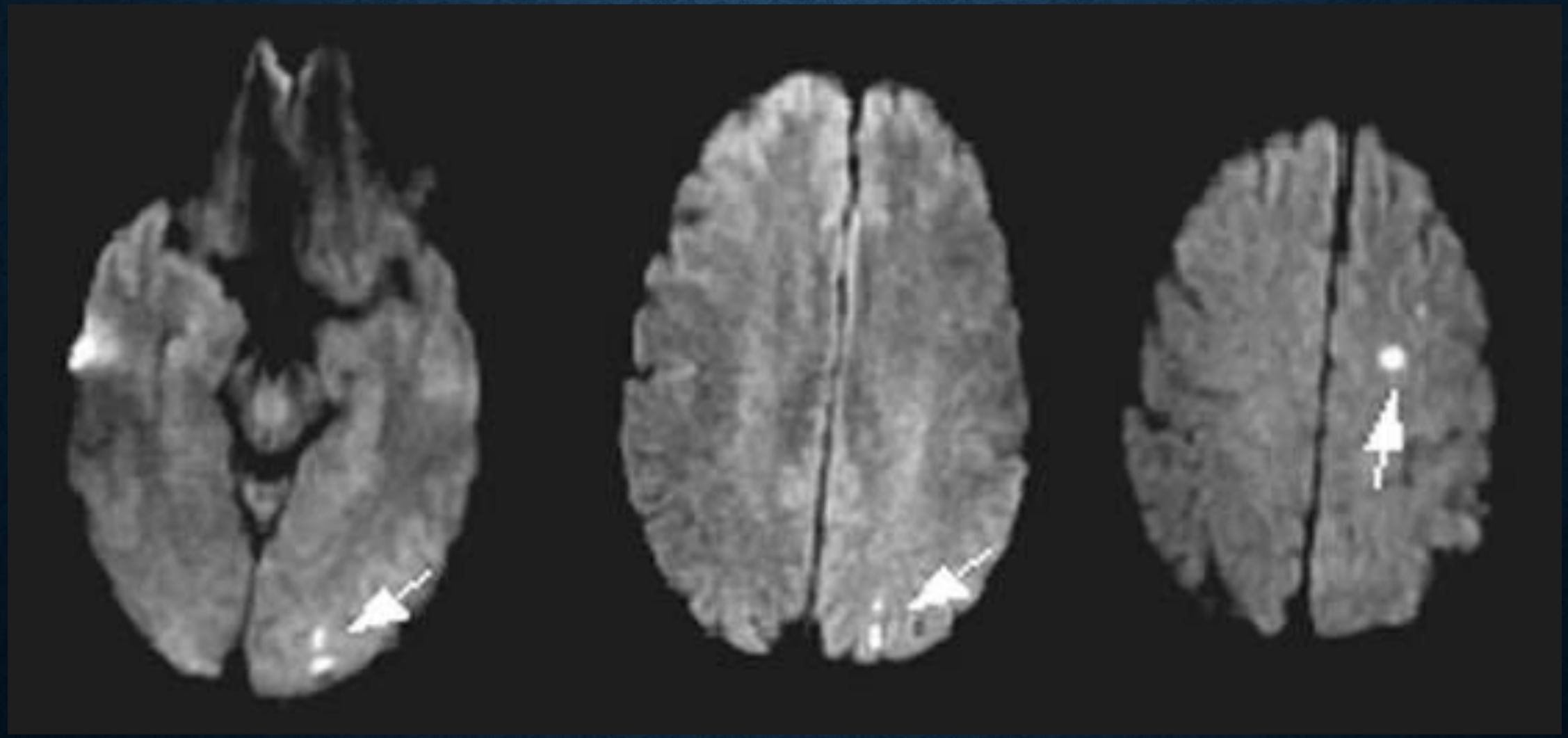
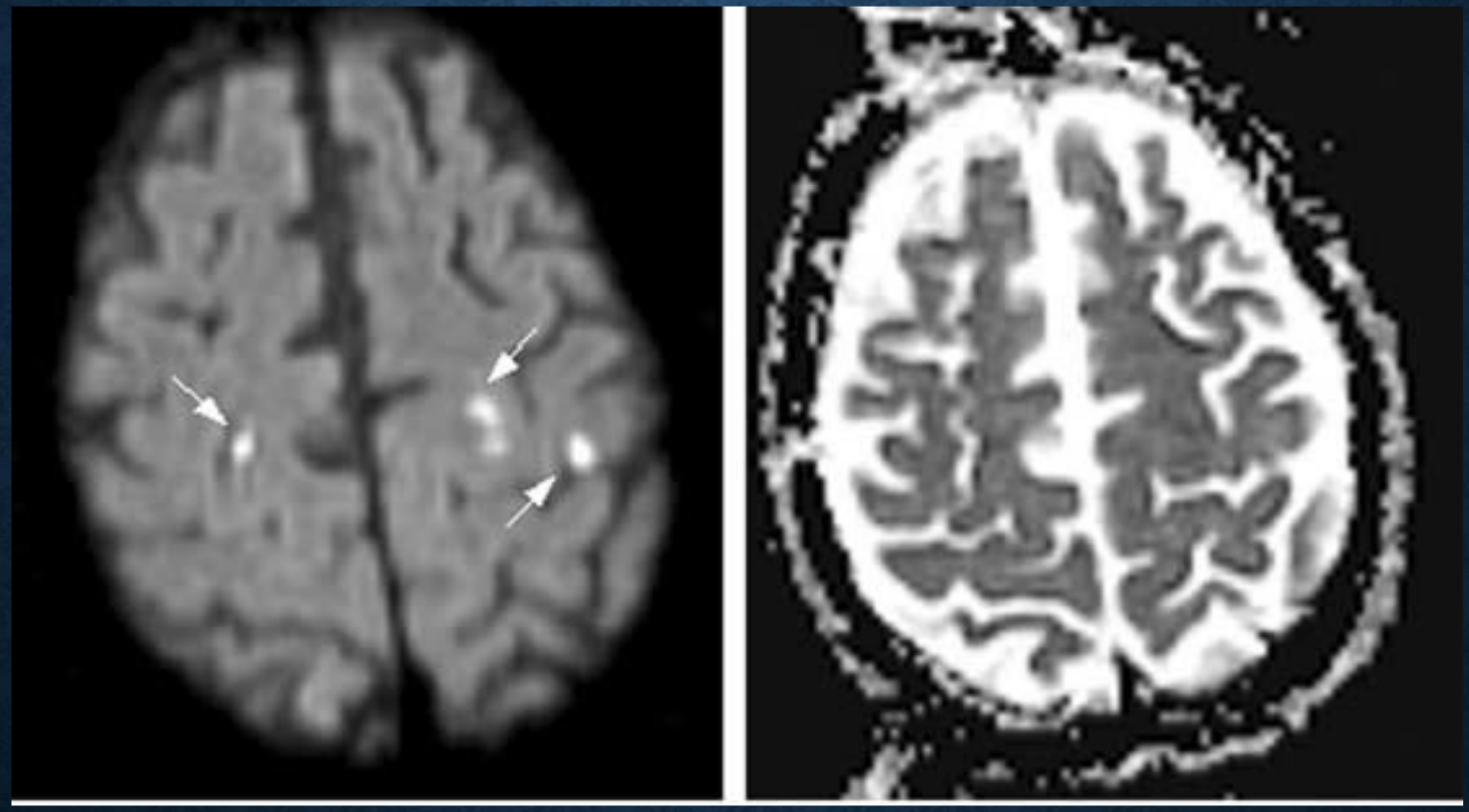
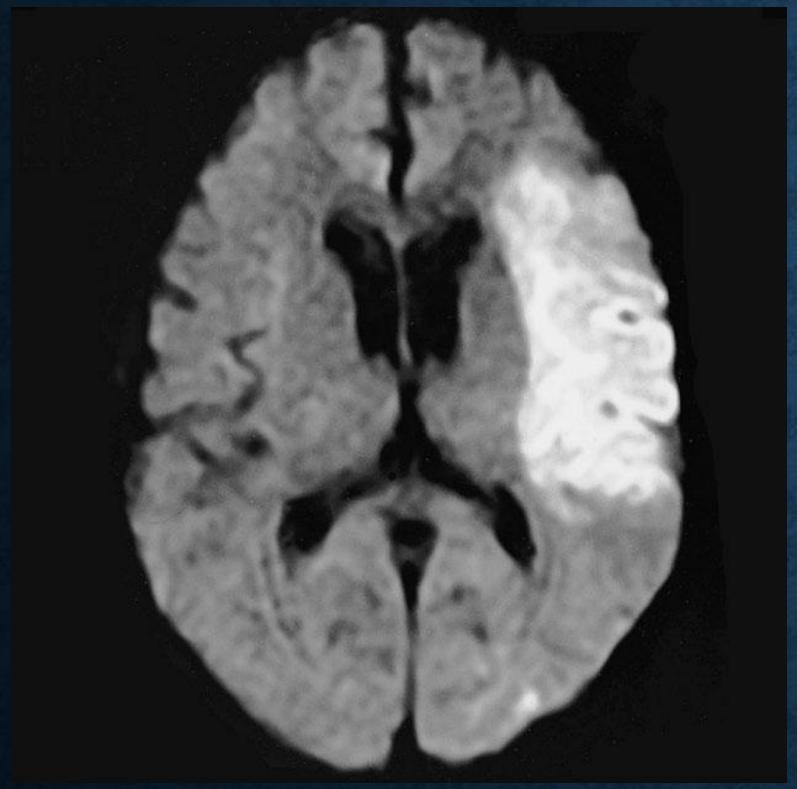
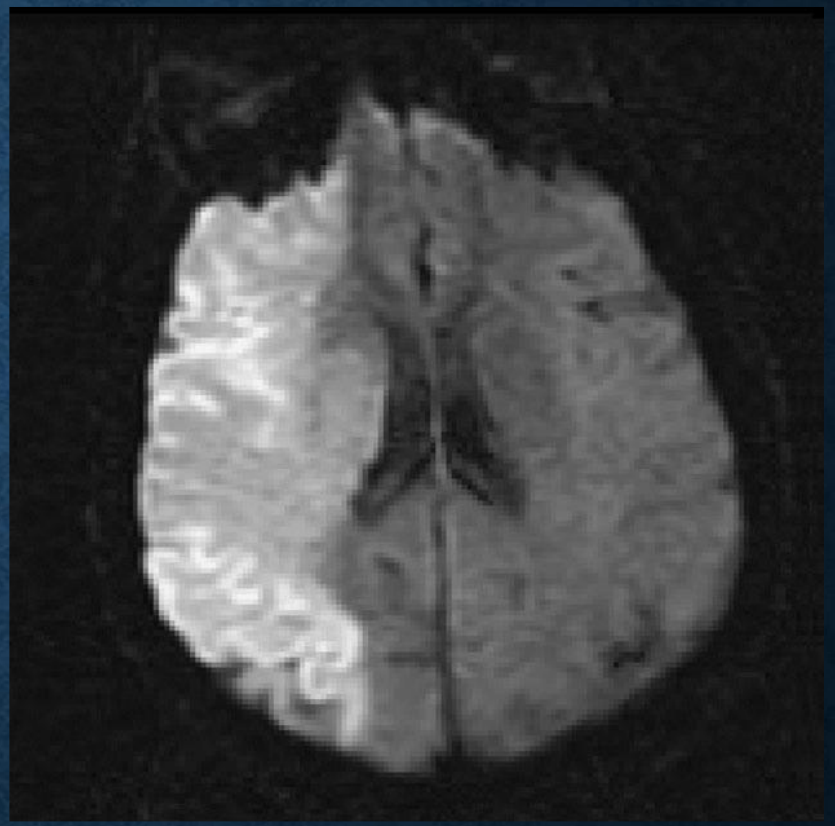
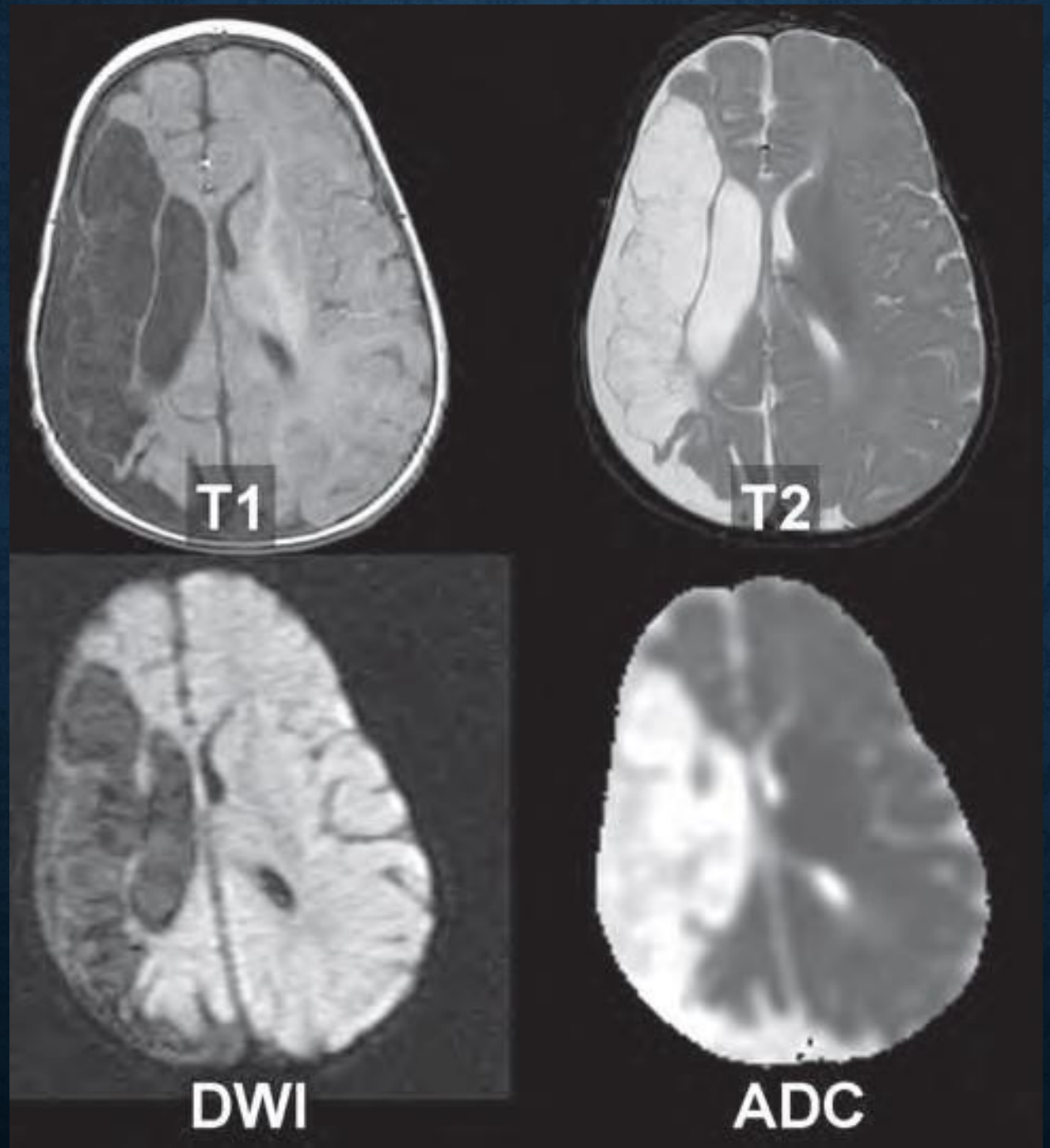











T1
T2
DWI
ADC

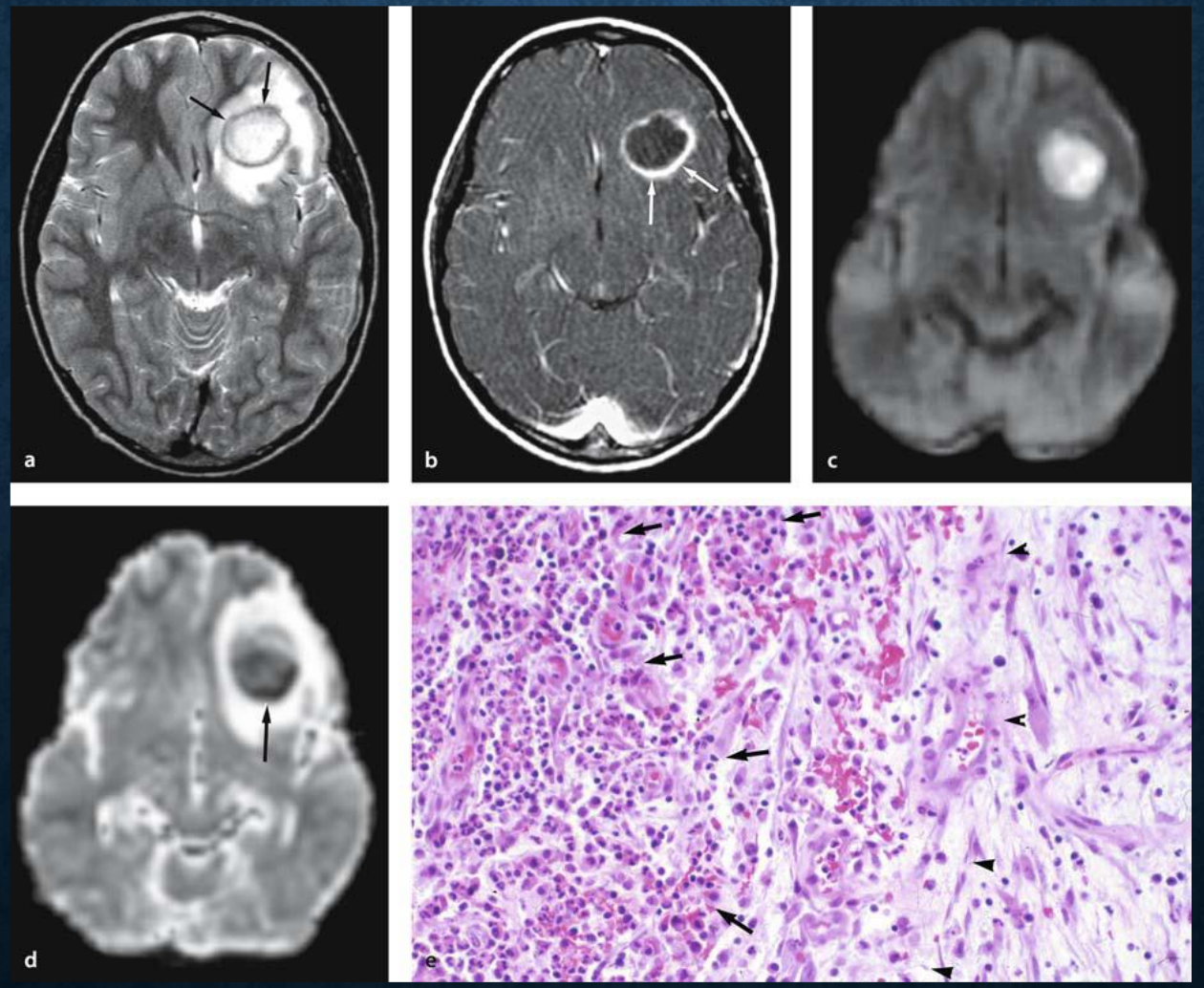
INFECTIOUS DISEASES

The imaging findings vary greatly depending on the organism causing the infection. Diffusion-weighted (DW) imaging is useful for diagnosis of infectious conditions of the brain by means of differentiating vasogenic edema from cytotoxic edema. DW imaging can also separate abscesses from cystic and necrotic tumors.

