RADIOLOGY OF BRAIN & SPINAL TUMORS--- INTERPRETATION OF CECT/MRI CONTRAST IMAGES

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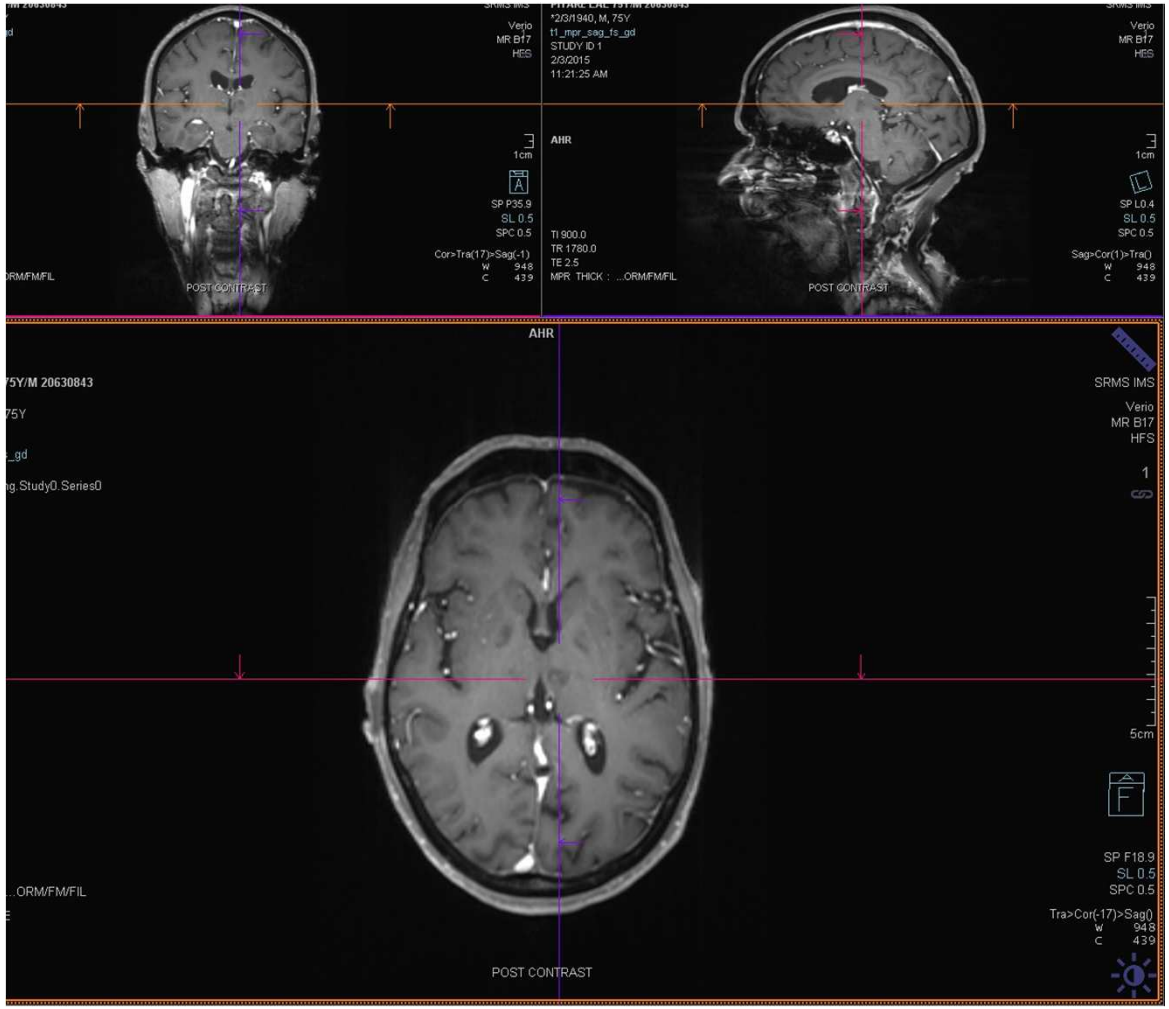
BRAIN

1. POST CONTRAST ISOTROPIC IMAGES
2. IMPORTANCE OF BASIC SEQUENCES IN MRI
3. WHY WE DO CONTRAST.
4. SOLID Vs CYSTIC, ABSCESS Vs TUMOR NECROSIS
5. CONTRAINDICATION OF CONTRAST.
6. INTRA AXIAL Vs EXTRAAXIAL
7. NORMAL Vs ABNORMAL MENINGEAL ENHANCEMENT.
8. DIFFUSION CONTRAST ROLE
9. INTERPRETATION OF CT & MRI CONTRAST IMAGES OF BRAIN & SPINE.
10. WHAT IS BEYOND CONTRAST IMAGES
11. INTERPRETATION OF POST OP/ POST RT IMAGES
12. SUMMARIZE

SPINE

1. CLASSIFICATION OF LESIONS.
2. COMMON TUMOURS

POST CONTRAST ISOTROPIC MRI IMAGES



The sequences after contrast administration should include, if possible, a volumetric sequence (e.g., three-dimensional gradient-recalled echo) to allow for reconstruction in different planes and volumetric assessment of the lesions

3D Sequences with 01 mm or <01 mm slices. The term "isometric“ for "equal measure", reflecting that the scale along each axis of the projection is the same. It Can be taken on multislice CT also

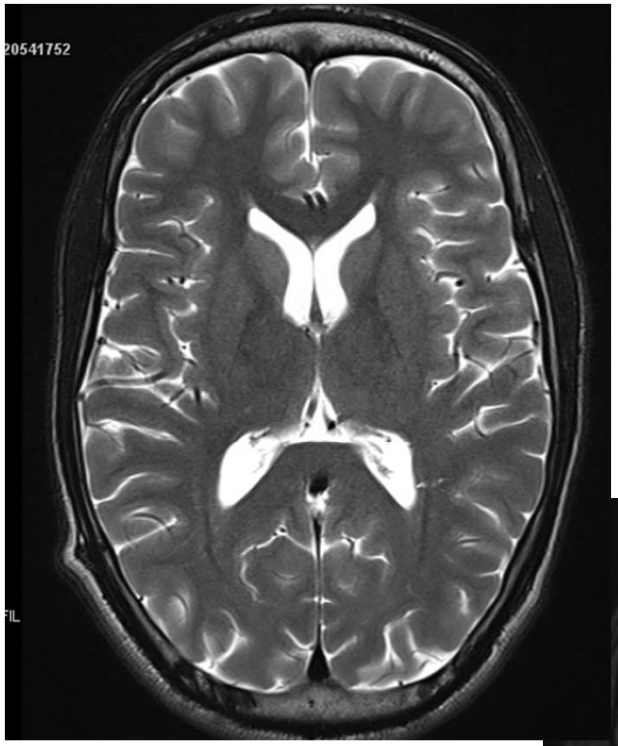
BASIC MRI SEQUENCES

First question arises ,Is there a lesion?. This question can be easily addressed by using an appropriate brain tumor imaging Protocol in MRI (T1 SE, T2 FSE, FLAIR -Tra/Cor, post contrast Isotropic T1 3D GRE .