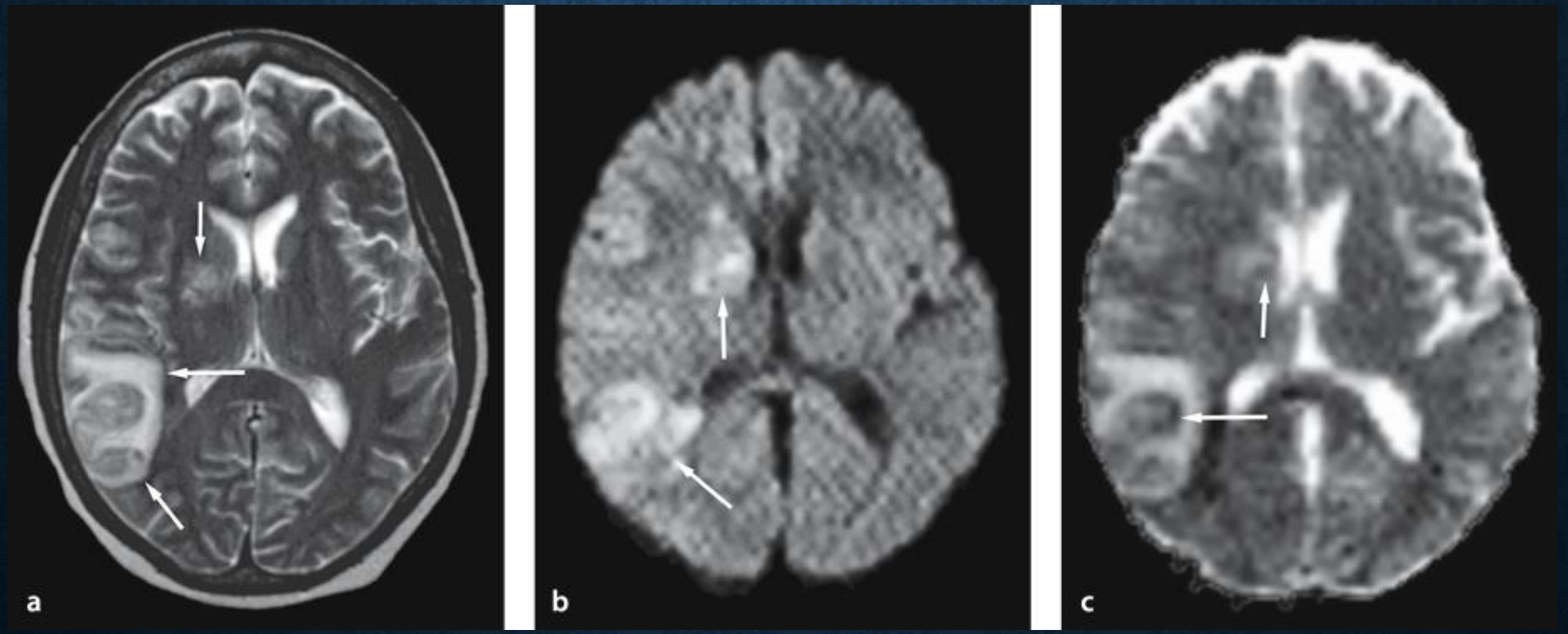
INFECTIOUS DISEASES

The early phase of the brain abscess has a homogeneous, bright signal on DW imaging associated with decreased apparent diffusion coefficient (ADC). On follow-up DW imaging, the chronic phase of an abscess can still show hyperintensity, but ADC values are partially increased. A possible explanation for the high signal on DW imaging is restriction of water mobility due to the high viscosity of coagulative necrosis and the hypercellularity of polynucleated neutrophils in the pus.

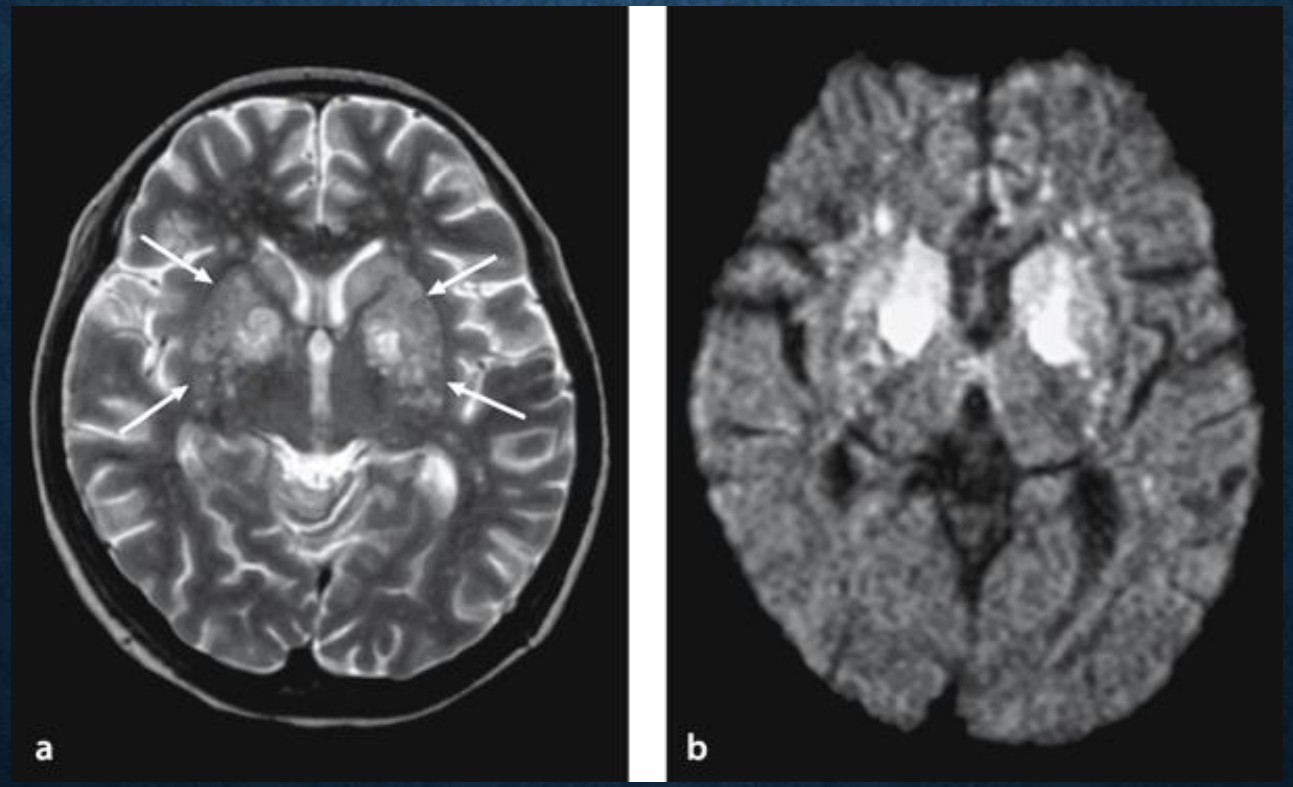
CHRONIC STREPTOCOCCAL BRAIN ABSCESS



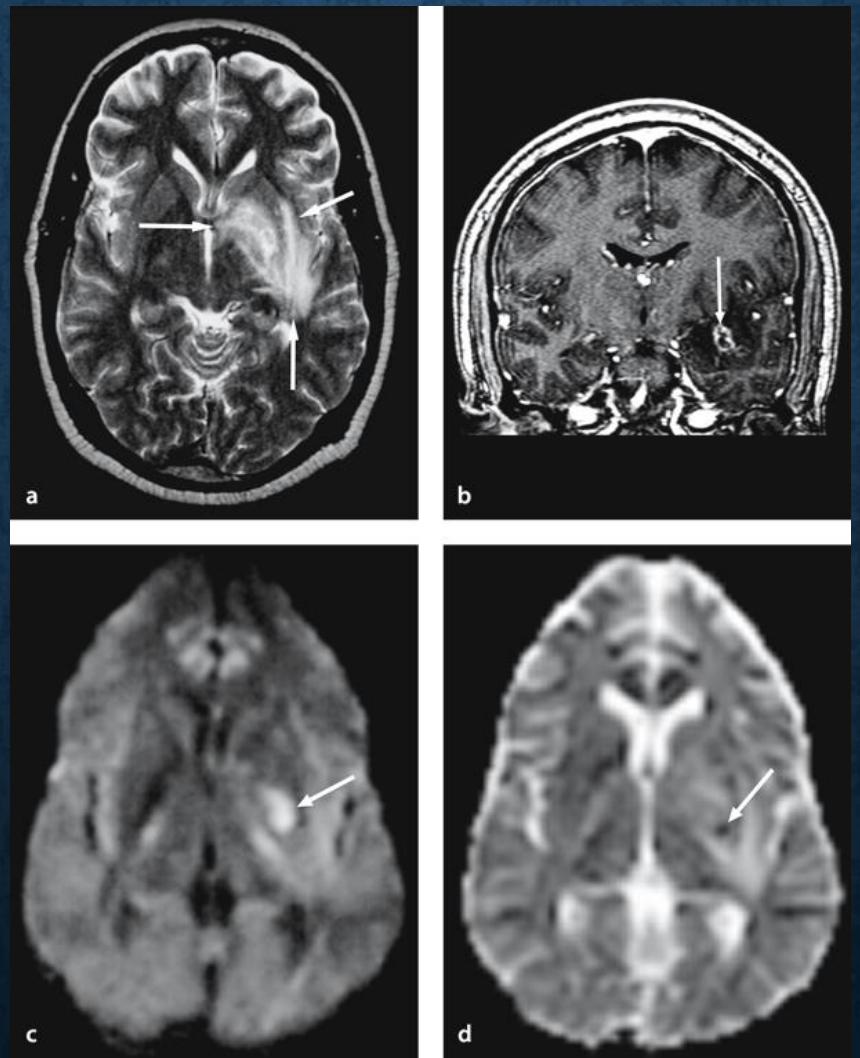
SEPTIC EMBOLI FROM STAPHYLOCOCCUS ENDOCARDITIS

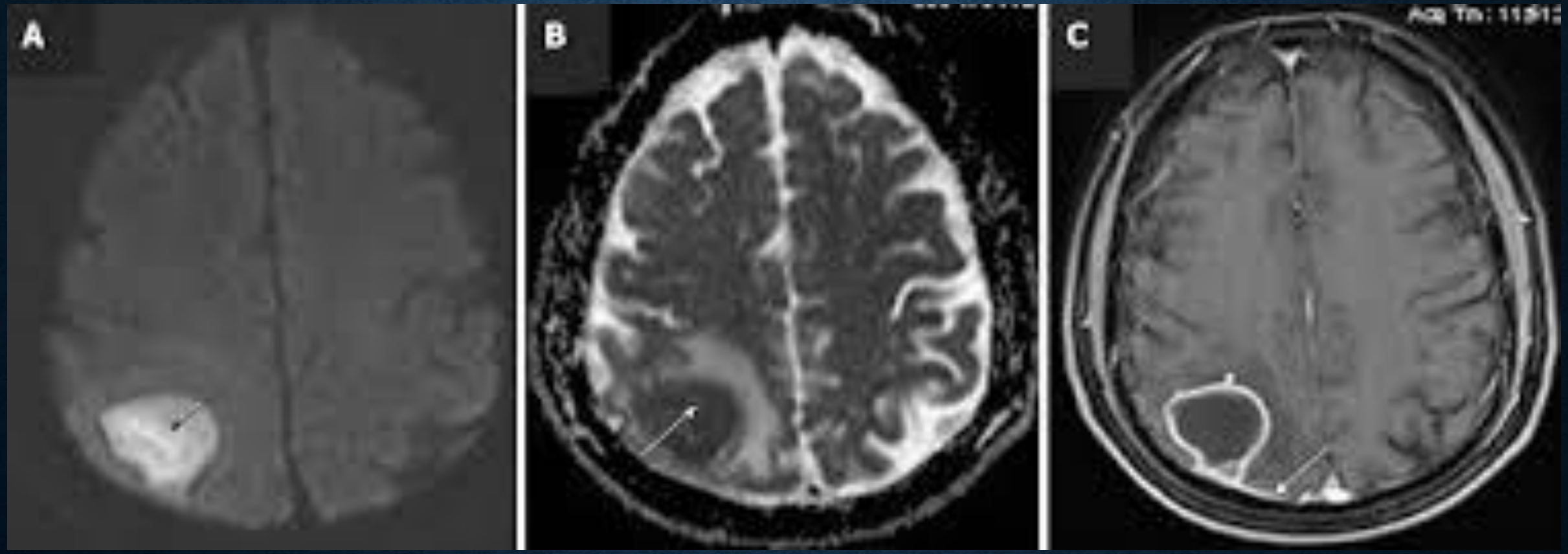


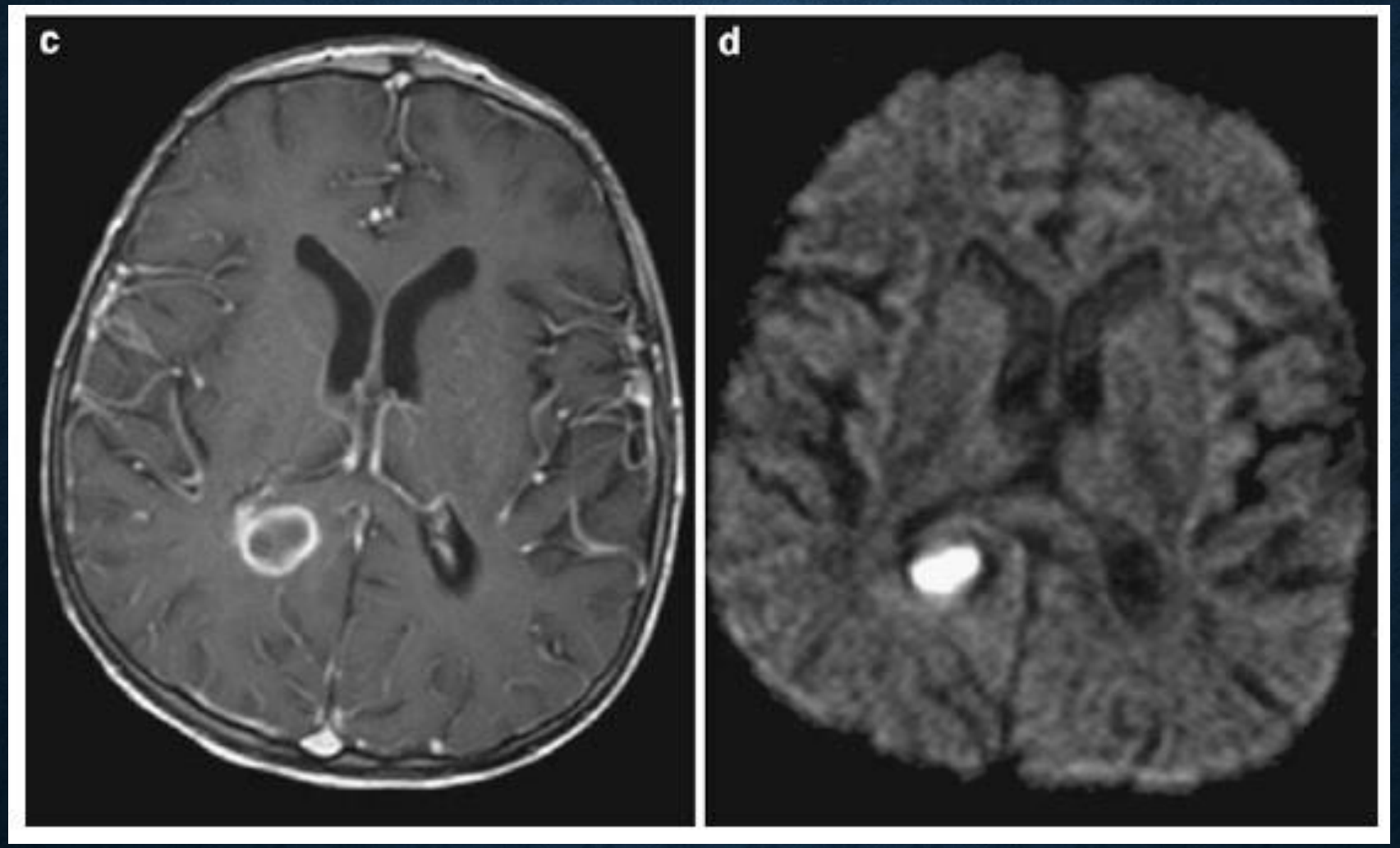
BRAIN ABSCESSES DUE TO GRAM-NEGATIVE BACTERIA

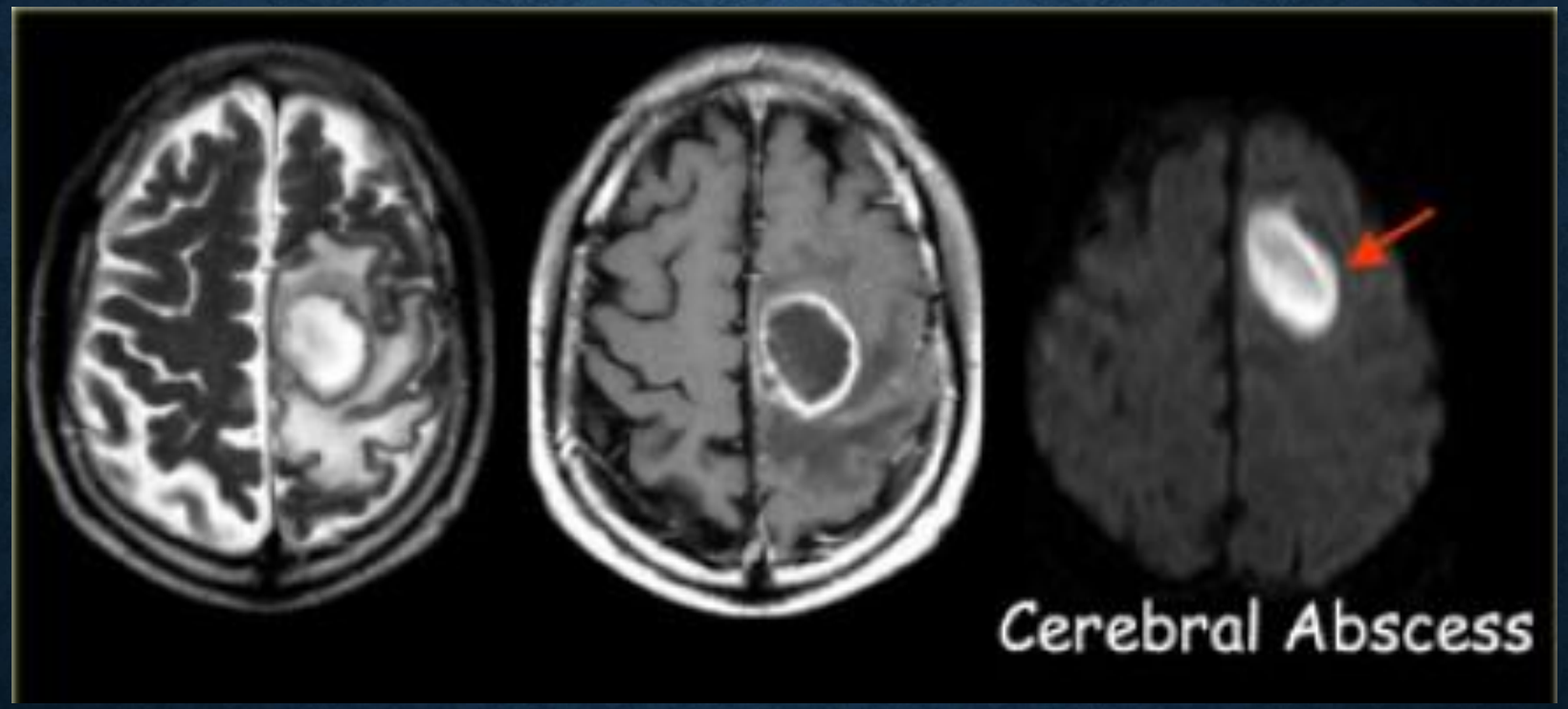


LISTERIA MENINGOENCEPHALITIS

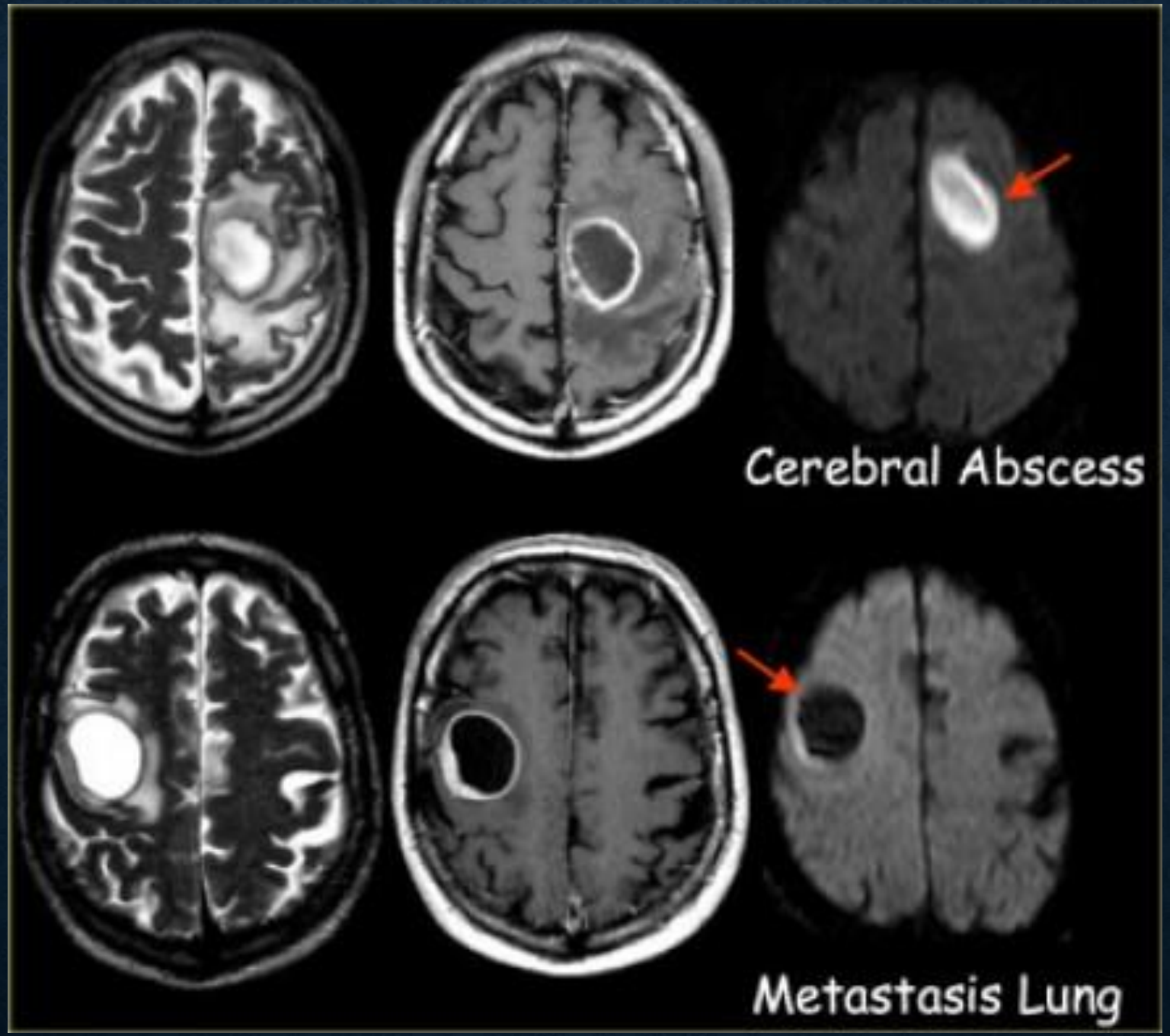


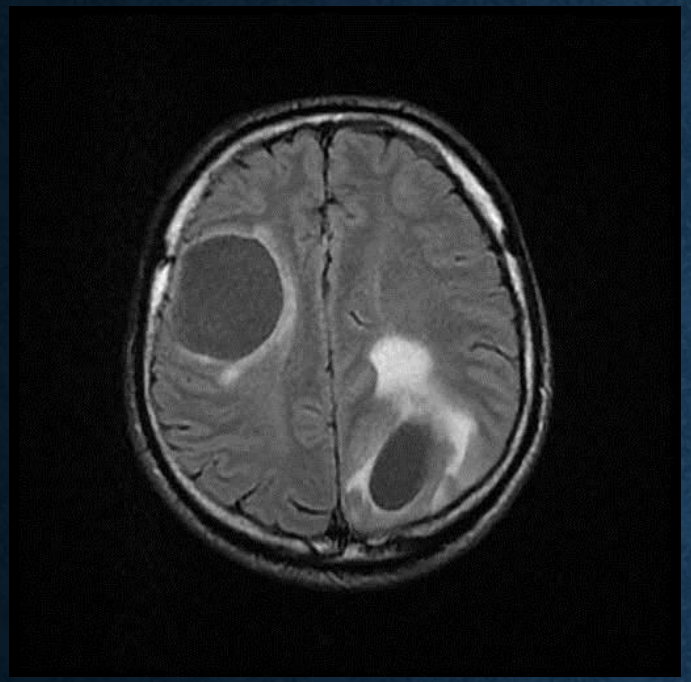
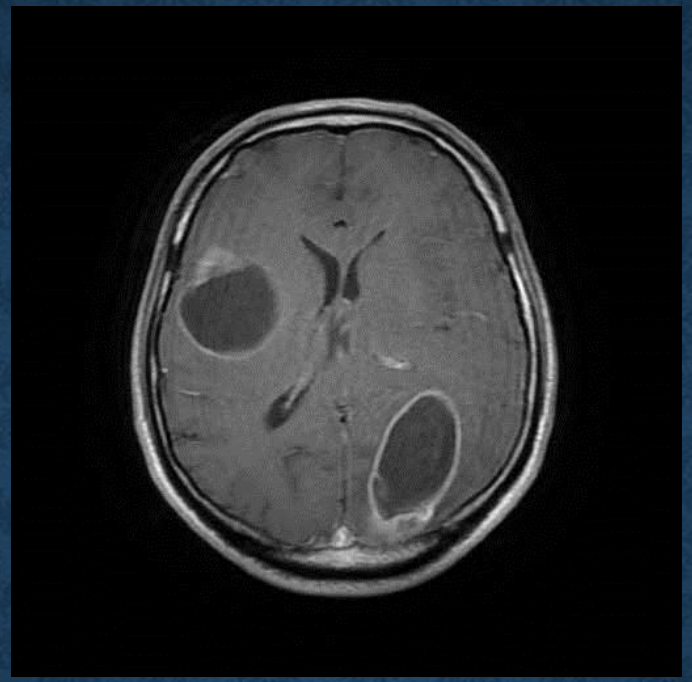
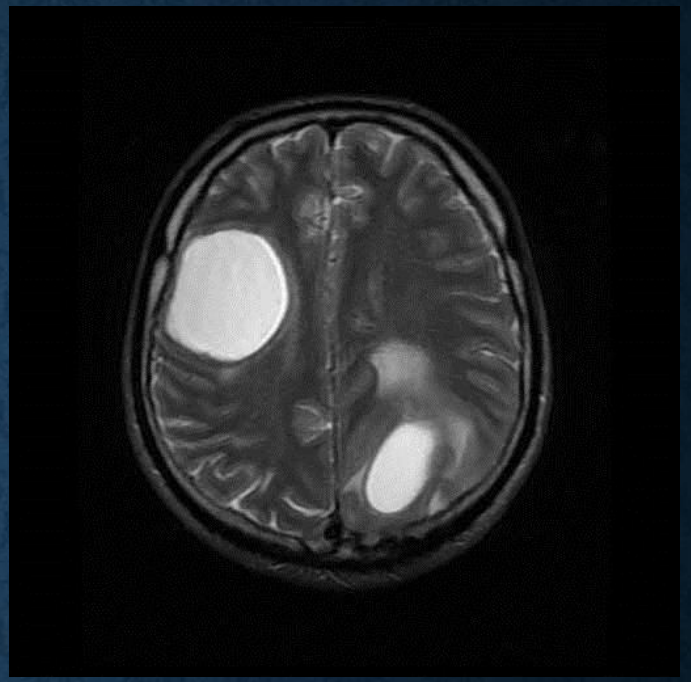
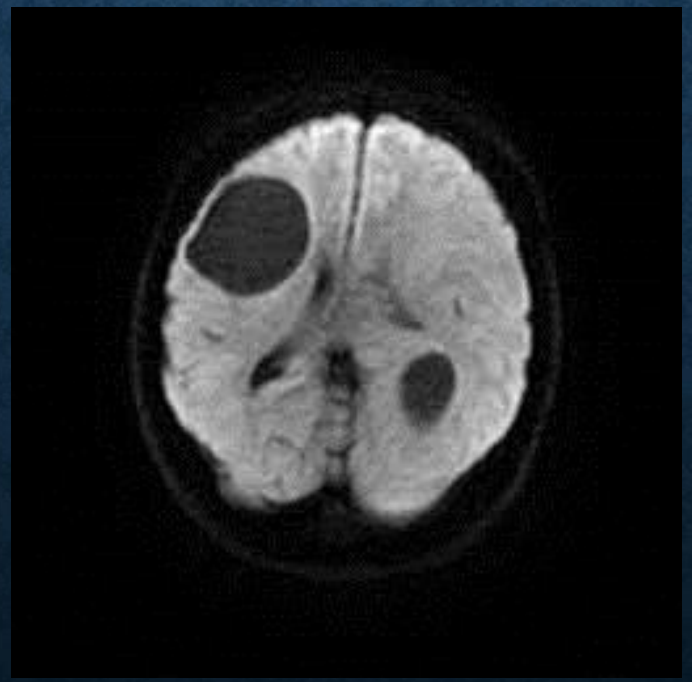