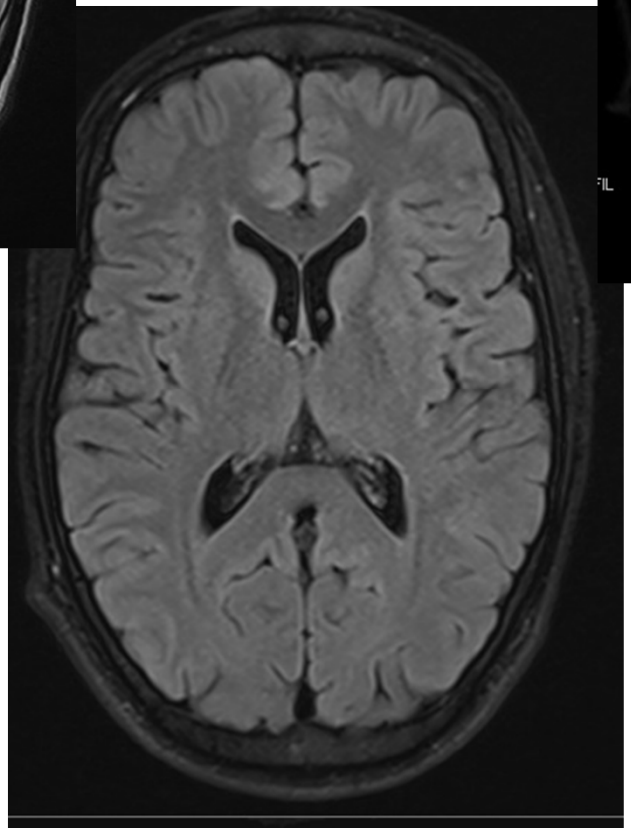
Alternative Sequences are DWI/DTI, PWI, MRS & T1 Dynamic MRI

NOTE:(*3D GRE,* Three-dimensional gradient-recalled echo; *Cor,* coronal; *FLAIR,* fluid-attenuated inversion recovery; *FSE,* fast spin echo; *SE,* spin echo; *Tra,*transverse.*DWI,* diffusion-weighted imaging, *DTI,* diffusion tensor imaging; *PWI,* perfusion-weighted imaging; *MRS,* magnetic resonance spectroscopy)

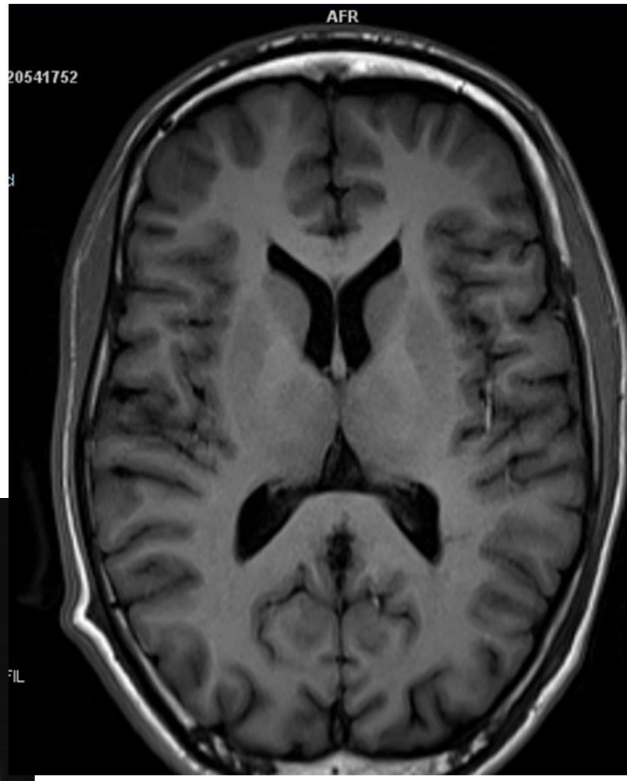
Basic sequences
Axial scans



T2WT



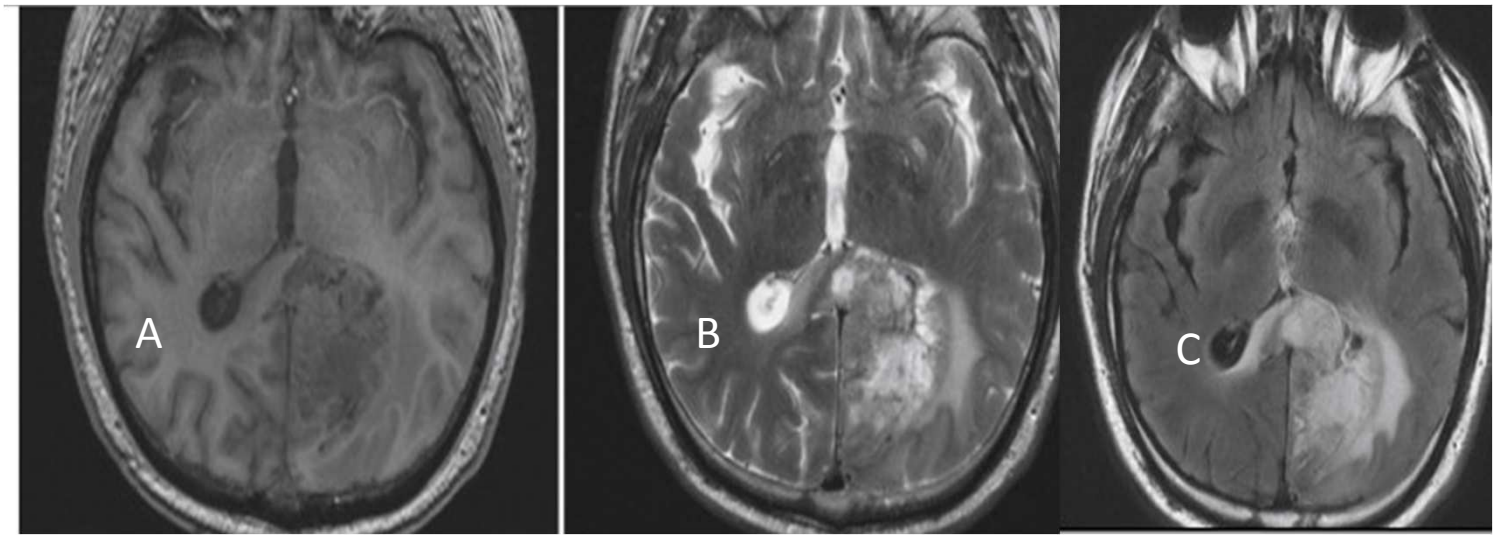
FLAIR



T1WI

IMPORTANCE OF BASIC MRI SEQUENCES..cont

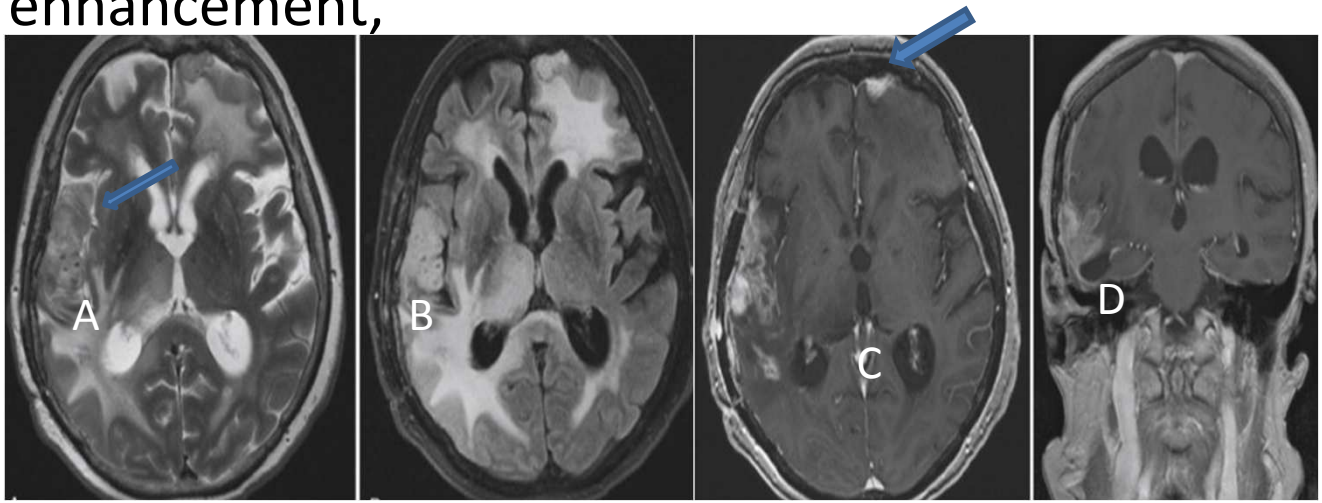
1. Diagnosis is made on axial images. Most of the time lesion is identified on T2/Flair. 2.T2Wt- and FLAIR are used to display the margins of a tumor and its surrounding edema or a direct tumor infiltration.
2. The sagittal & coronal images are used to confirm exact location
3. T1wt images are for anatomy details .
4. All post contrast study are T1wt, preferably with fat suppressed images, as fat & contrast are both bright in post contrast study