A
B
C

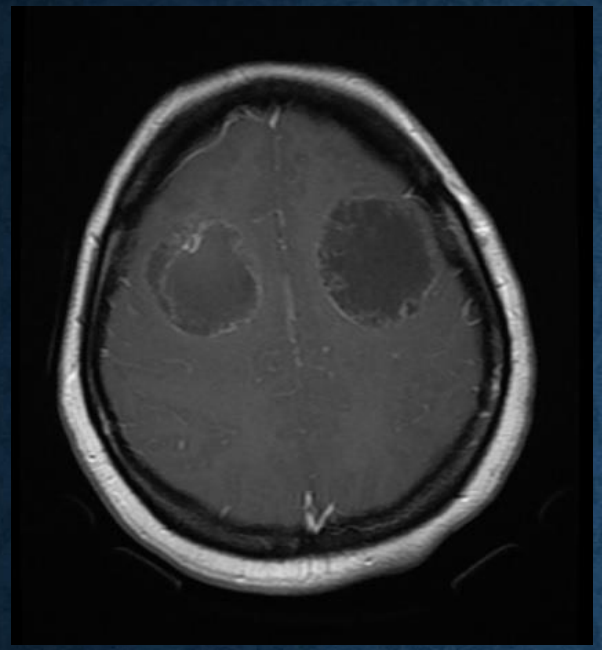

c
d

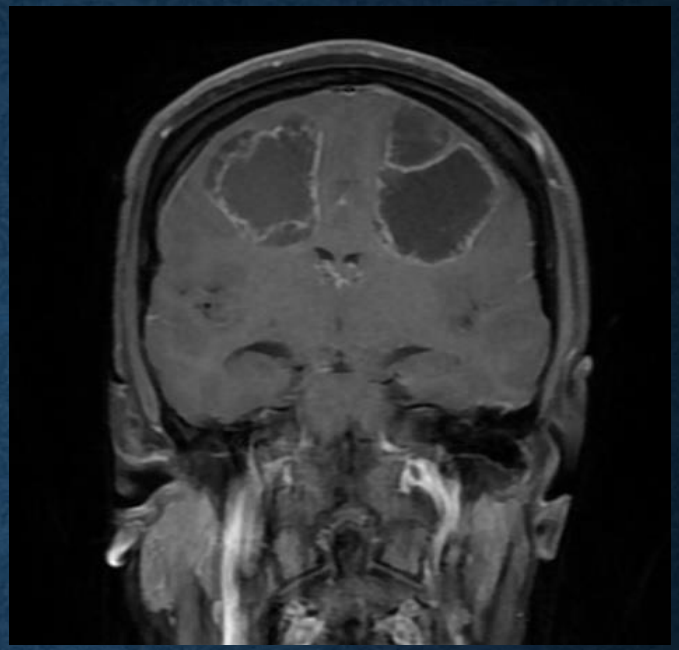

Cerebral Abscess

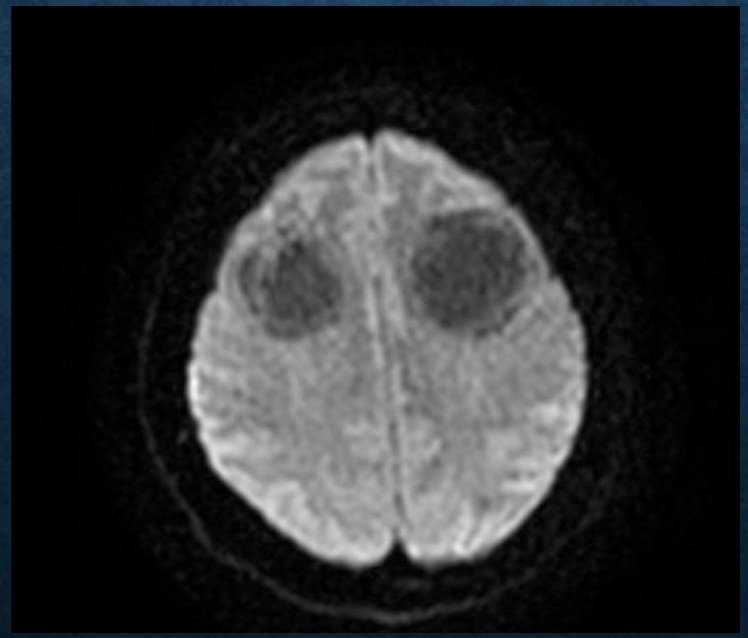
NEOPLASMS IN DWI


Cerebral Abscess
Metastasis Lung

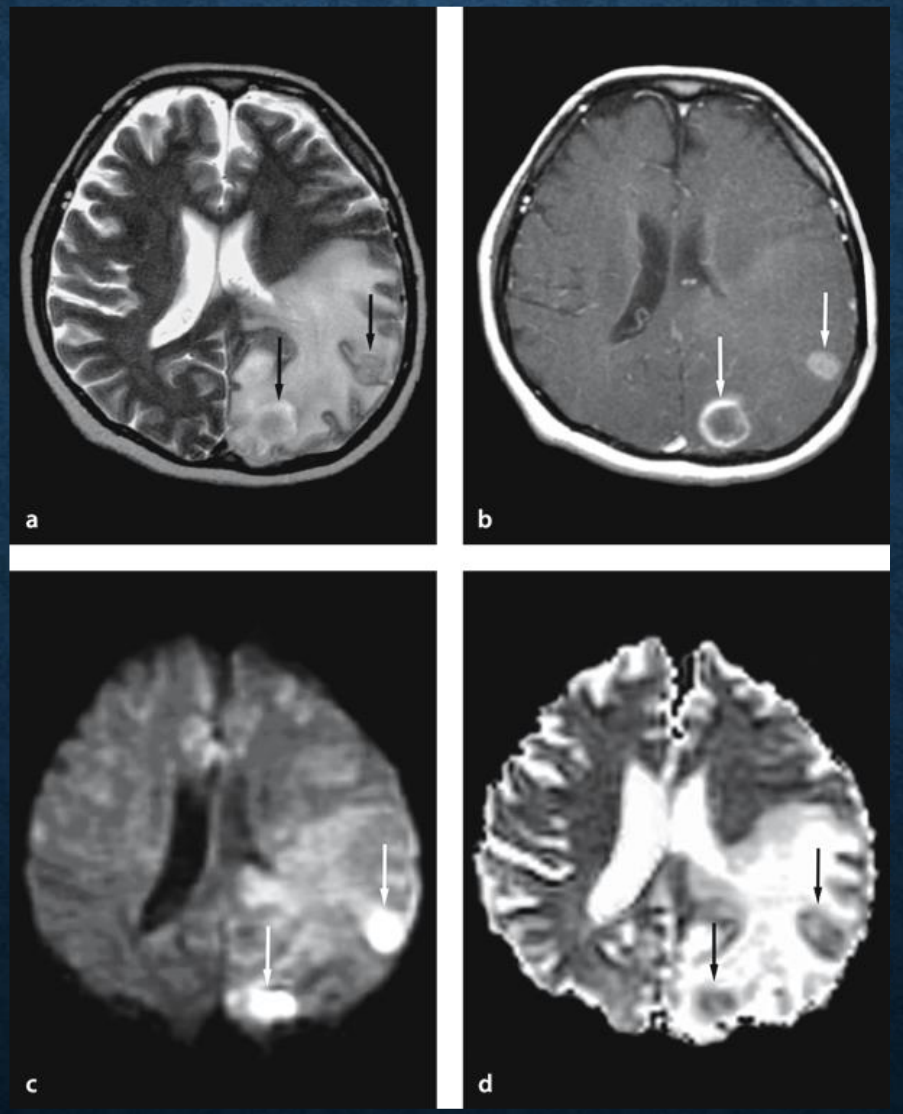











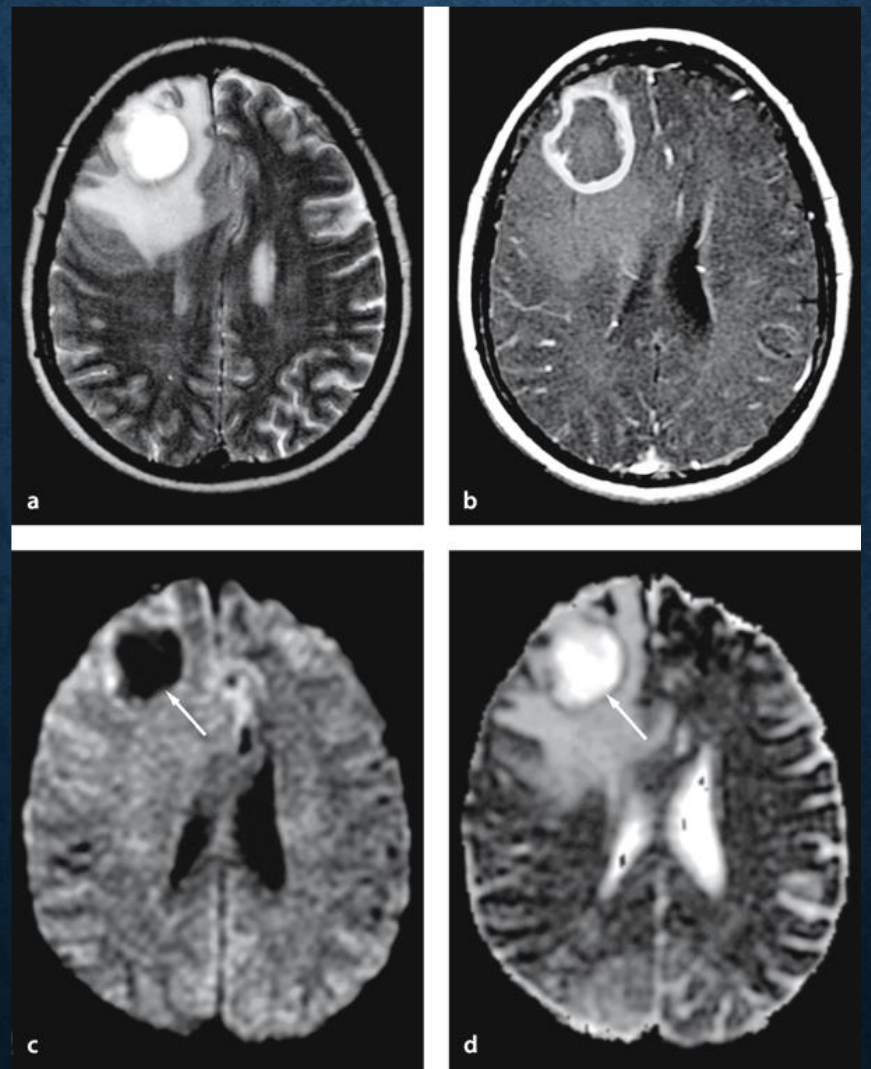
BRAIN METASTASIS FROM GI CARCINOMA



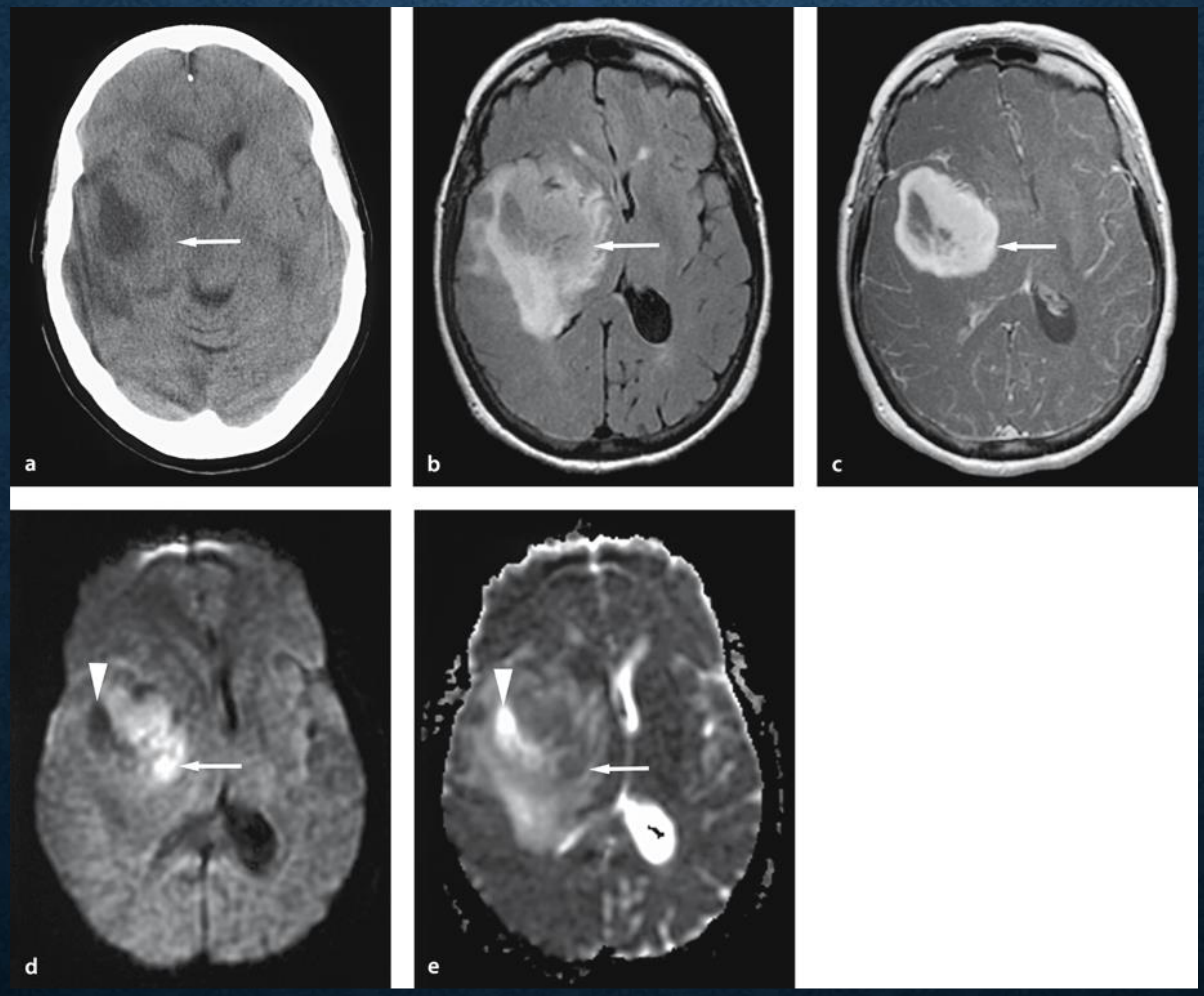
GLIOMAS

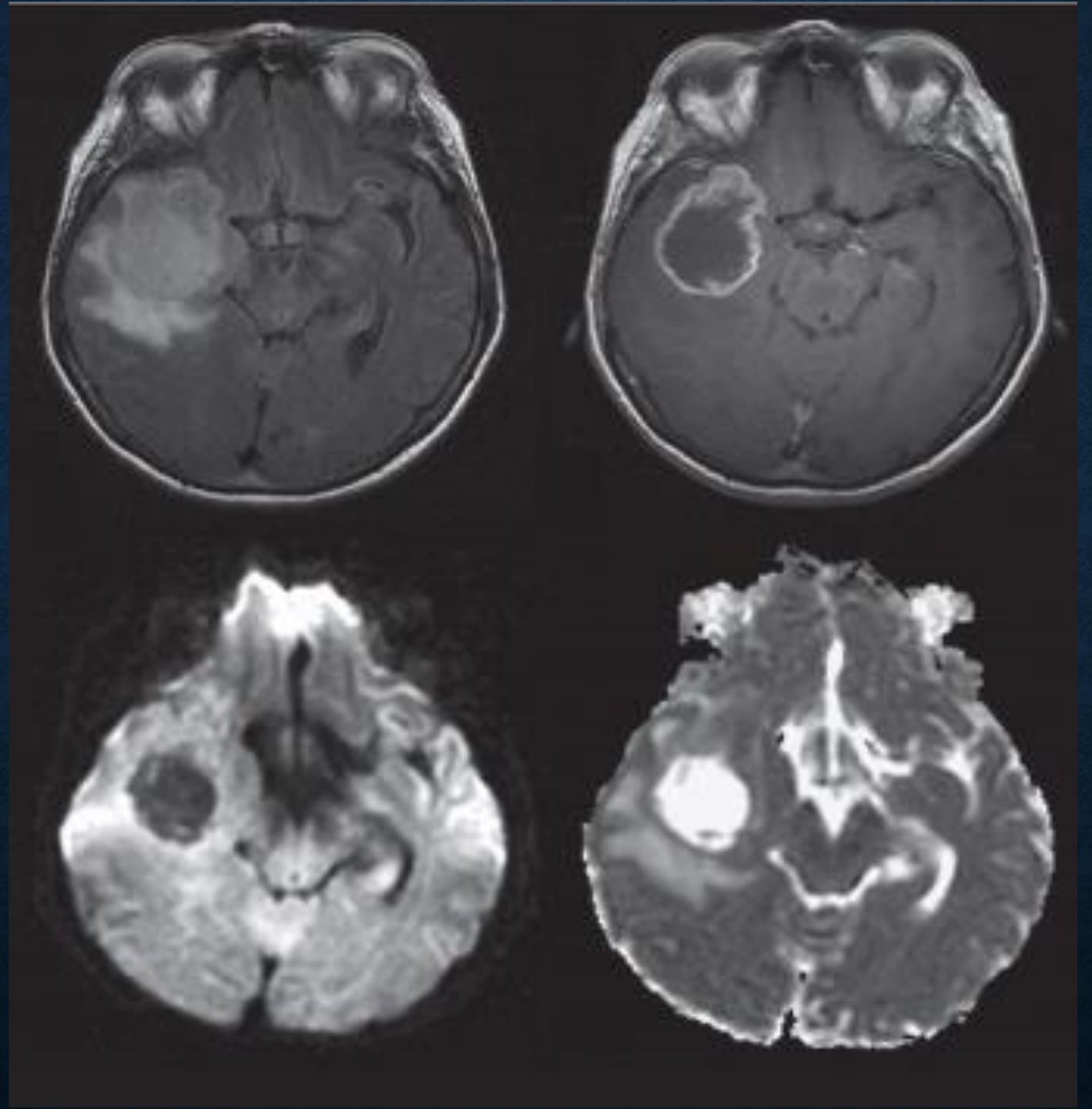
The signal intensity of gliomas on DW images is variable and depends mainly on their T2 and apparent diffusion coefficient (ADC) values. Thus, some gliomas are hyperintense on DW images with decreased ADC, which reflects a reduced volume of the extracellular space. Other gliomas have a normal or increased ADC, that is the DW signal is a T2 shinethrough effect.

GLIOBLASTOMA MULTIFORME



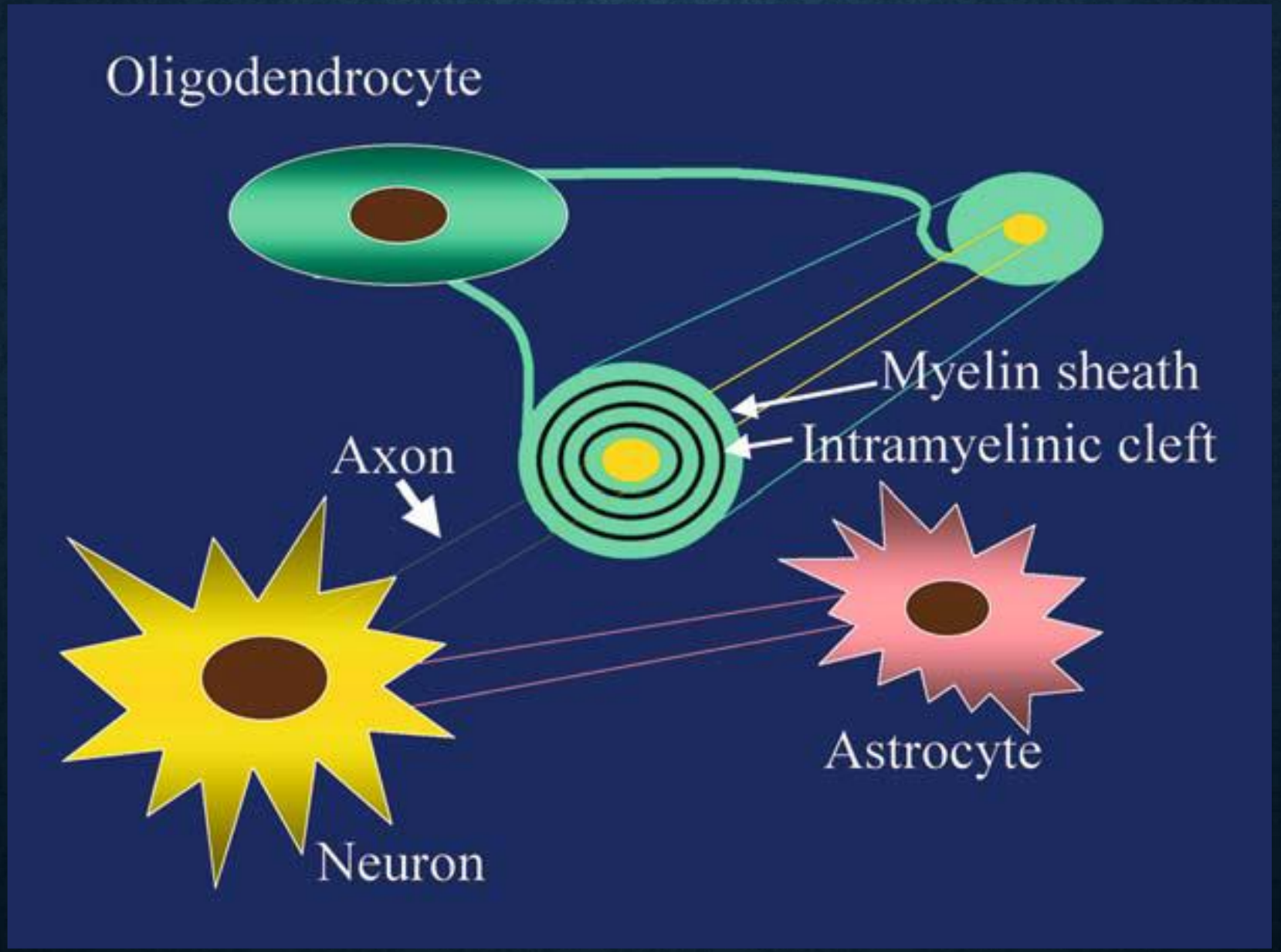
GLIOBLASTOMA MULTIFORME

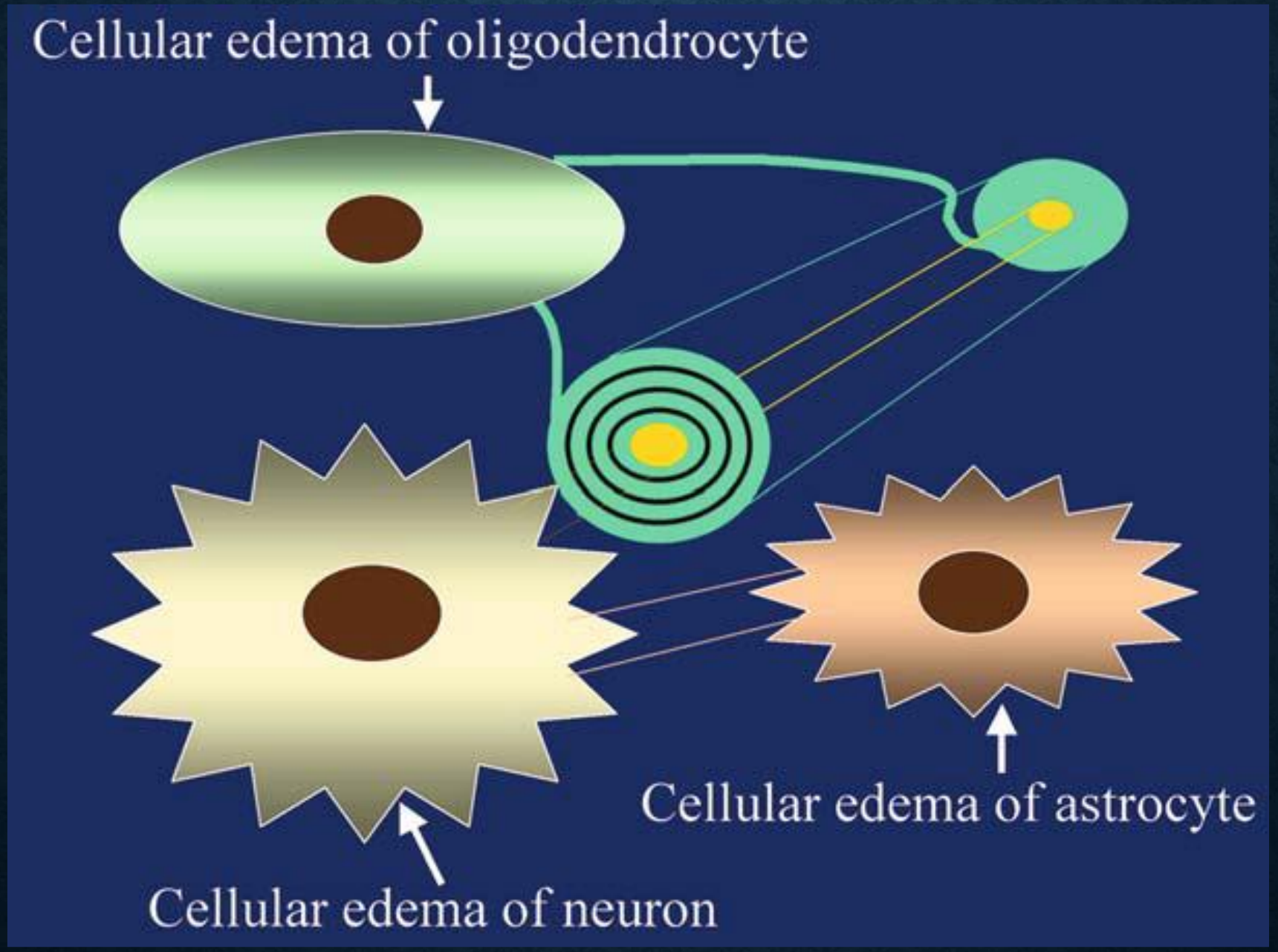




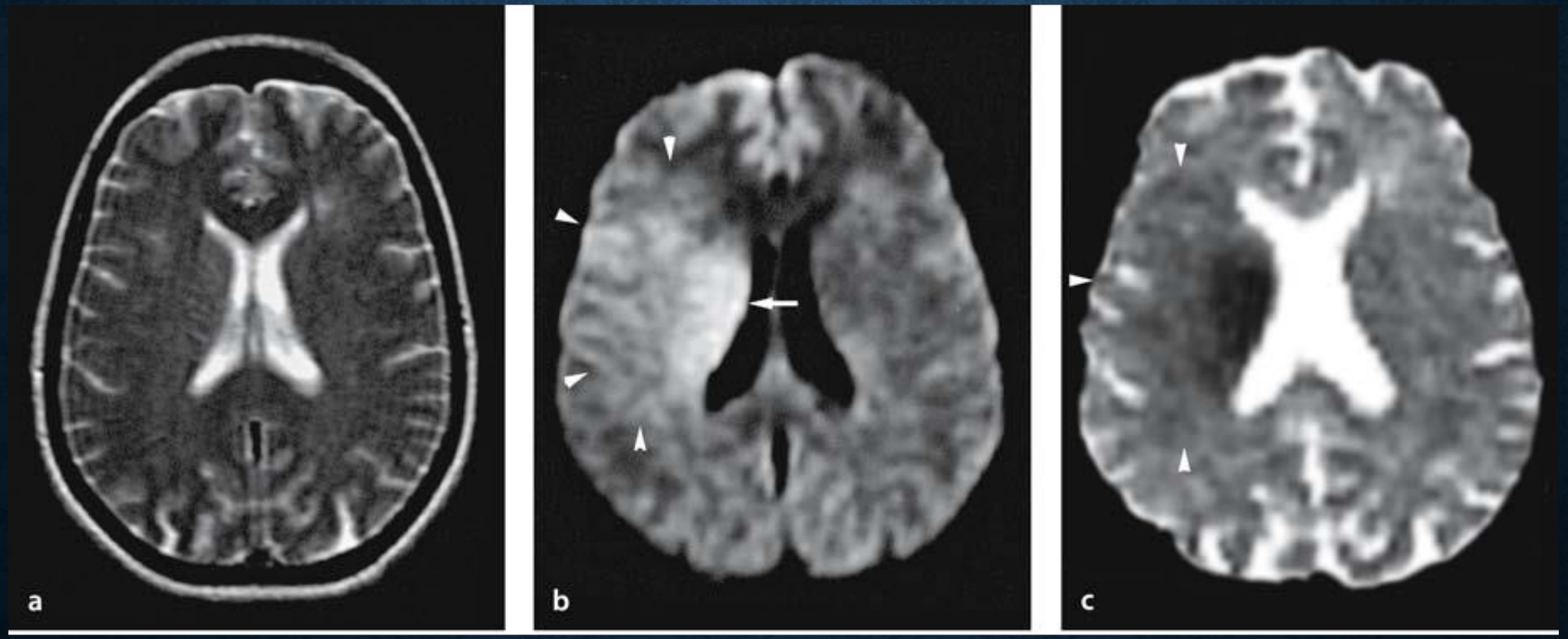
BRAIN EDEMA IN DWI

- Cytotoxic edema characteristically shows hyperintensity on DW images associated with decreased apparent diffusion coefficient (ADC).

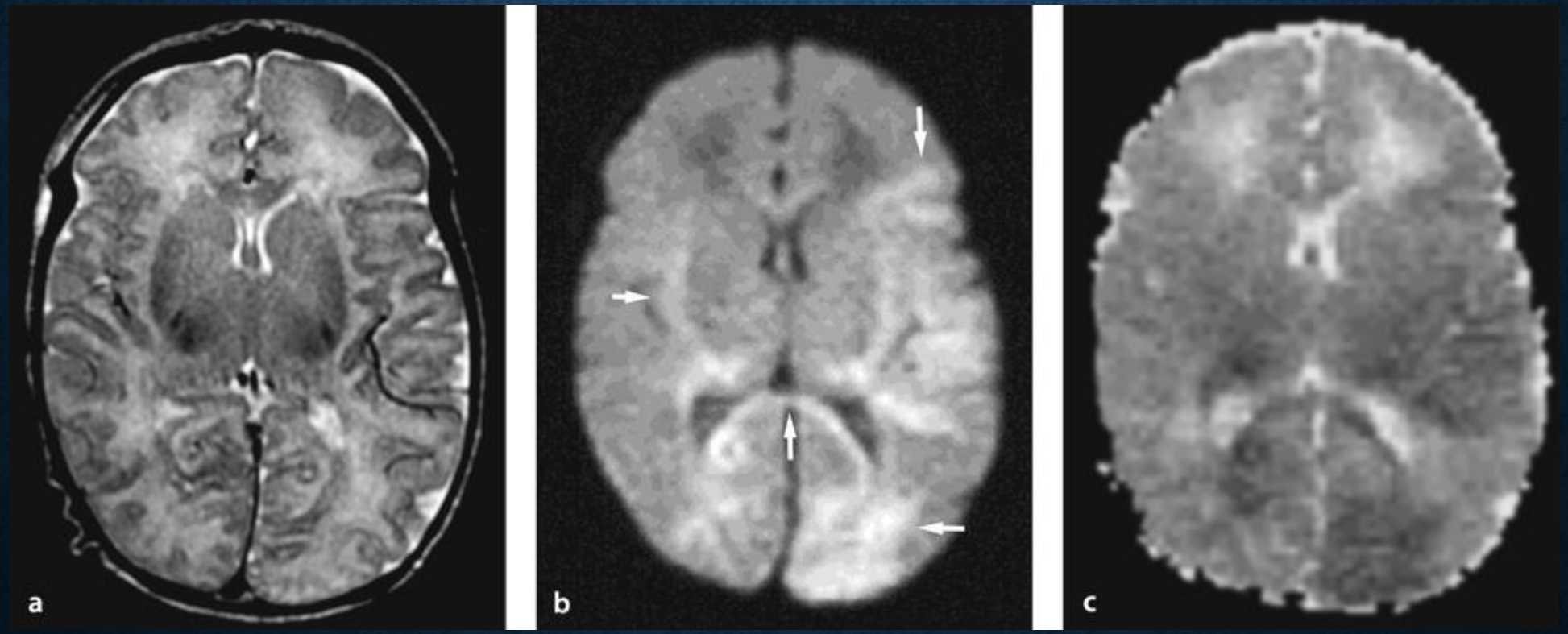

Oligodendrocyte
Myelin sheath
Intramyelinic cleft
Axon
Astrocyte
Neuron


Cellular edema of oligodendrocyte
Cellular edema of astrocyte
Cellular edema of neuron