Magnetic resonance imaging of a patient with anaplastic (grade III) astrocytomas. On unenhanced T1-weighted **(A)** and T2-weighted **(B)**images, one can speculate about an infiltration of the corpus callosum. However, the best visualization of the infiltration can be achieved by using FLAIR acquisition(C)

WHY WE DO CONTRAST?.

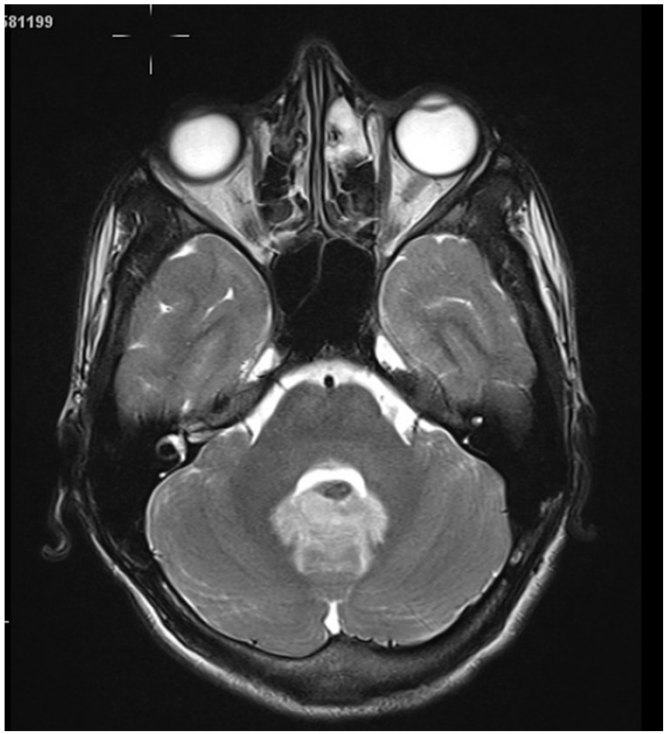
Contrast enhancement studies are mandatory in the assessment of patients with cerebral tumors. It helps in distinguishing tumors from other pathologic processes and enables optimal characterization of tumor, Delineate extent of tumor ,response to therapy, such as change in size, morphology, and degree of contrast material enhancement,

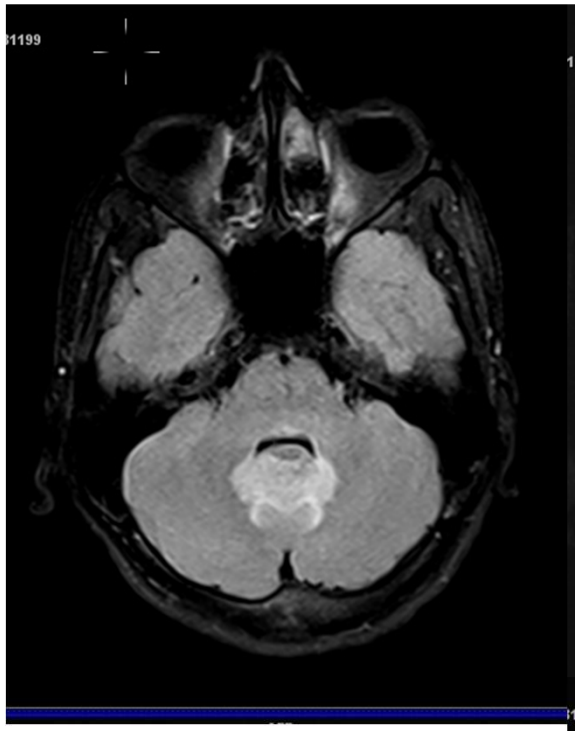


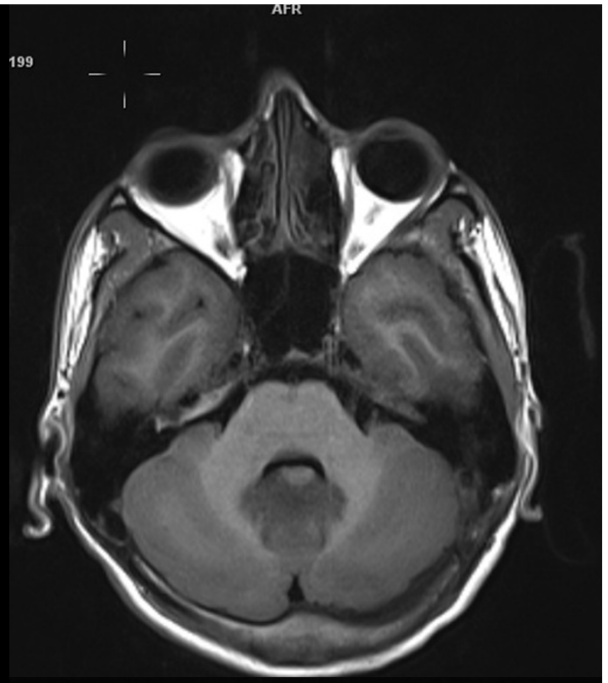
Contrast-enhanced MRI of with recurrent malignant glioma (WHO grade IV).

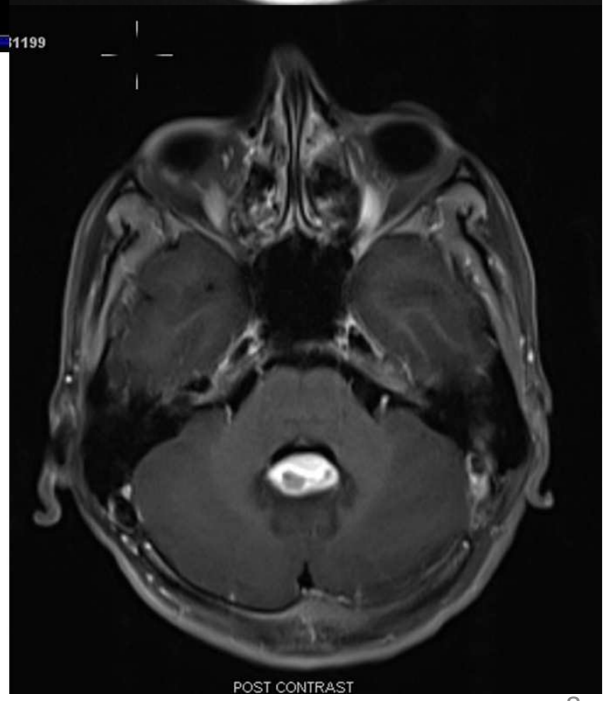
T2-weighted **(A)** and FLAIR **(B)** images showing a large area of T2 hyper intensity with small foci of lower signal representing the high cellular density tumor areas.

C, D: These tumor areas show pronounced contrast media enhancement. The frontal lesion can be considered tumor spread via the meninges.