CYTOTOXIC EDEMA



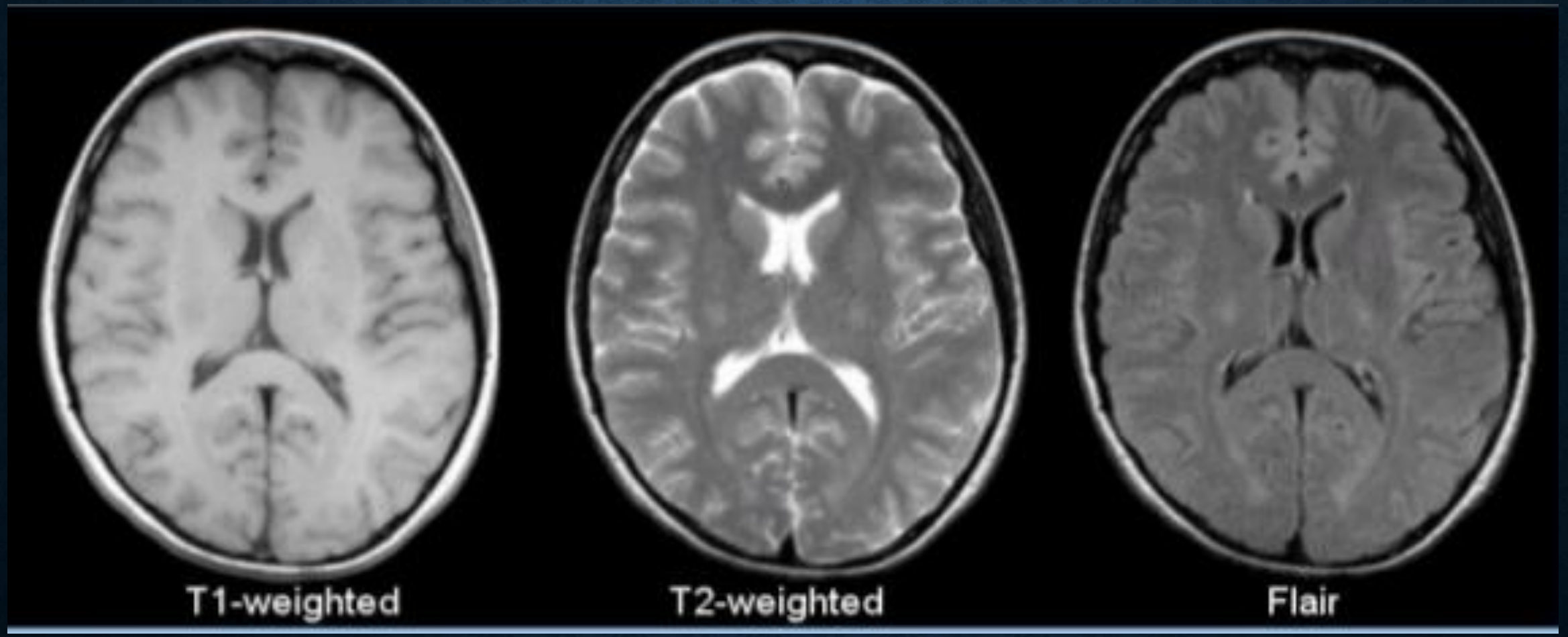
HYPOXIC ISCHEMIC ENCEPHALOPATHY CYTOTOXIC EDEMA

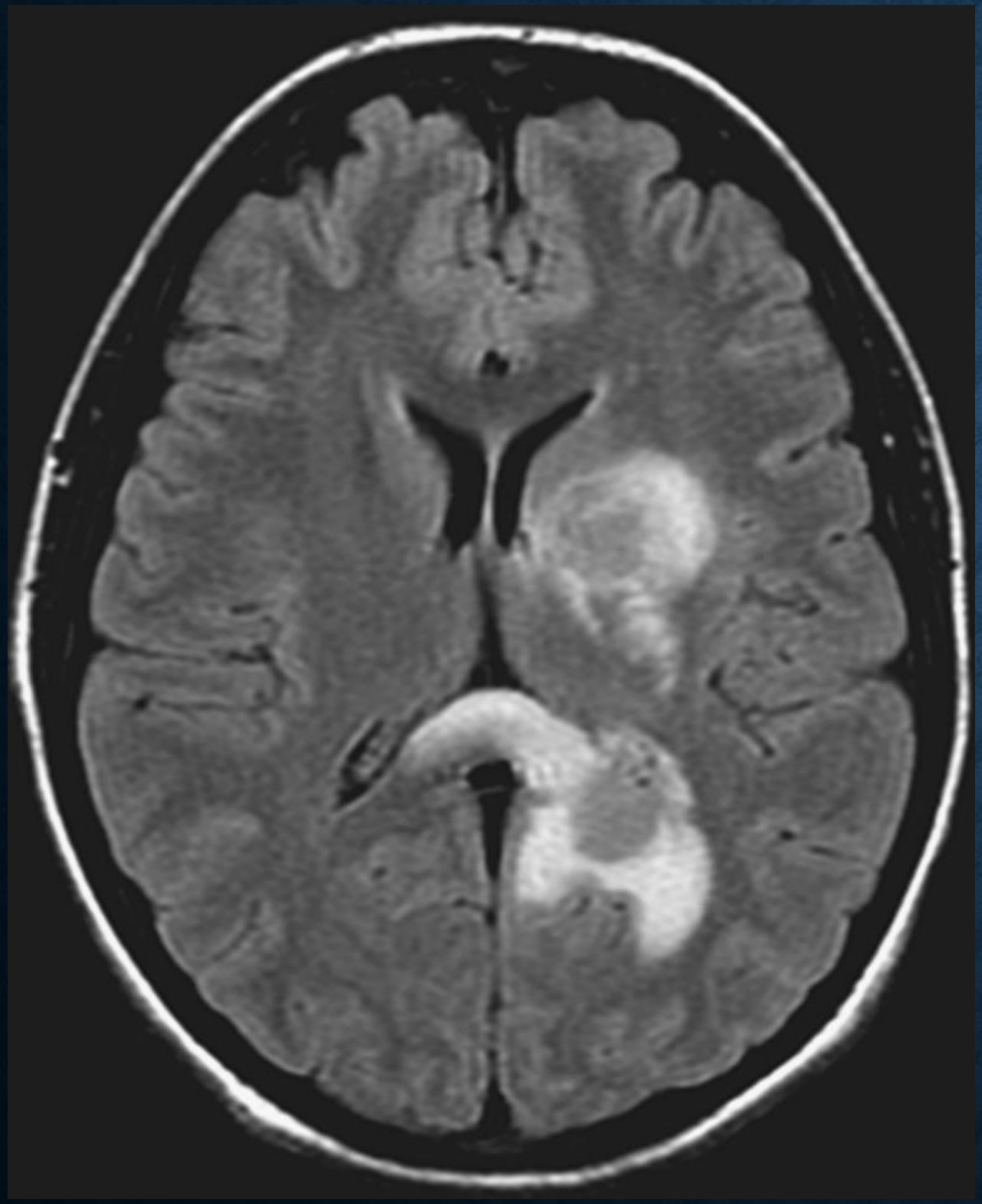


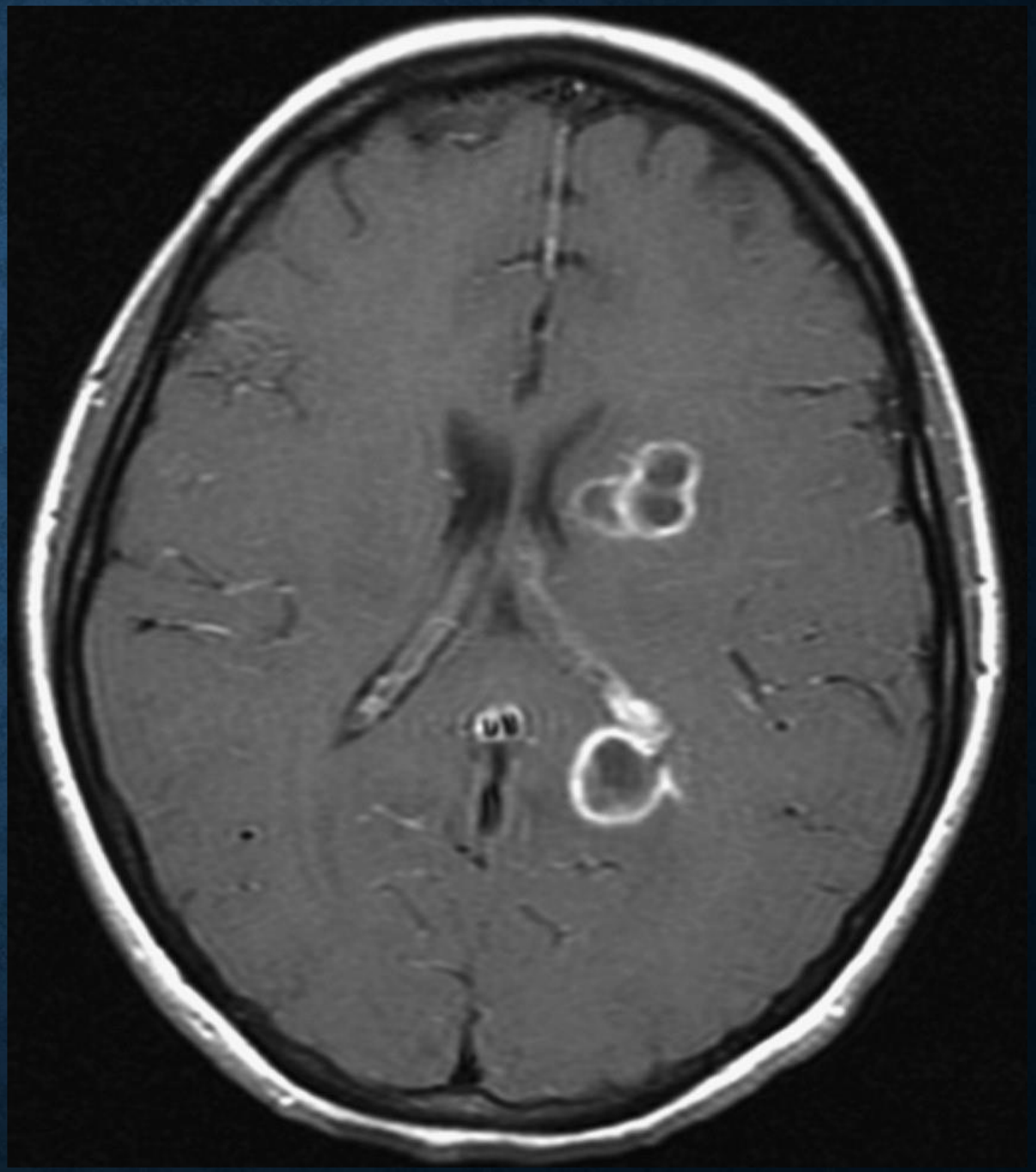
INVERSION RECOVERY MODALITIES

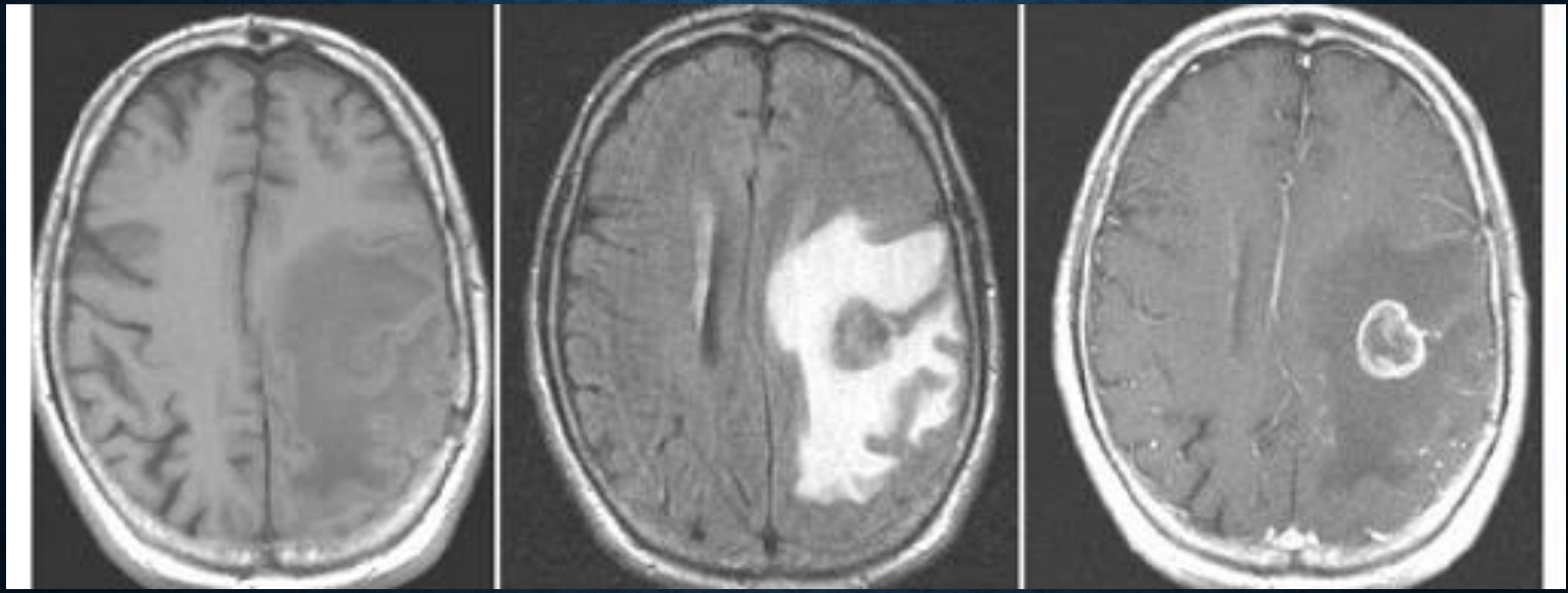
- Short Tau Inversion Recovery (STIR)
- Fluid Attenuated Inversion Recovery (FLAIR)

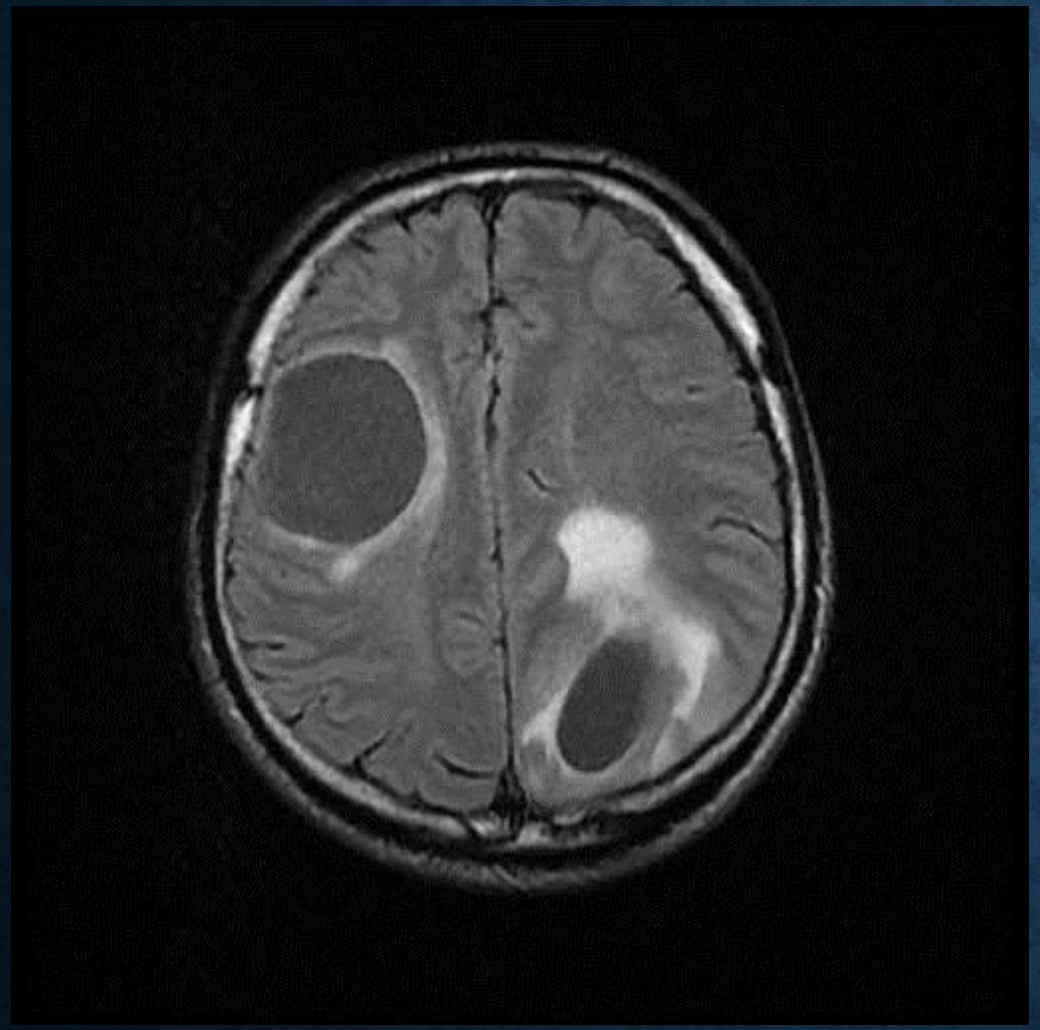
FLUID ATTENUATED INVERSION RECOVERY (FLAIR)