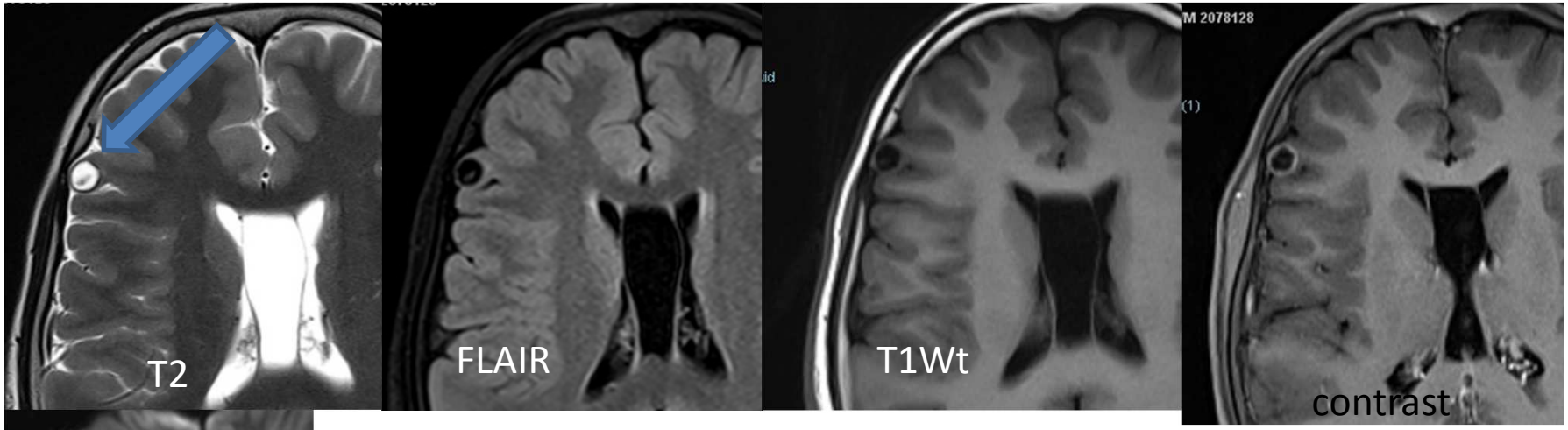


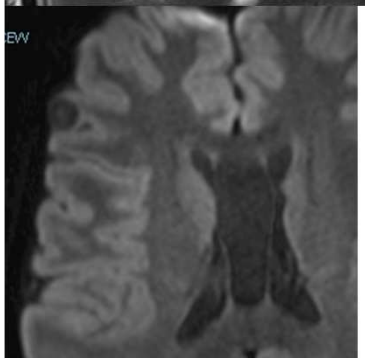




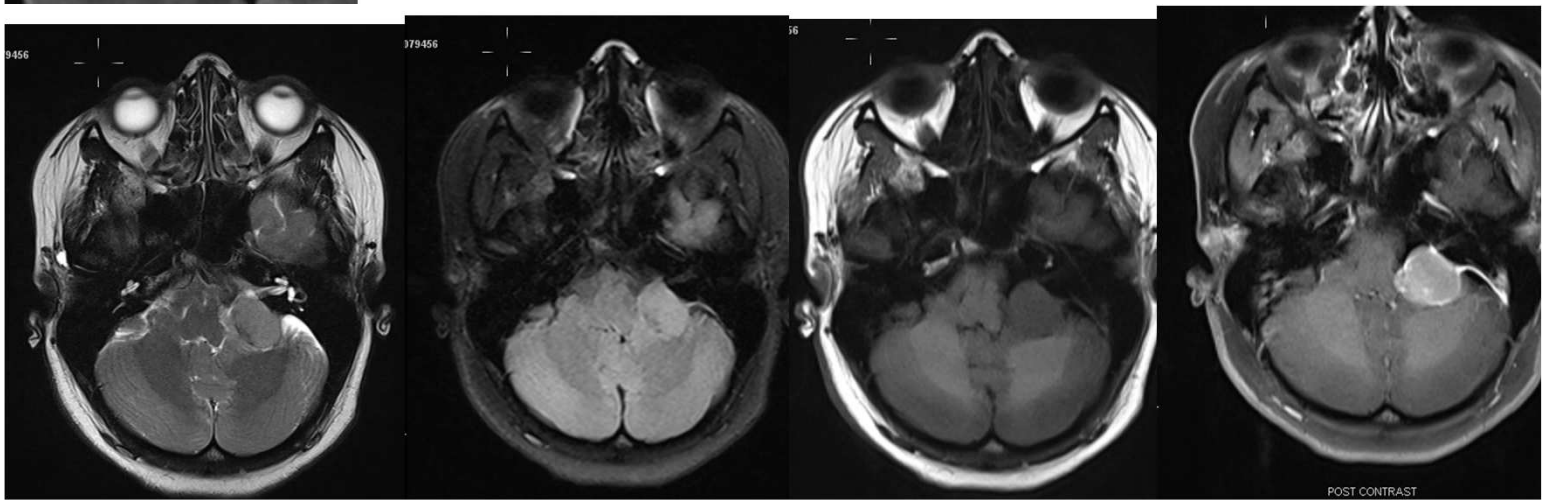
T2,FLAIR& T1WT PLAIN STUDY WERE NOT ABLE TO DILINEATE THE LESIONSPOST CONTRAST STUDY DELINEATE LESIONS FROM EDEMA

IDENTIFICATION OF CYSTIC & SOLID LESIONS



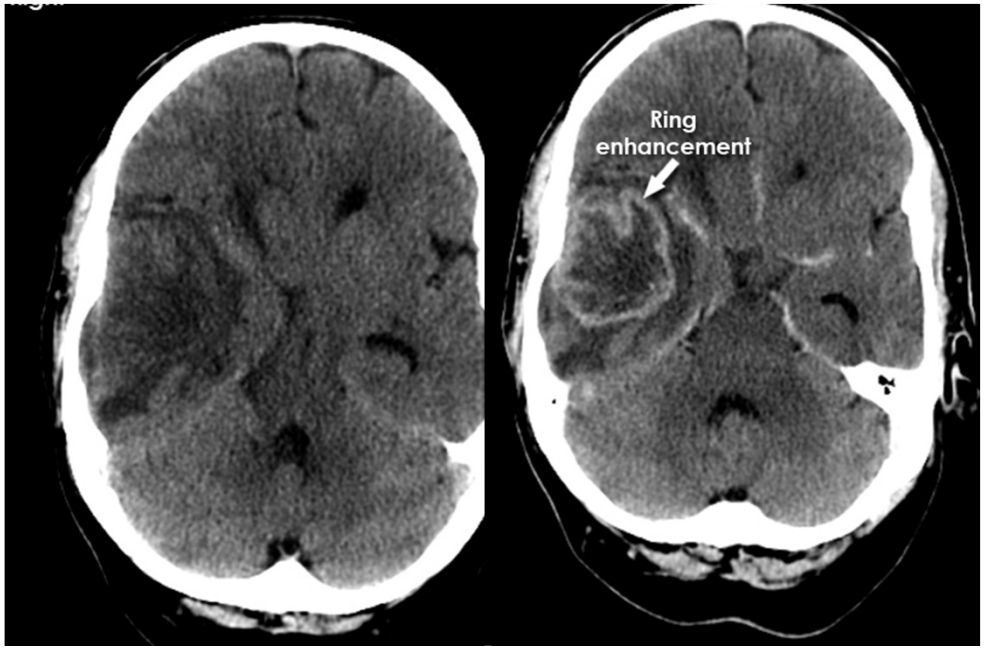


A cystic lesion will appear of CSF signal intensity on all sequences
Unless it has more protenicious components. Post contrast
With or without peripheral enhancement.

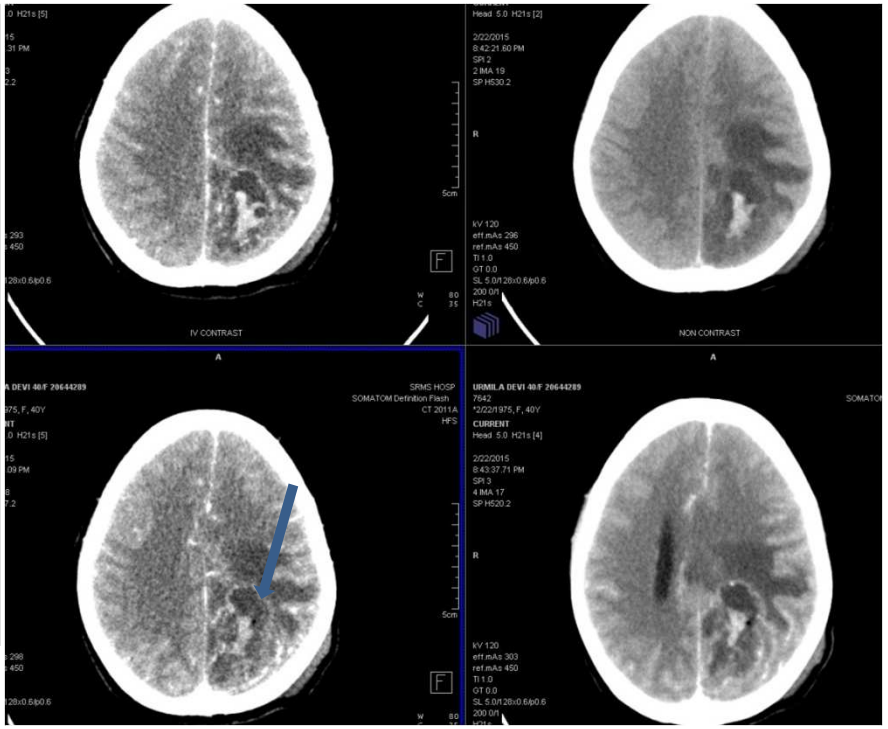


Solid sol
Hypo/hyper
On T2/flair
Enhancement
Of\solid
component

TUMOR NECROSIS VS ABSCESS

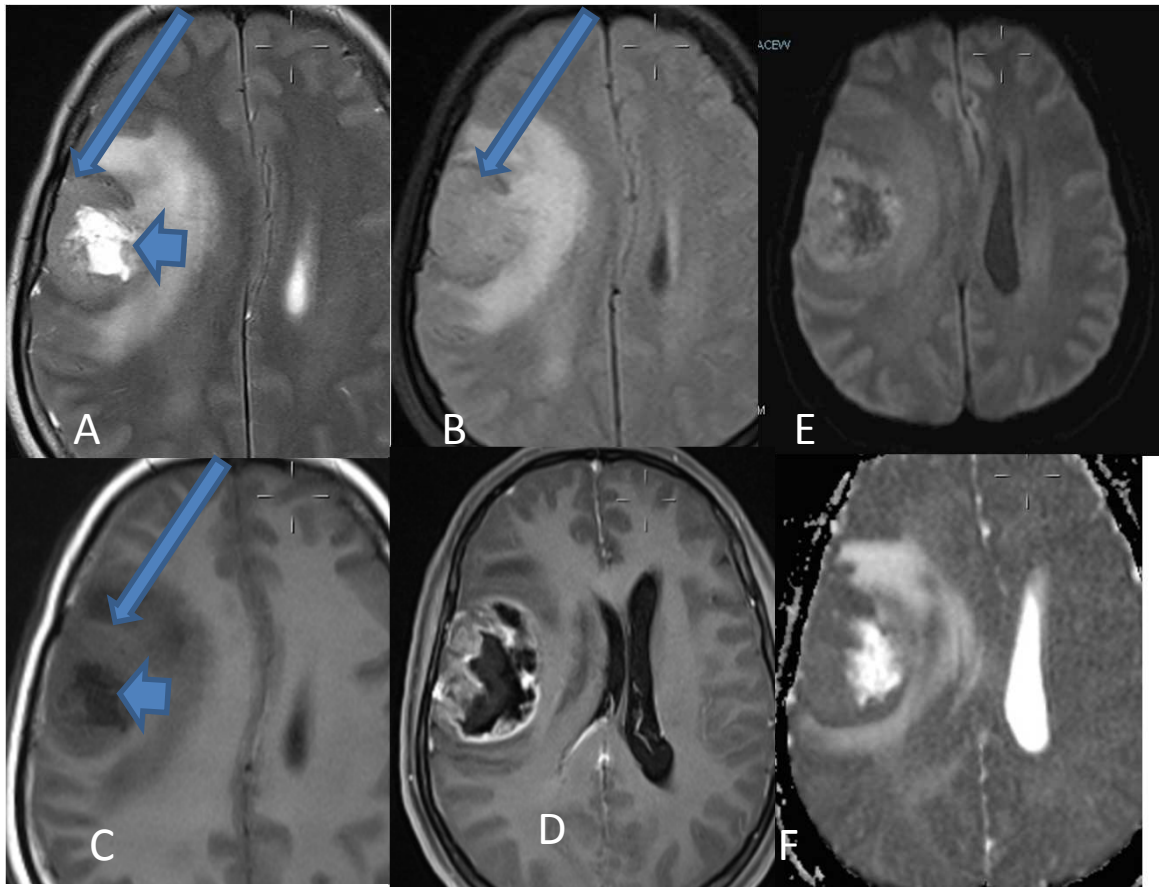


On CECT at time it is difficult to Differentiate Between Tumor necrosis Vs Abscess



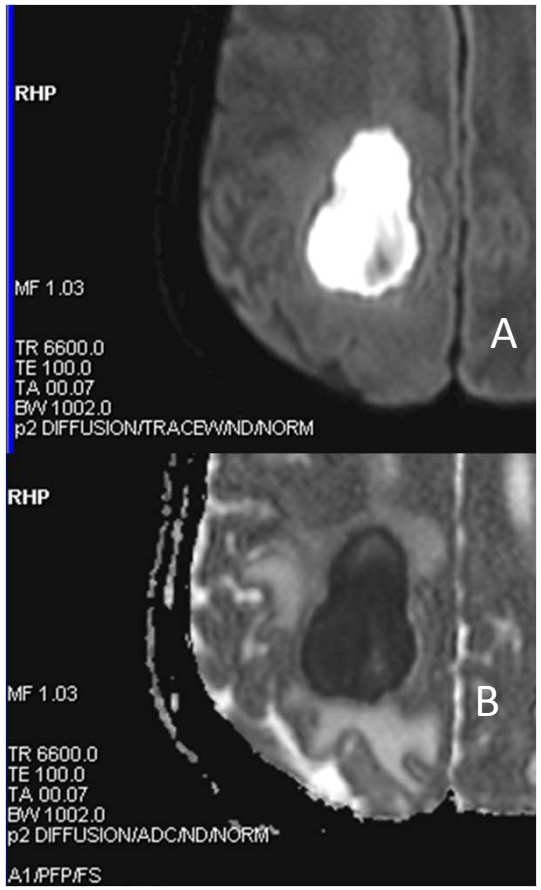
Typical tumor enhancement as irregular Peripheral margin with a large solid Enhancing component and no enhancement Of necrotic hypo dense Component(Arrow)