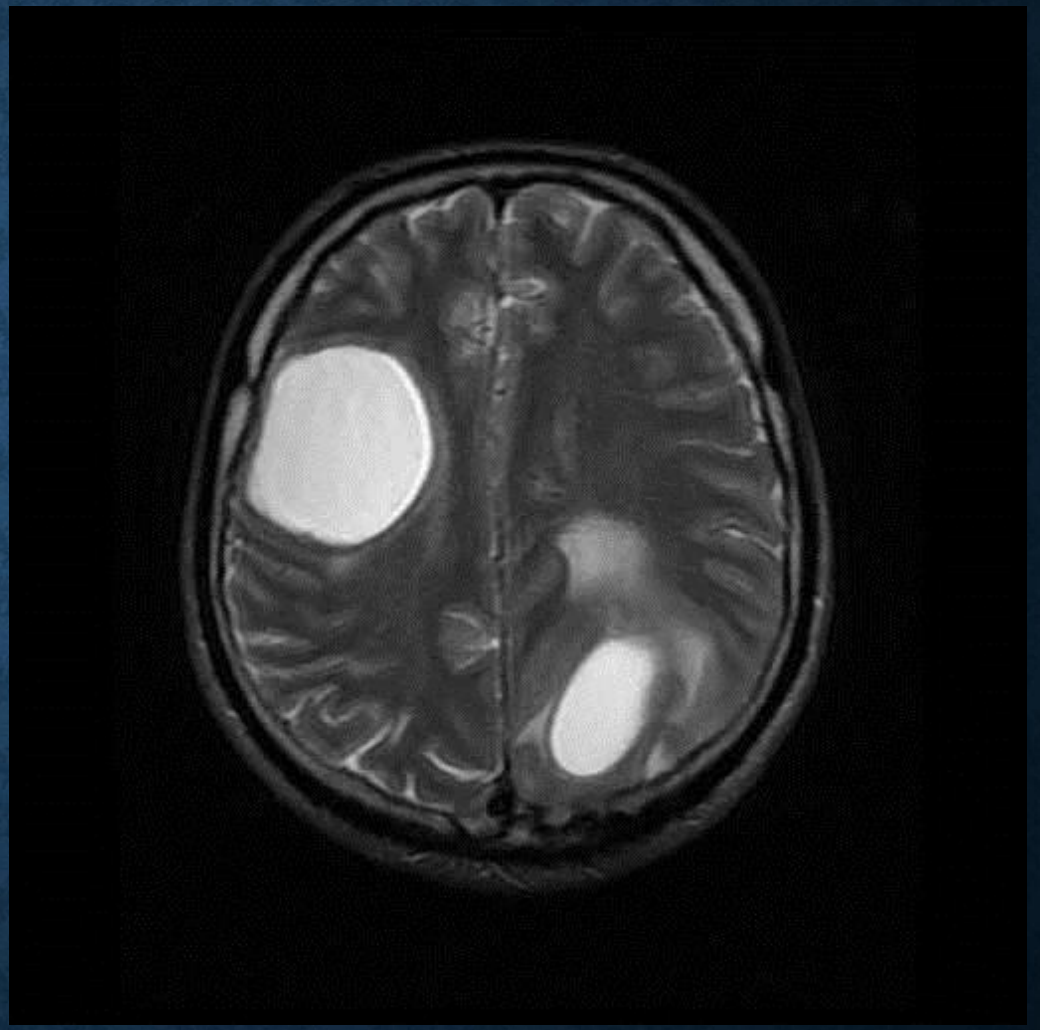

T1-weighted
T2-weighted
Flair

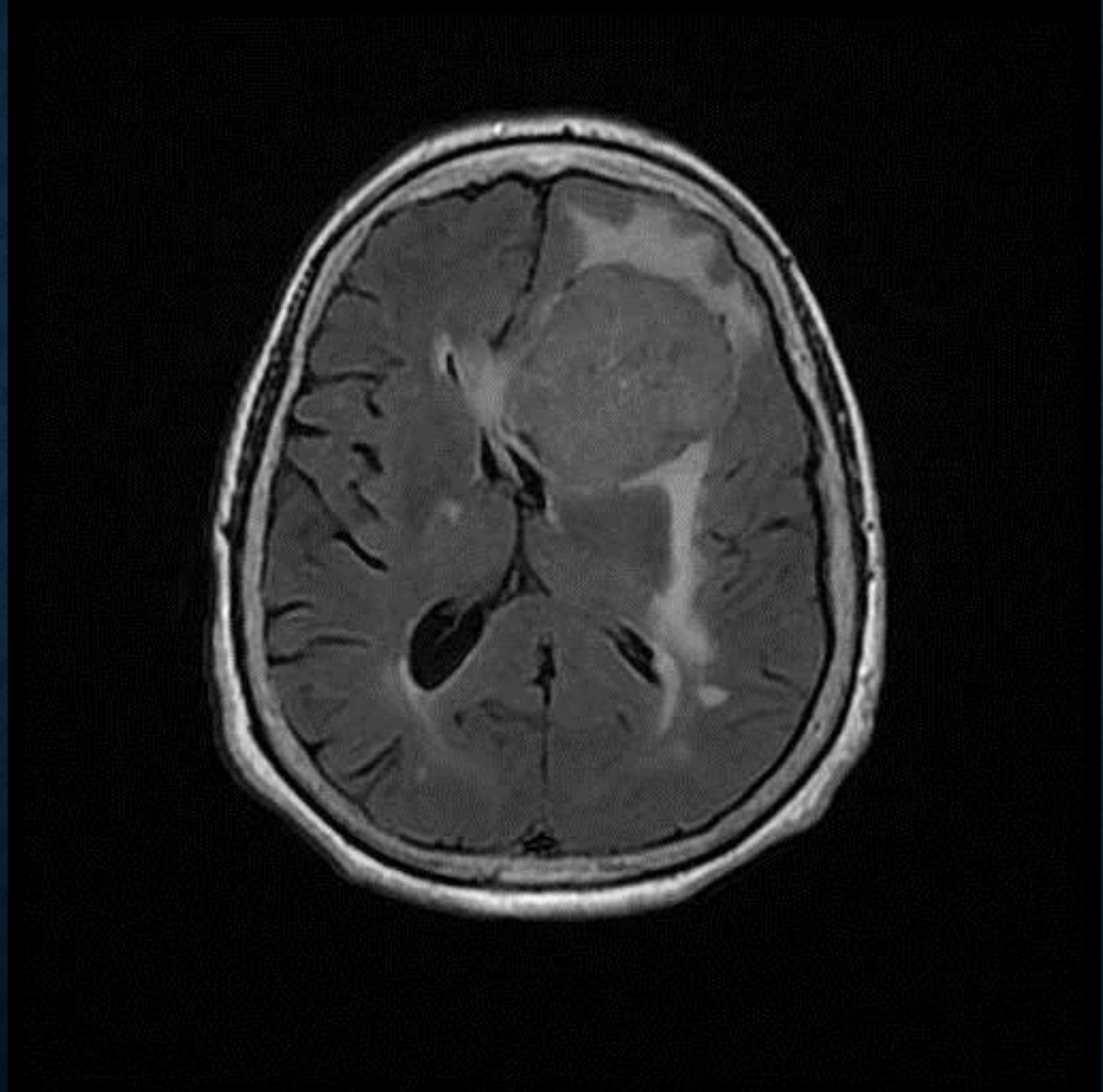


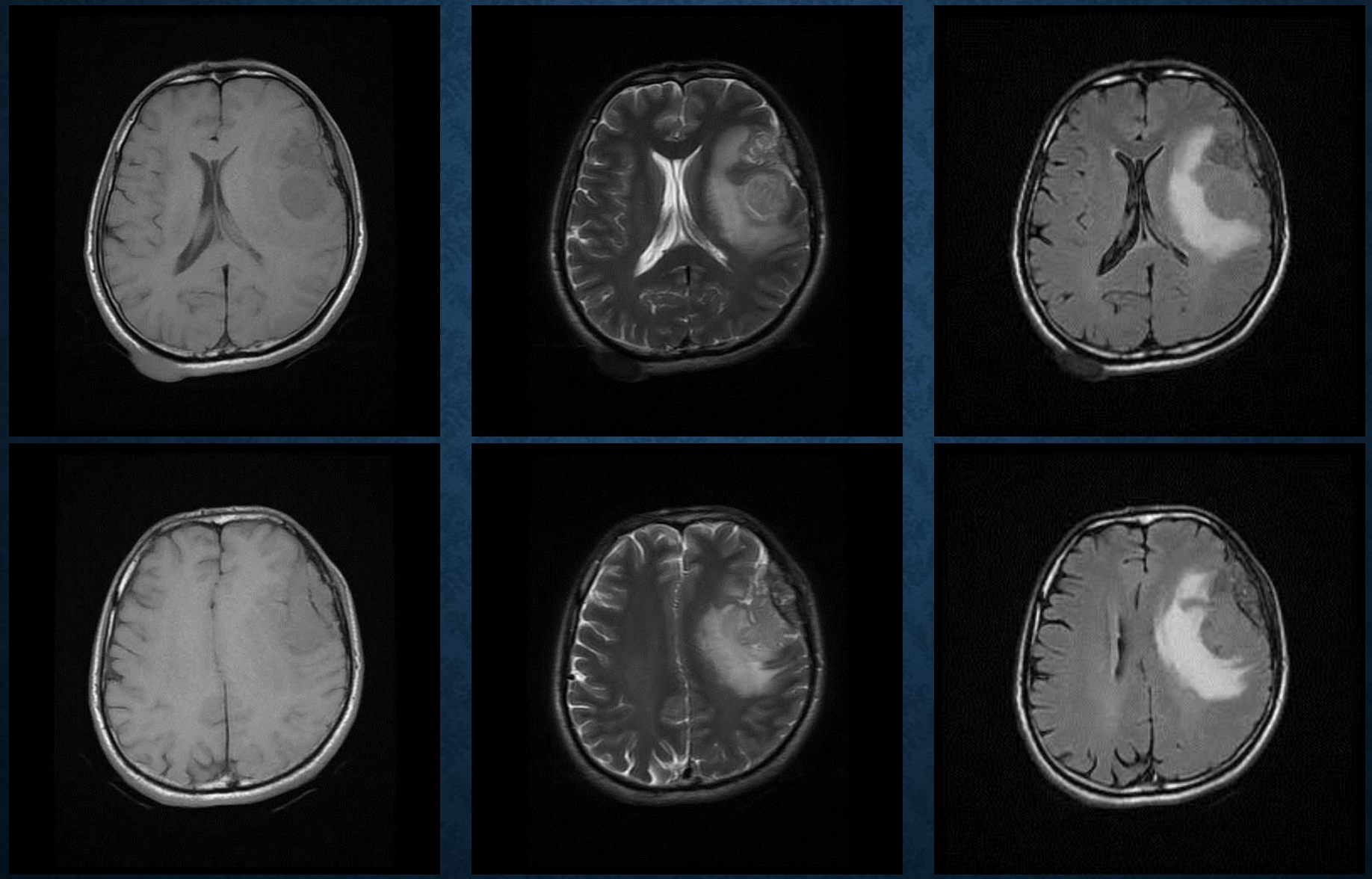


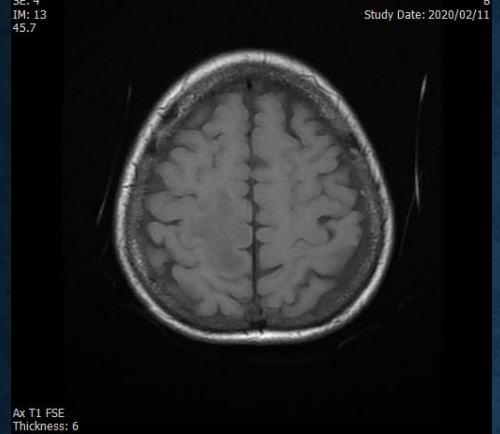








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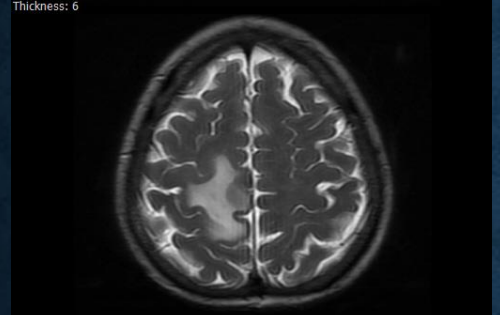
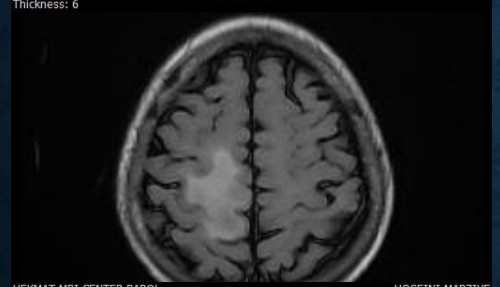
45.7
Ax T1 FSE
Thickness: 6


Ax FSE T2
Thickness: 6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HOSEINI MARZIYE
Signa HDxt
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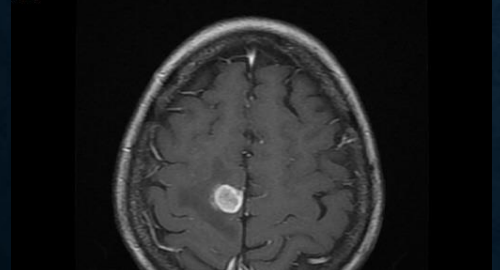
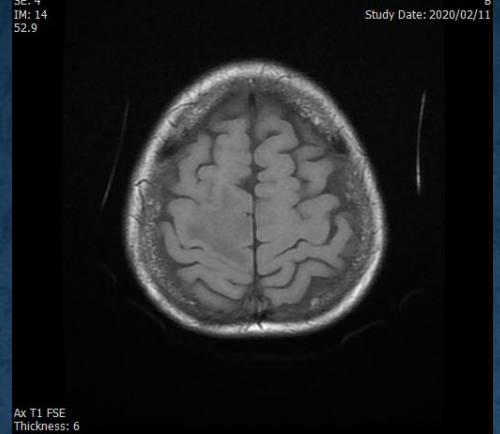
SE: 4
L
IM: 17
Study Date: 2020/02/13
43.3

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52.9
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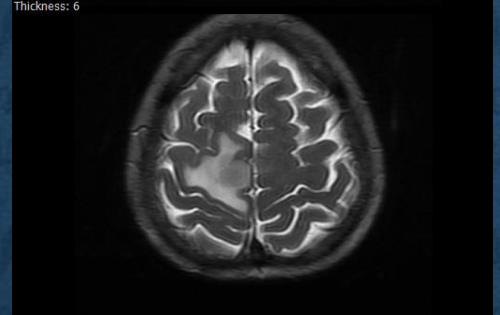
Thickness: 6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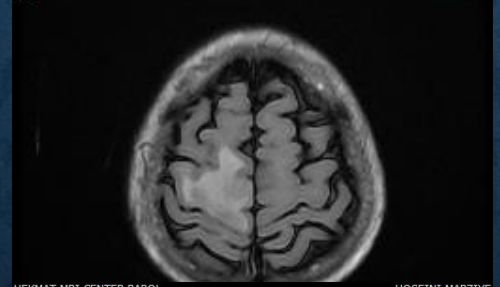
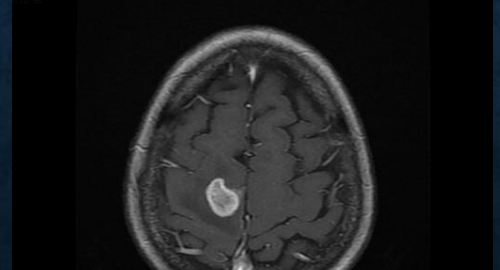
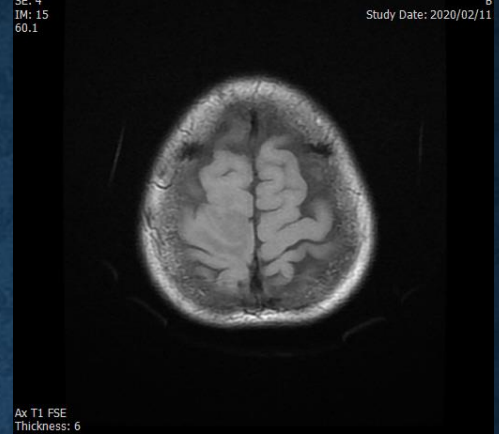

HEKMAT MRI CENTER BABOL
HOSEINI MARZIYE
Signa HDxt
AGE: 75Y
6379
F 154898
SE: 4
L
IM: 18
Study Date: 2020/02/13
49.3

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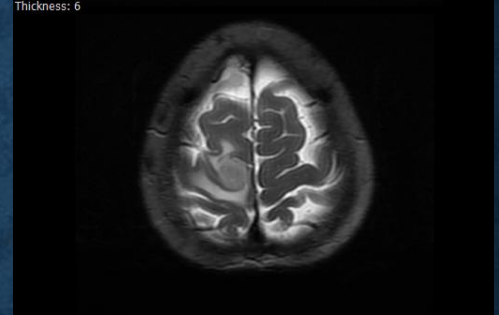
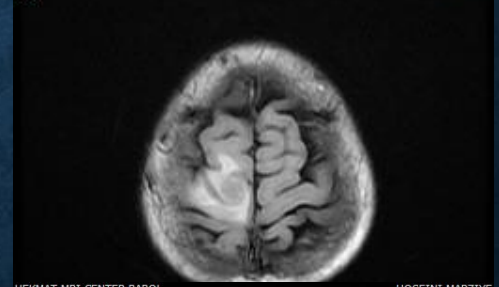
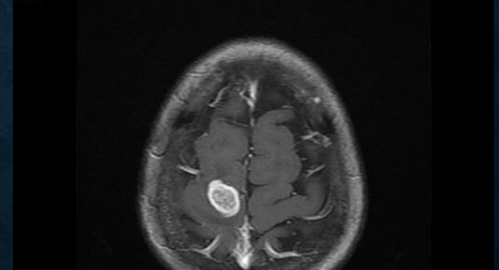
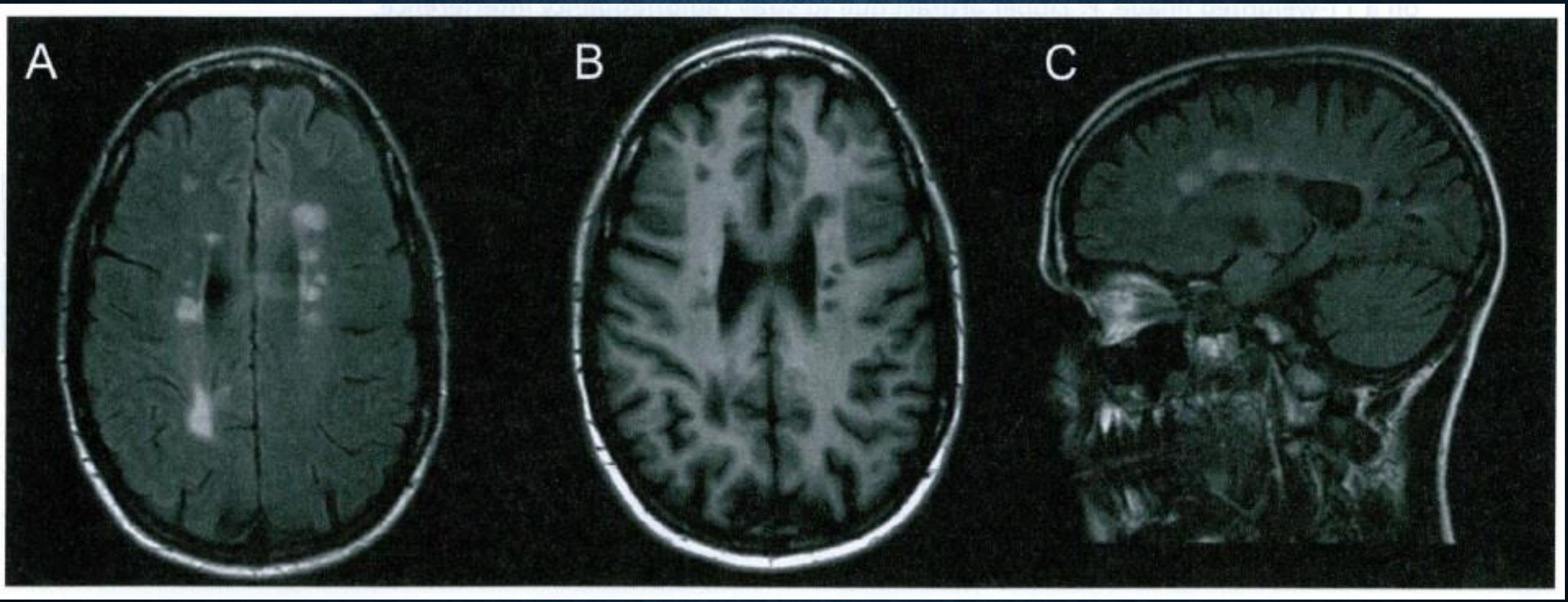
Thickness: 6


Ax FSE T2
Thickness: 6


HEKMAT MRI CENTER BABOL
HOSEINI MARZIYE
Signa HDxt
AGE: 75Y
6379
F 154898
SE: 4
L
IM: 19
Study Date: 2020/02/13
55.2