HOW TO IDENTIFY TUMOR NECROSIS FROM ABSCESS

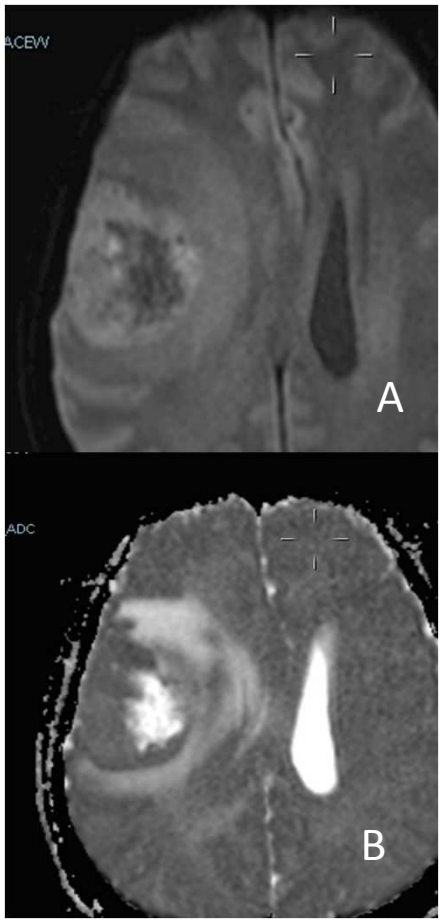


A is T2W, B is flair, C is T1Wt, D is T1wt post contrast, E is Diffusion B1000 & F is ADC. Solid component of the tumor is hypointense on T2& Flair, isointense on T1 (long arrow)and necrotic Component (short arrow)is hyperintense on T2Wt/Flair and hypointense on T1. Post contrast shows enhancement of solid component. Diffusion shows no restriction (bright)in necrotic component of tumor and ADC shows high signal in it..suggest necrosis

HOW TO IDENTIFY TUMOR NECROSIS FROM ABSCESS



ABSCESS.. Diffusion(A) restriction(bright)
ADC(B)-Low.
Sensivity & Specificity > 90%



TUMOR NECROSIS—Diffusion no restriction
ADC-HIGH

Diffusion B1000 shows restriction in abscess making it bright with low ADC. No restriction in necrotic component of tumor and ADC shows high signal in it..suggest Tumor necrosis

CONTRAST CONTRAINDICATION

CT –Non-ionic /ionic iodine contrast

MRI -Gadolinium-based contrast

1.Gadolinium-based contrast not recommended in pregnancy unless benefit justifies risk (crosses the placenta).

2.Both contrast unsafe with renal parenchyma disease.

a. The eGFR threshold below which withholding contrast should be considered is between 60 and 30 ml/min/1.732m^2.

b. The rate of contrast nephropathy in patients with a GFR > 40 ml/min was 0.6% and < 40 ml/min/1.732 was 4.6%,

c. Thus a threshold of 45 ml/min/1.732 seems an appropriate cut-off.

d. A creatinine of 1.6 roughly corresponds to an estimated GFR of approximately 45 ml/min/1.732.

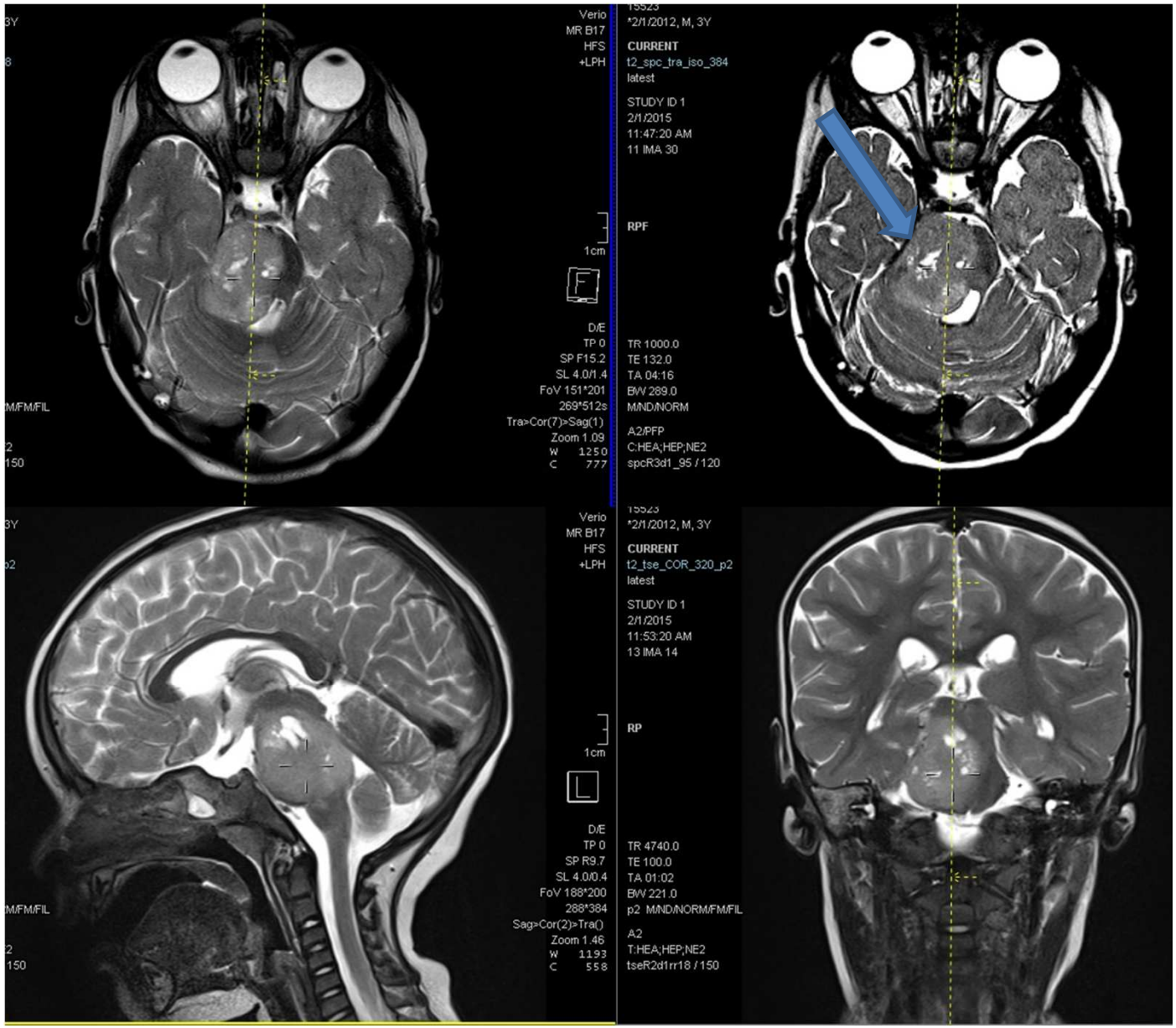
MRI Unsafe for pacemaker, cochlear implant & nerve stimulators

BRAIN: EXTRAAXIAL Vs INTRAAXIAL MASSES

MRI is better than CT FOR peripheral brain tumors for accurate localization.

The findings most often seen in extra axial lesions include interruption of bone, white matter buckling, **widening of adjacent subarachnoid space or cistern,** and medial displacement of pial arteries or veins.

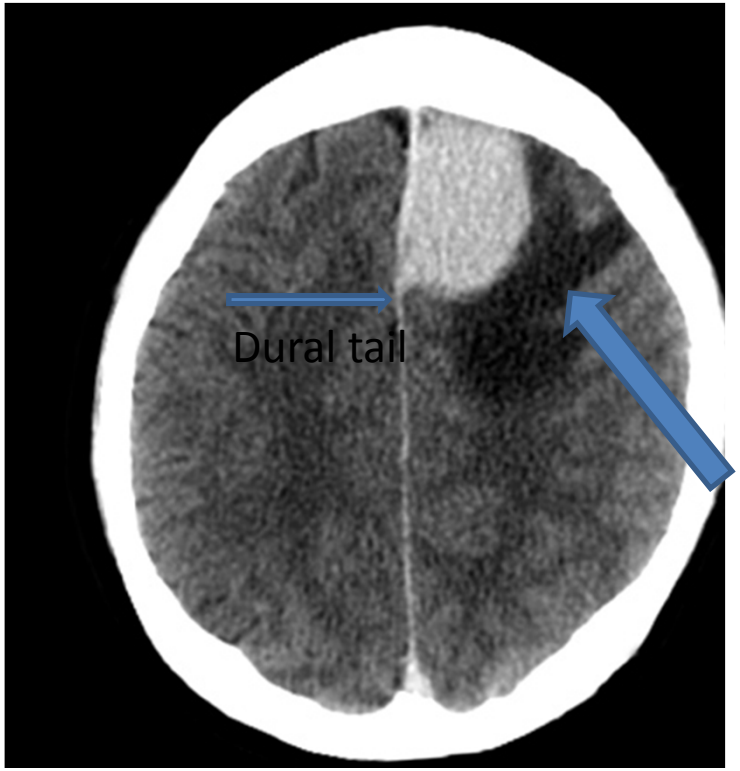
Invasion of the cortex without white matter buckling, as well as flattening and lateral displacement of the surface veins, **obliteration of cisterns** was most often seen in intraaxial lesions.



3 YRS OLD BOY

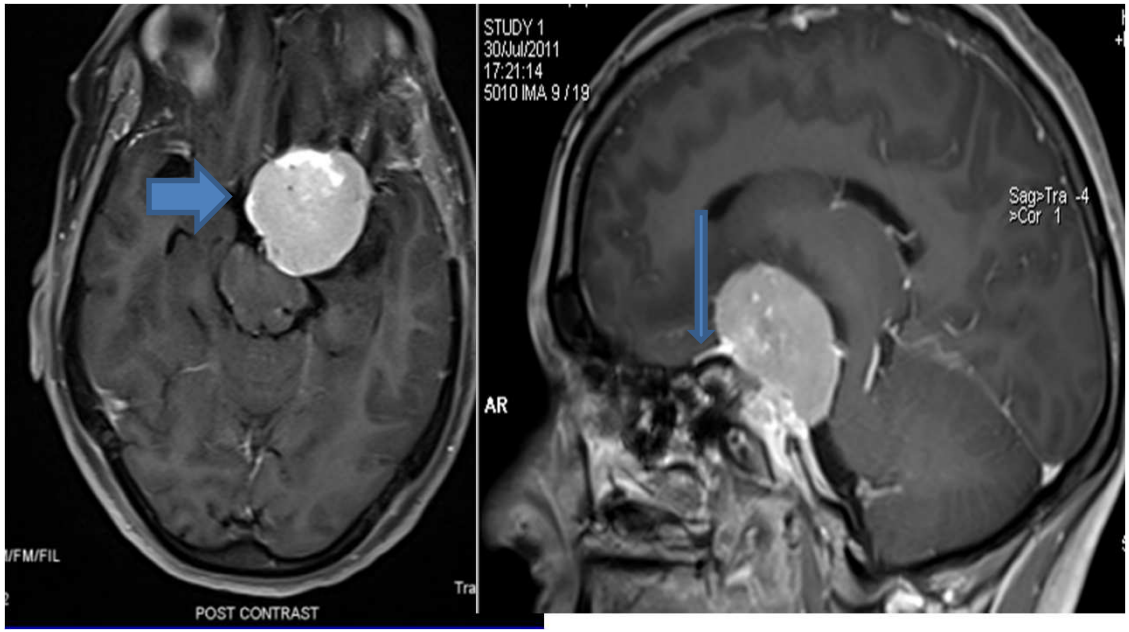
Intra-axial tumor: Adjacent cisterns are obliterated

EXTRAAXIAL SOLS: MENINGIOMAs



CECT

Parafalx meningioma Buckling of white matter(Thick arrow) and dural tail enhancement(thin arrow)



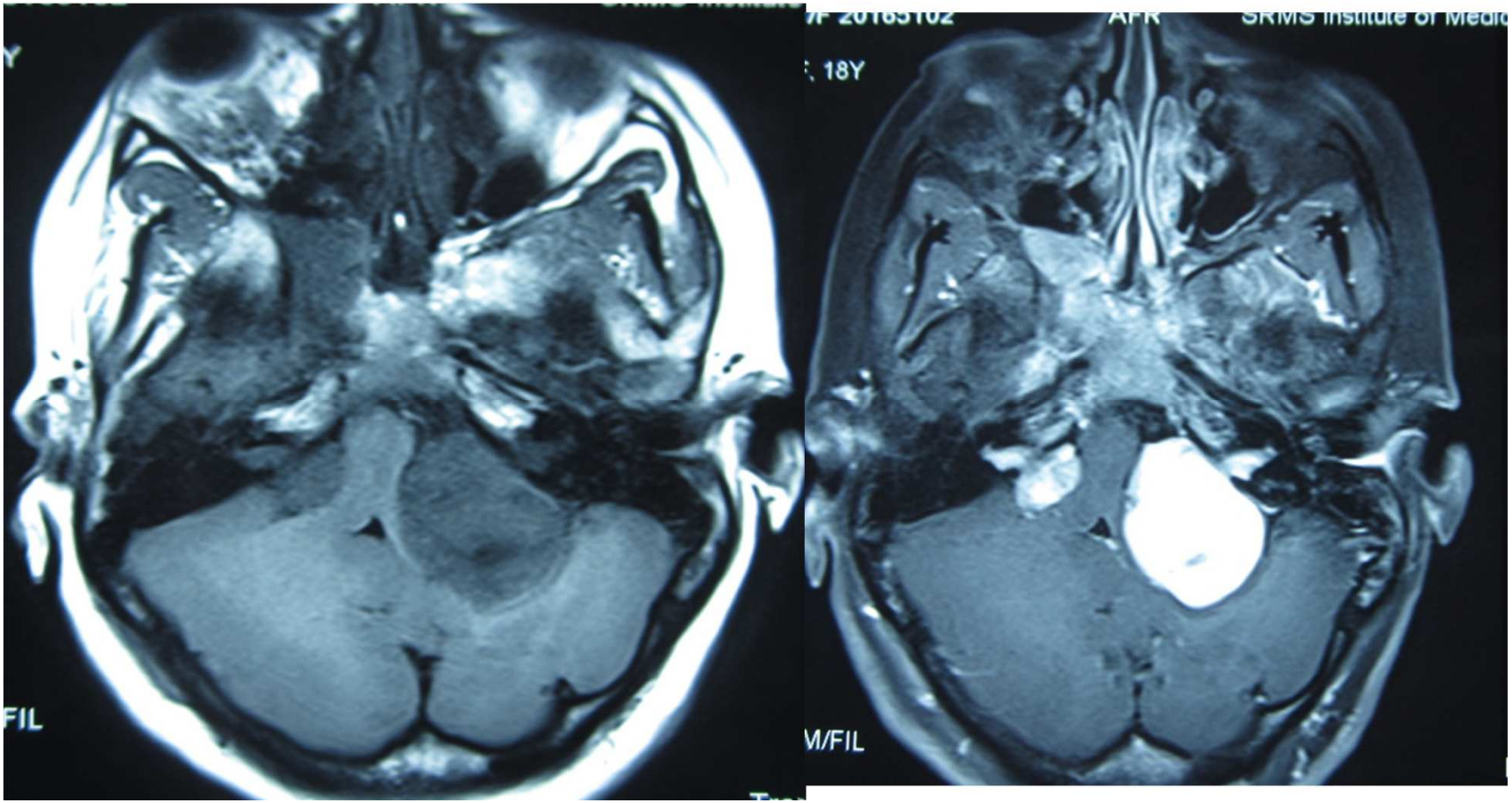
MRI:POST CONTRAST AXIA & SAGITTAL:

Sphenoid wing meningioma

Cisterns are widened(thick arrow)