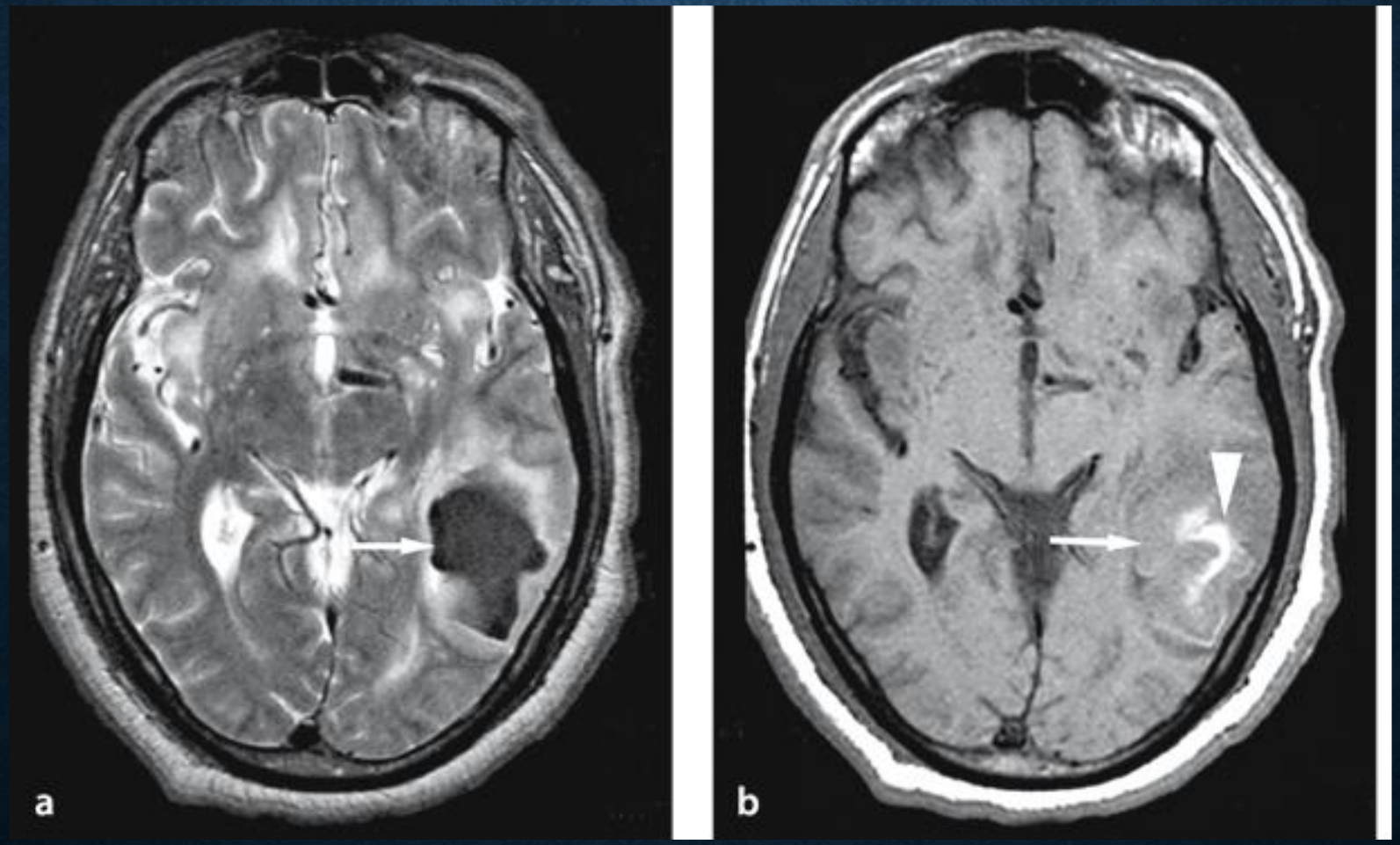



A
B
C

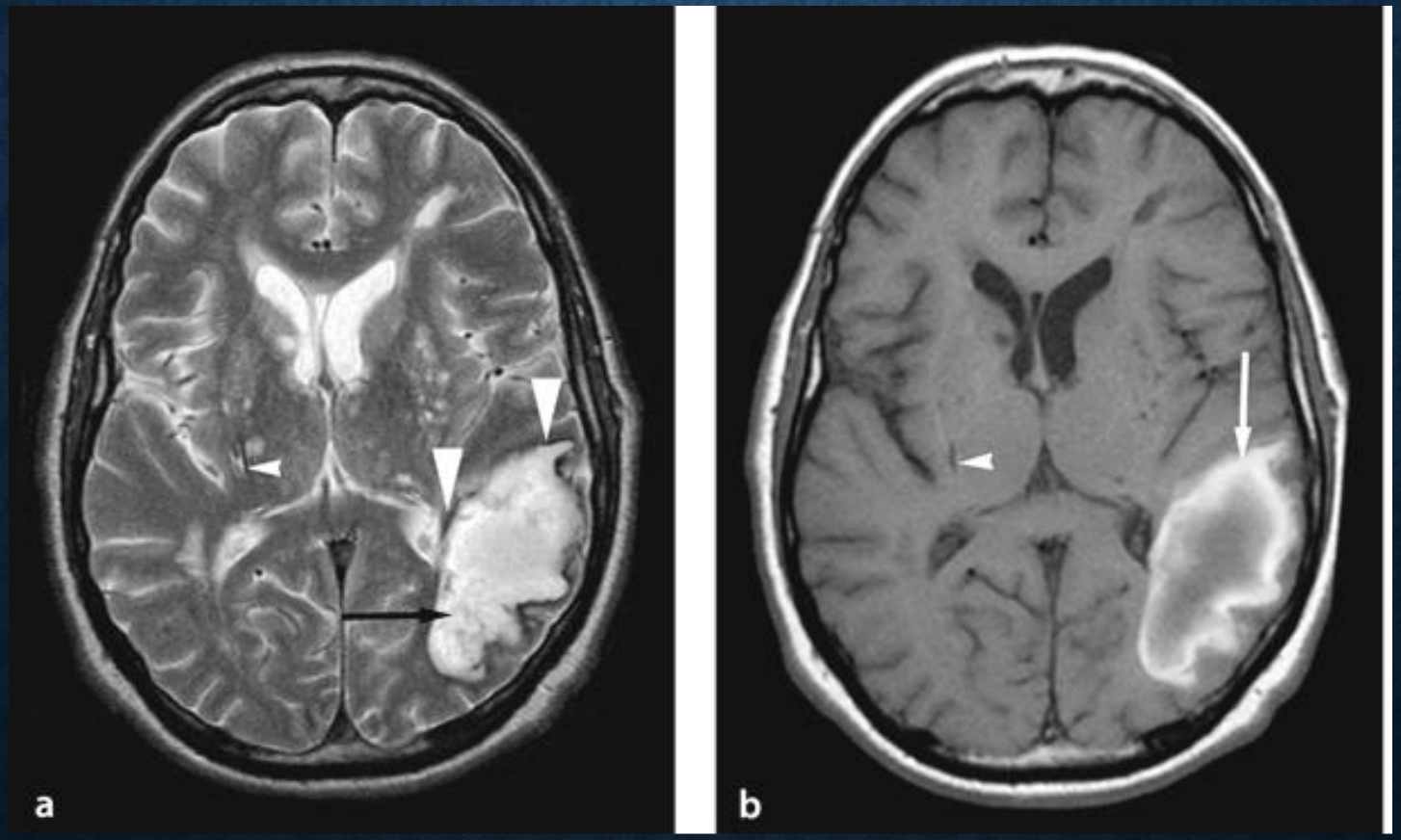
BLOOD IN MRI

Intraparenchymal Hematoma (IPH or ICH)			
	T_1	T_2	
Hyper-acute Within first few hours	Isointense	Hyperintense	اکسی هموگلوبین (اینتراسلولار)
Acute Several hours to ≈2 days	Isointense Hypointense	Hypointense	داکسی هموگلوبین (اینتراسلولار)
Early Sub-acute 2 days to 1 week	Hyperintense	Hypointense	مت‌هموگلوبین (اینتراسلولار)
Late Sub-acute 1 to a few weeks	Hyperintense	Hyperintense	مت‌هموگلوبین (اکستراسلولار)
Chronic ≥1 month	Isointense Hypointense	Hypointense	فریتین و هموسیدرین (اکستراسلولار)

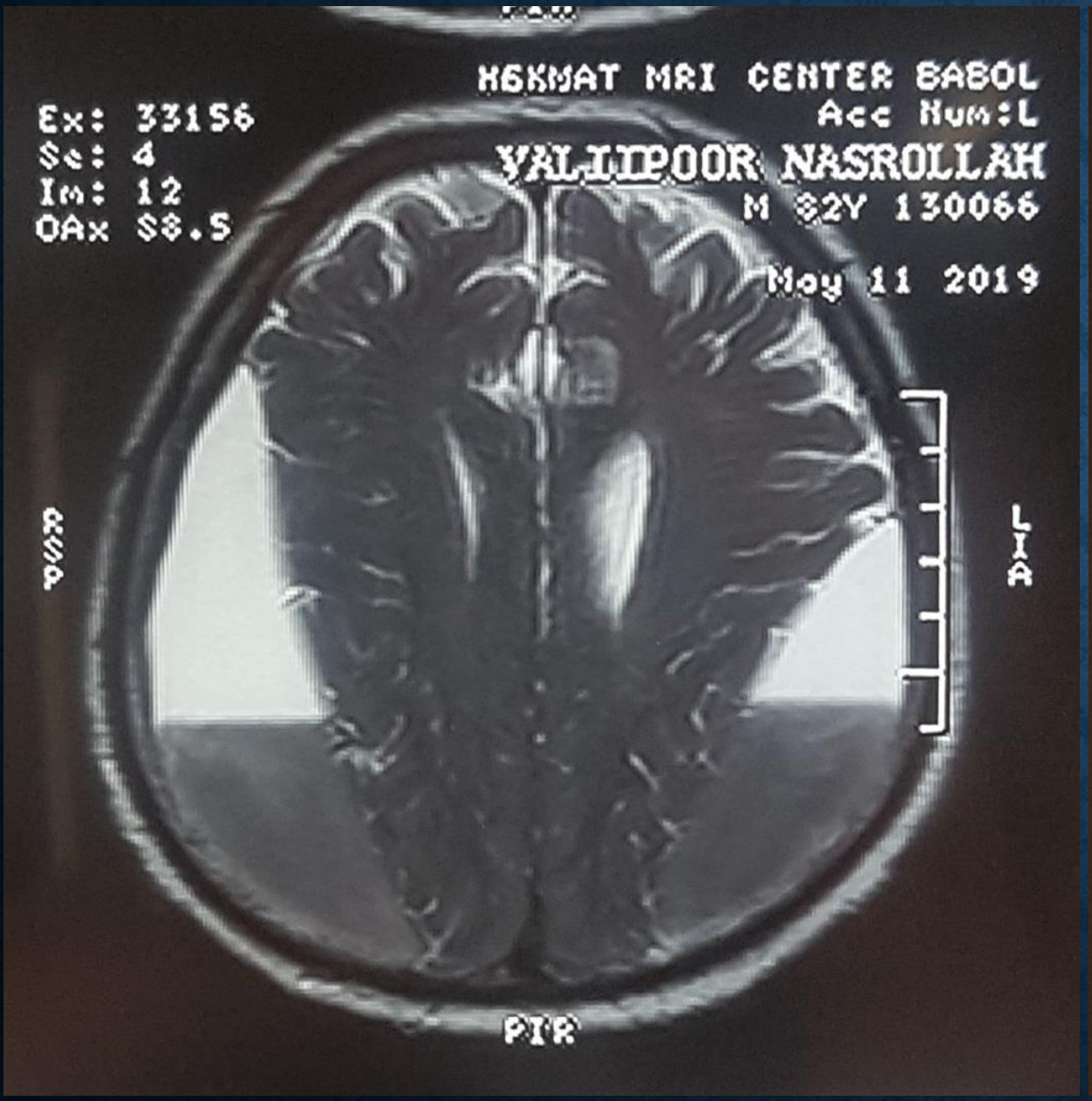
ACUTE TO EARLY SUBACUTE HEMORRHAGE

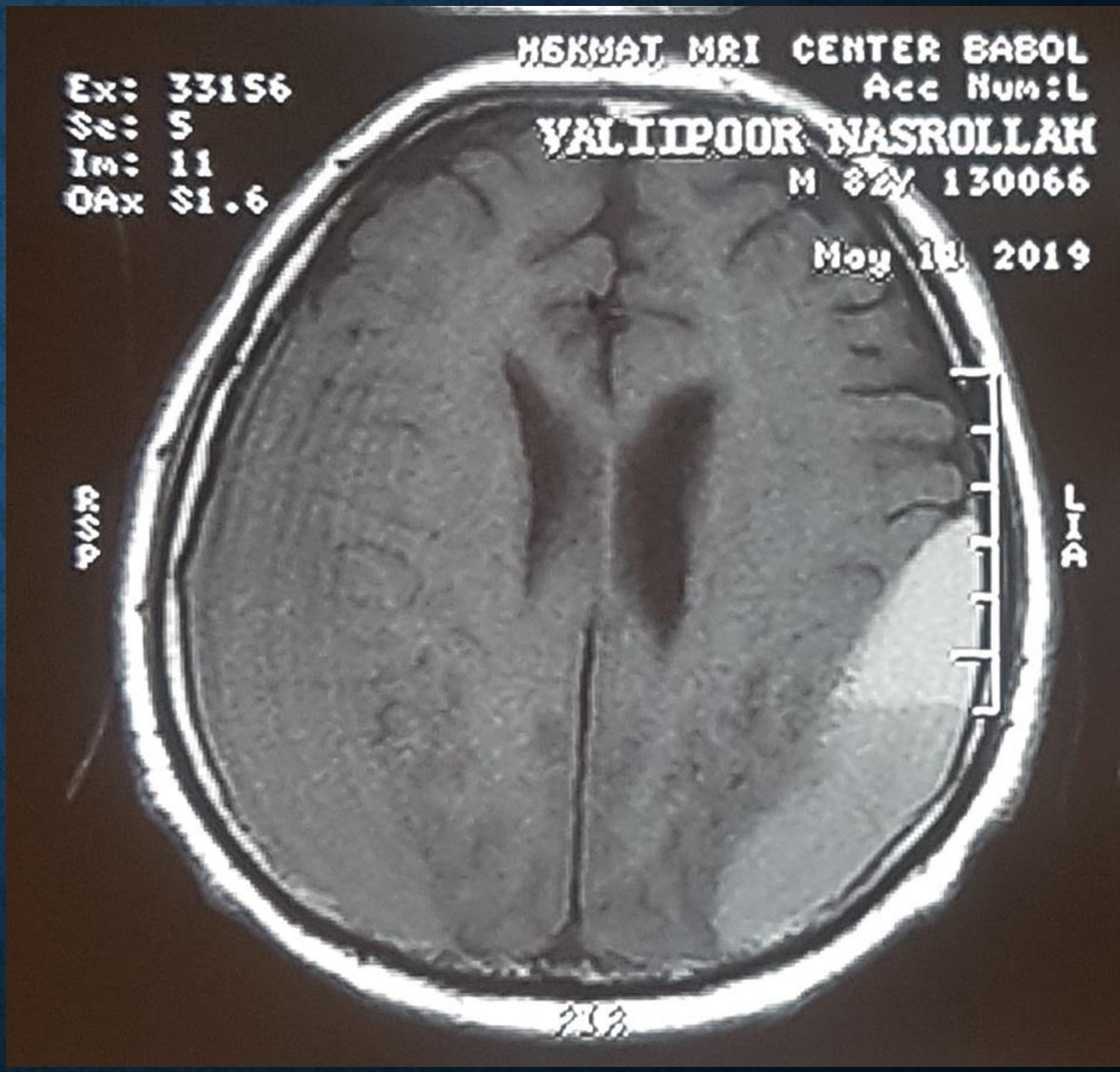


LATE SUBACUTE HEMORRHAGE



SDH and EDH			
	T_1	T_2	
Acute	Isointense Hypointense	Hypointense	داکسی هموگلوبین (اینتراسلولار)
Sub-acute	Hyperintense	Hyperintense	مت‌هموگلوبین (اکستراسلولار)
Chronic	Hypointense	Hyperintense	فریتین و هموسیدرین (اکستراسلولار)


HEKMAT MRI CENTER BABOL
Acc Num:L
VALIPOOR NASROLLAH
M 82Y 130066
May 11 2019
Ex: 33156
Se: 4
Im: 12
OAx 88.5
RSP
LIA
PIR


HEKMAT MRI CENTER BABOL
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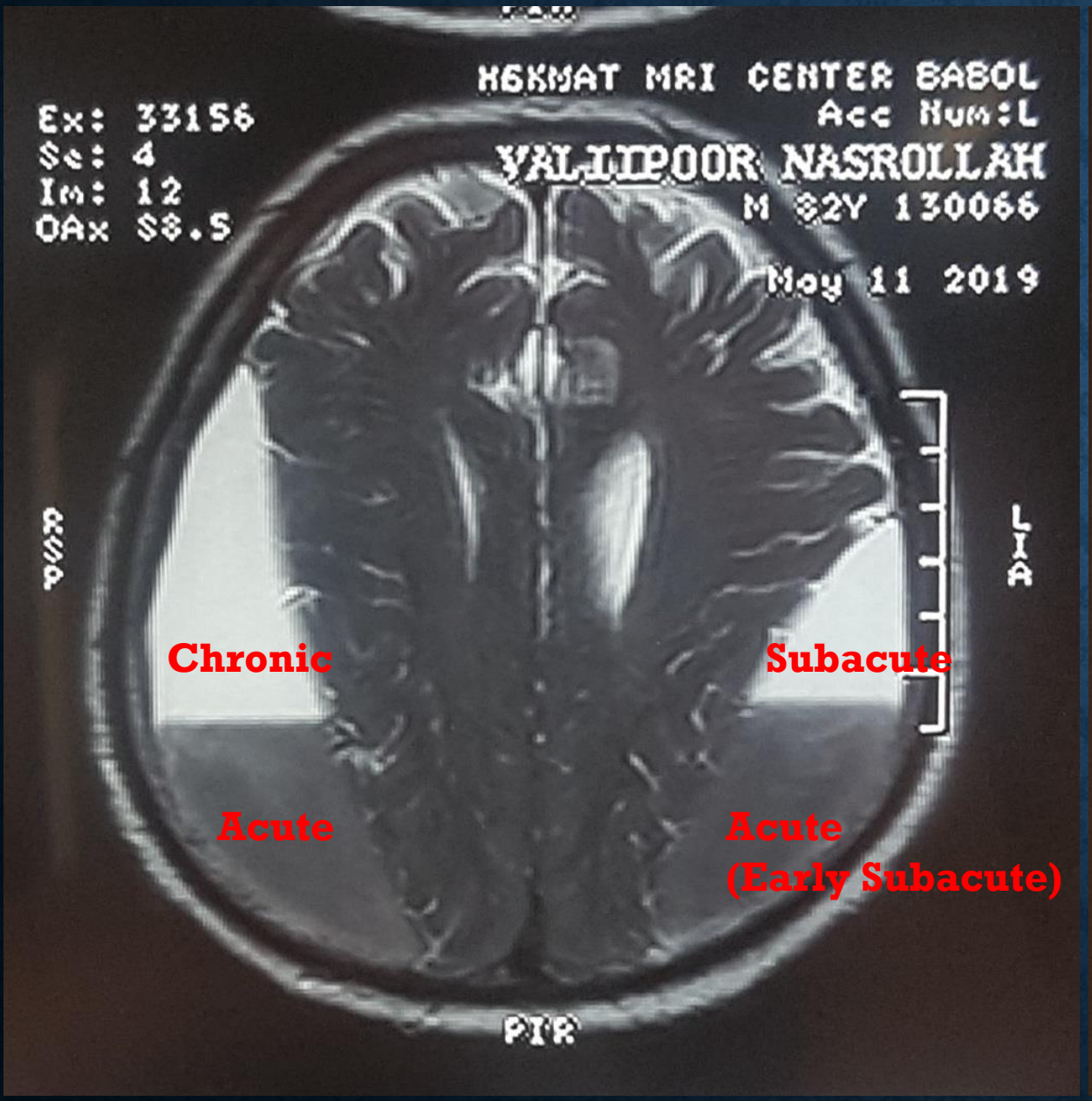
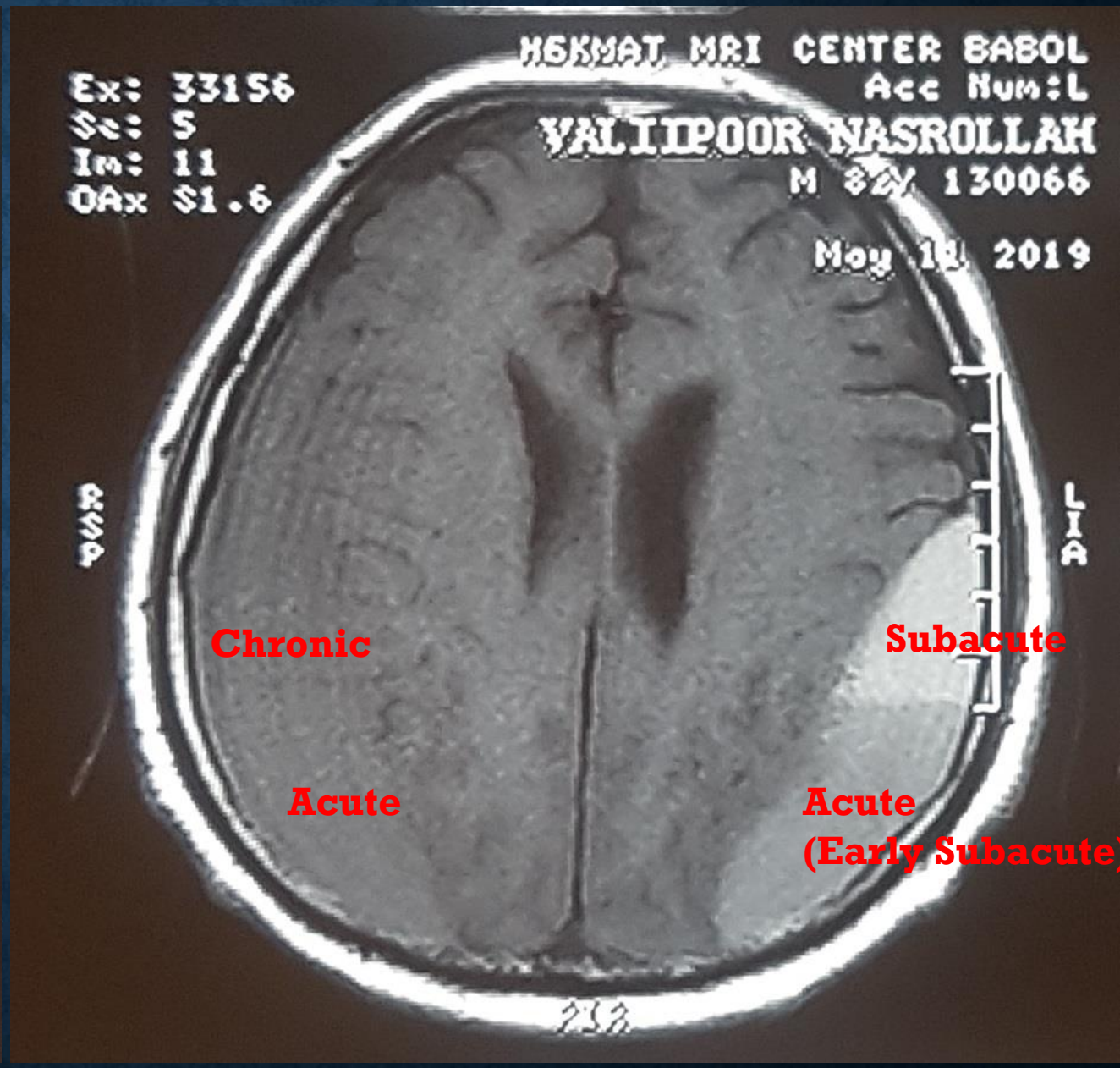
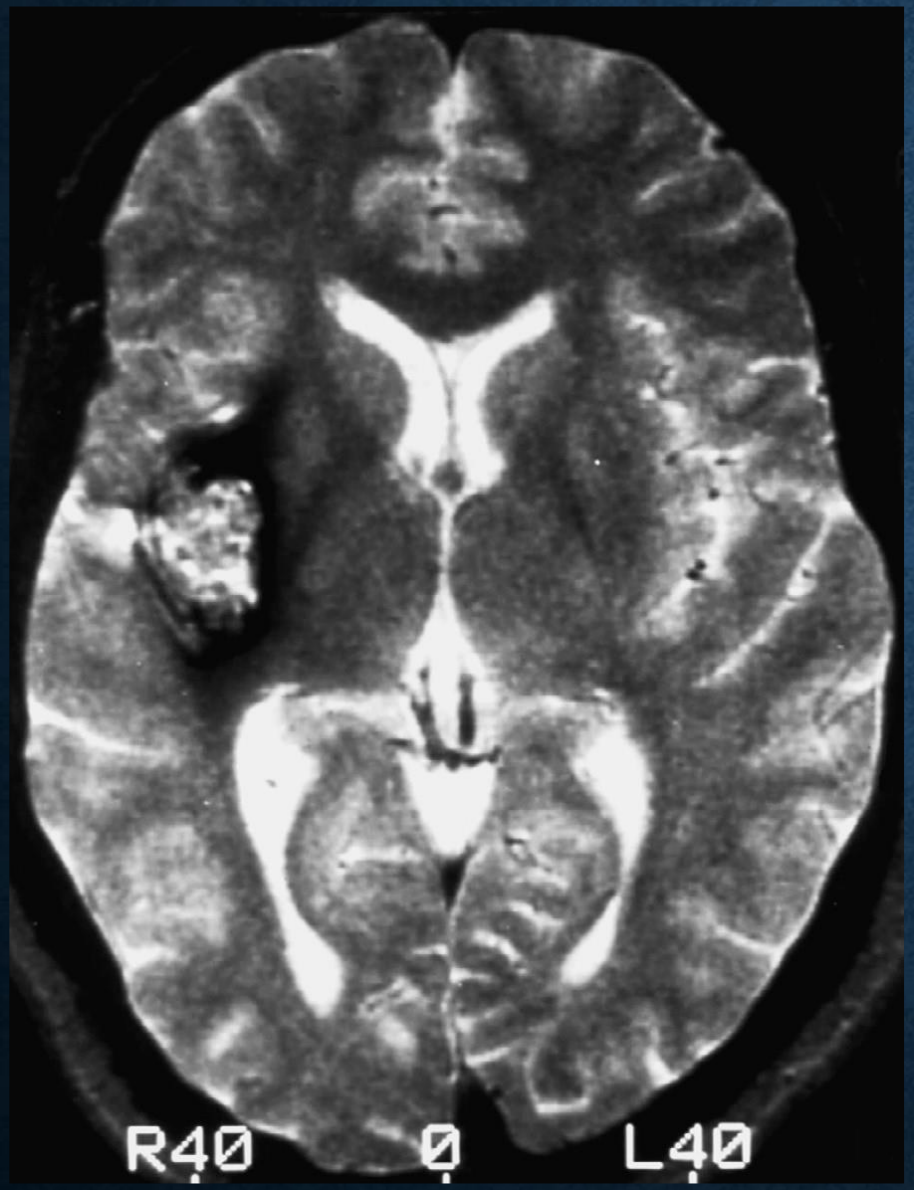
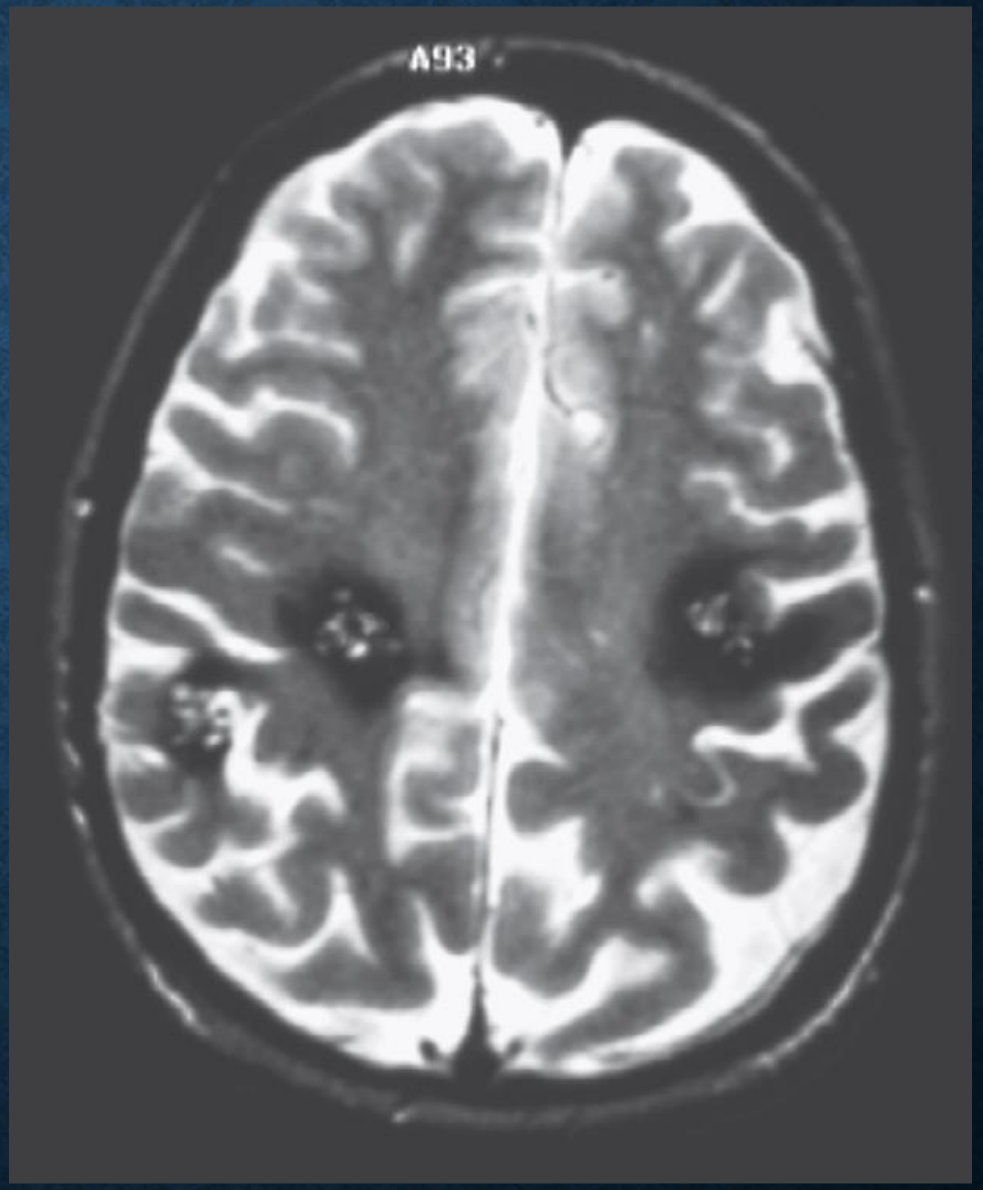
M 82Y 130066
May 11 2019
Ex: 33156
Se: 5
Im: 11
OAx 51.6
RSP
LIA

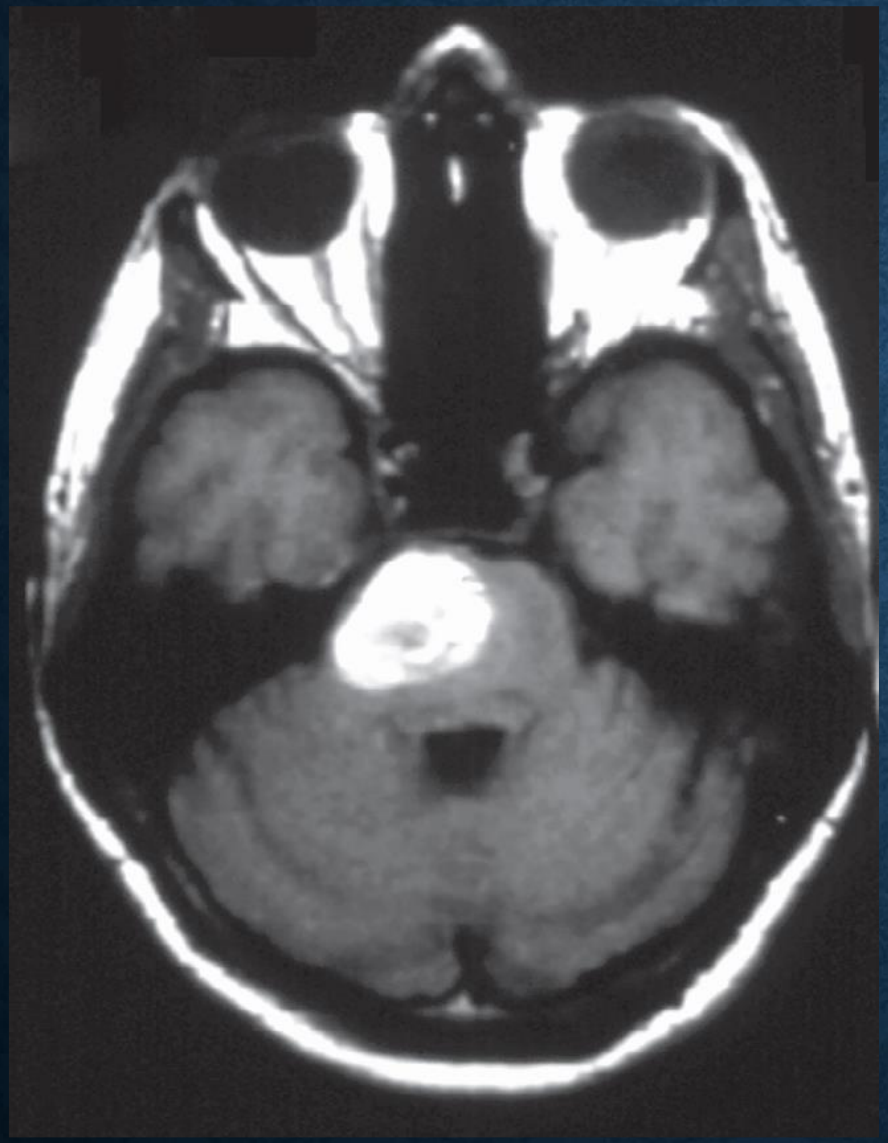

HEKMAT MRI CENTER BABOL
Acc Num:L
VALIPOOR NASROLLAH
M 82Y 130066
May 11 2019
Ex: 33156
Se: 4
Im: 12
OAx 88.5
RSP
LIA
PIR
Chronic
Subacute
Acute
Acute
(Early Subacute)

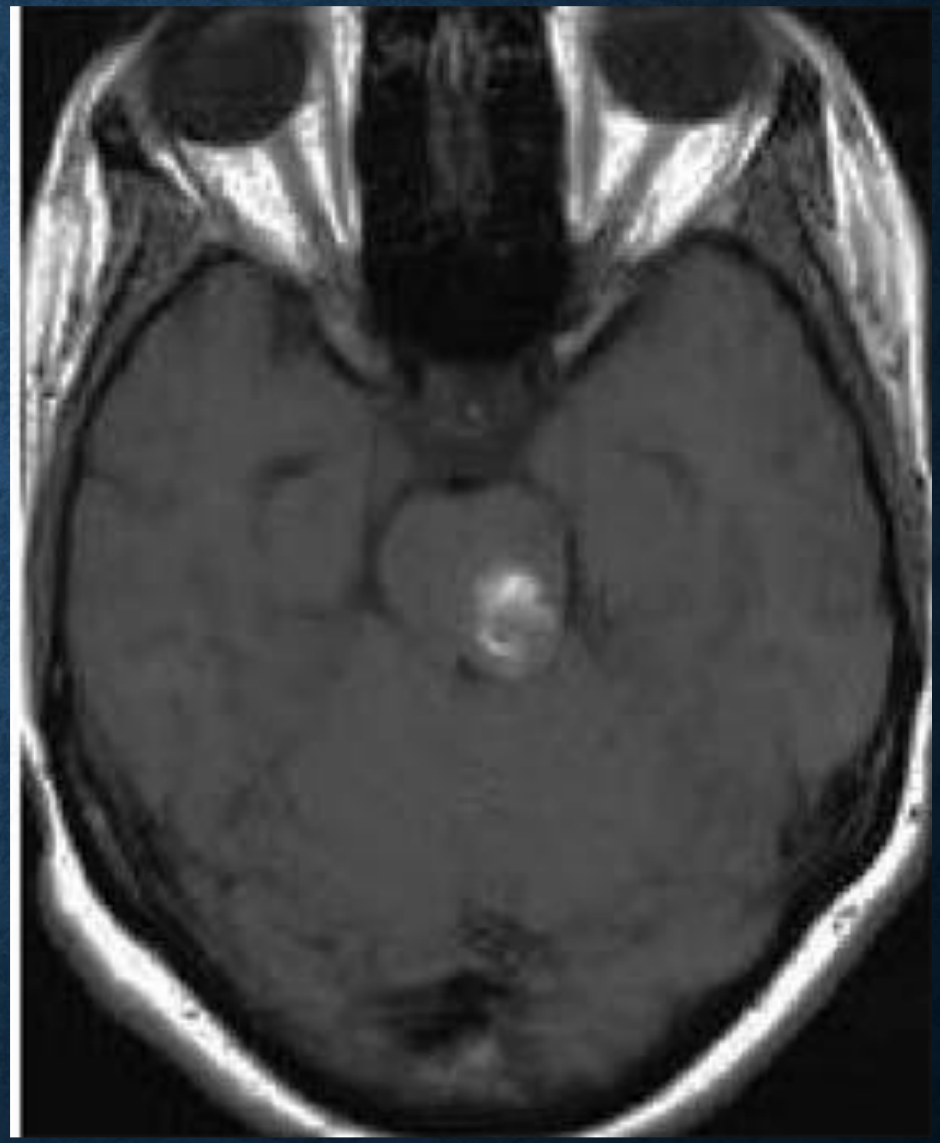

HEKMAT MRI CENTER BABOL
Acc Num:L
VALIPOOR NASROLLAH
M 82Y 130066
May 11 2019
Ex: 33156
Se: 5
Im: 11
OAx 81.6
RSP
LIA
Chronic
Subacute
Acute
Acute
(Early Subacute)

CAVERNOMA


R40
0
L40


A93





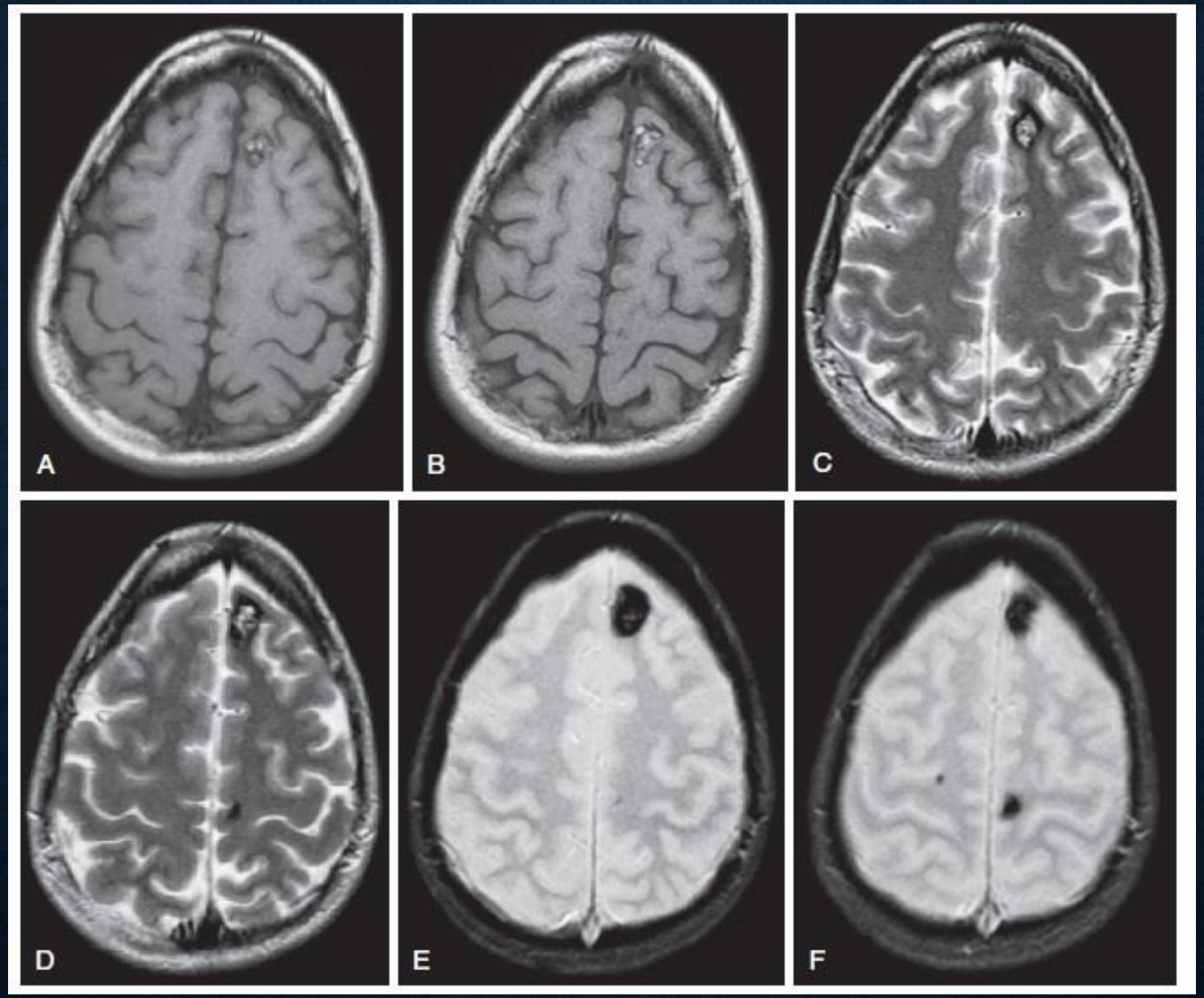

A
B
C
D
E
F

TABLE 409-2 Magnetic Resonance (MR) Imaging Classification for Cavernous Malformation

Lesion Type	MR Signal Characteristic	Pathologic Characteristics	Natural History: Risk of Hemorrhage
Type IA	T1: hyperintense focus of hemorrhage T2: hyperintense or hypointense focus of hemorrhage extending through at least one wall of the hypointense rim that surrounds the lesion (see Figs. 409-3 and 409-4); focal edema* may be present (see Fig. 409-8)	"Overt" extralesional focus of hemorrhage extending outside the lesion capsule.	Almost all lesions are symptomatic. High risk of recurrent symptomatic hemorrhage of up to 60%/yr for brainstem lesions (mean, 25%-30%/yr).
Type IB	T1: hyperintense focus of hemorrhage T2: hyperintense or hypointense focus of hemorrhage surrounded by a hypointense rim (see Fig. 409-5)	Subacute focus of intralesional hemorrhage.	Risk of symptomatic hemorrhage related to presentation and location. Higher for symptomatic lesions in the brainstem and basal ganglia (5%-10%/yr). Lower in asymptomatic lesions (0.5%-1%/yr).
Type II	T1: reticulated mixed signal core T2: reticulated mixed signal core surrounded by a hypointense rim (see Figs. 409-1 and 409-2, *straight arrows*)	Loculated areas of hemorrhage and thrombosis of varying age surrounded by gliotic, hemosiderin-stained brain; in large lesions, areas of calcification may be seen.	Risk of symptomatic hemorrhage related to presentation. In symptomatic patients risk of recurrent hemorrhage 4%-5%/yr. Low risk for asymptomatic lesions (0.5%-1%/yr).
Type III	T1: isointense or hypointense T2: hypointense with hypointense rim that magnifies the size of lesion GE: hypointense with greater magnification than T2 (see Fig. 409-9)	Chronic resolved hemorrhage with hemosiderin staining within and around the lesion.	Rarely symptomatic. Lesions have a low risk of hemorrhage (<0.5%/yr).
Type IV	T1: poorly seen or not visualized at all T2: poorly seen or not visualized at all GE: punctate hypointense lesions (see Fig. 409-9)	Two lesions in the category have been pathologically documented to be telangiectasias.	Never symptomatic. Very low risk of hemorrhage.