& Dural tail enhancement(Thin arrow)

EXTRA AXIAL SOLs



B/L SCHWANNOMA. Cisterns are prominent. Contrast helful to shows Extension of sols into the porus acousticus

BRAIN TUMORS---INTERPRETATION OF CECT/MRI CONTRAST IMAGES

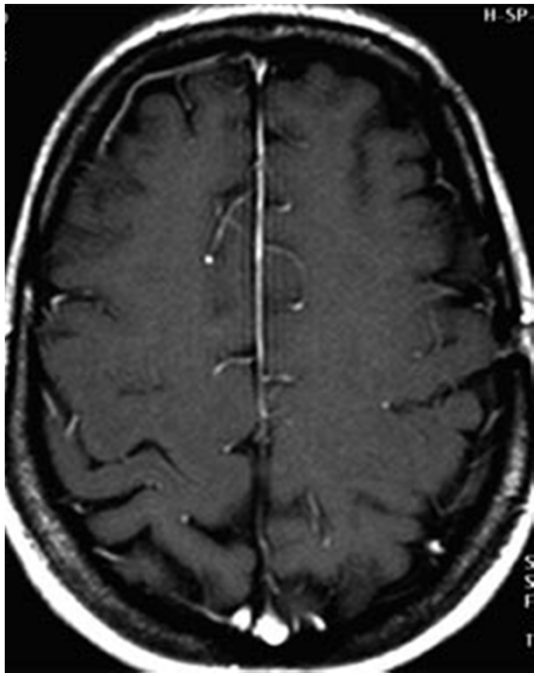
If the presence of a tumor & its location is determined , the best possible differential diagnosis should be given.

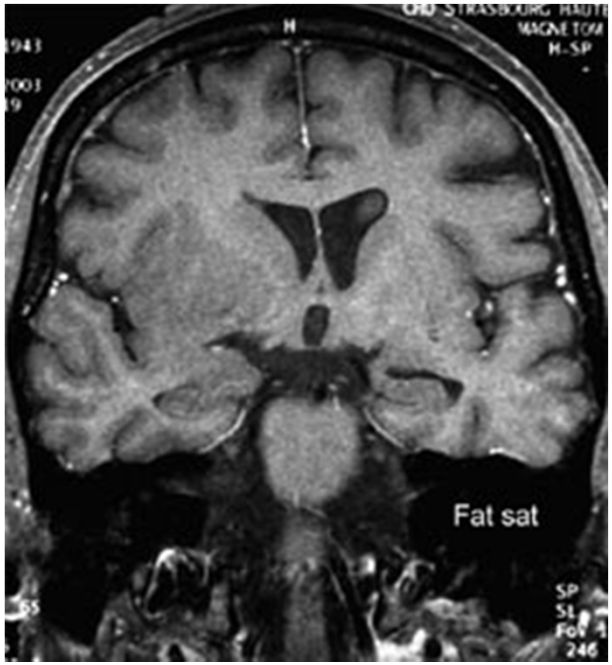
The following parameters that help with the differential diagnosis should be considered .

1.Age of the patient (most important in pediatric patients).
2.Relevant clinical history…Known case of malignancy
3.Previous available imaging studies and/or clinical and radiologic follow-up.
4.Location of lesion…Supratentorial Vs Infratentorial, Intraaxial versus extra axial location,
5.Single lesion versus multiple lesions.
6.Associated findings. Edema and/or tumor infiltration & Mass effect.

NORMAL MENINGEAL ENHANCEMENT

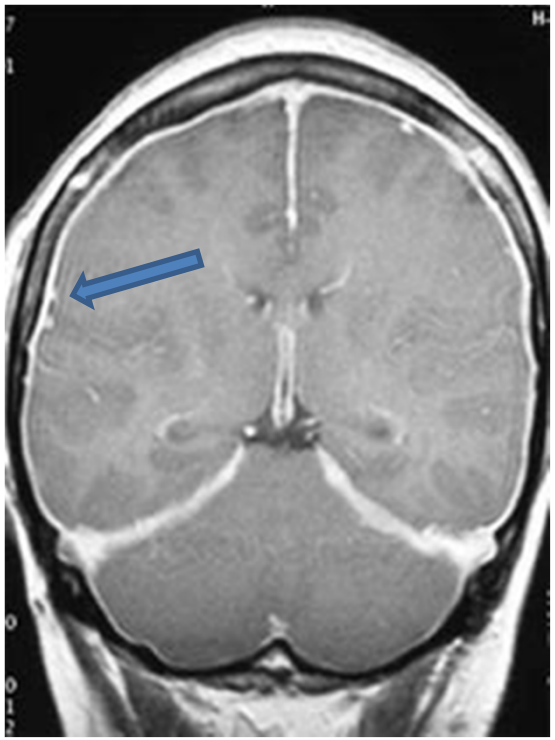
1.CT is of limited value for demonstrating meninges. Only falx cerebri & tentorium are routinely visualised in post contrast study.
2.MRI is better due to its high resolution



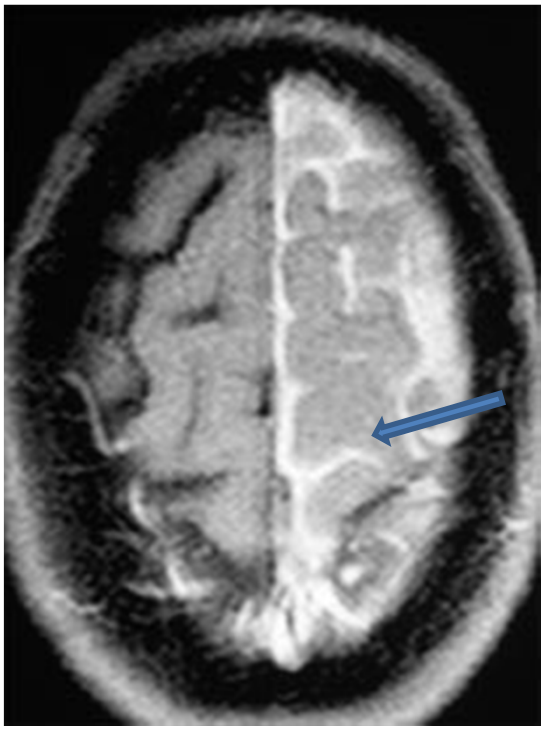


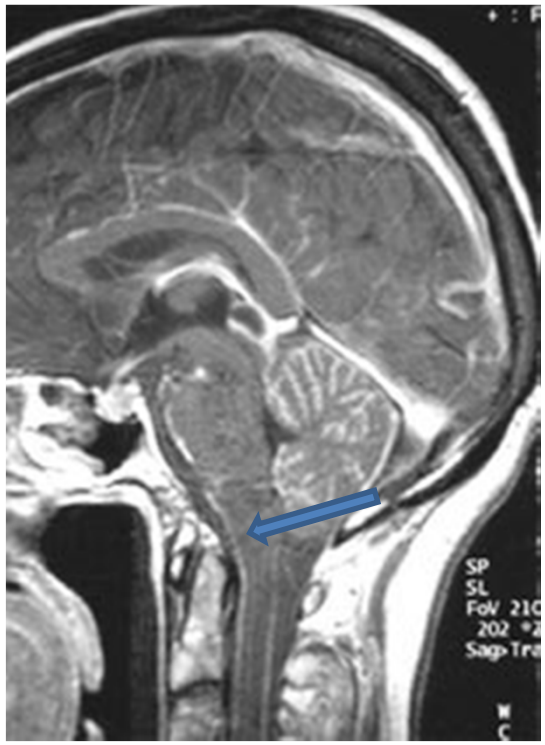
Axial & coronal images shows normal meningeal enhancement as linear, discontinuous, smooth enhancement of dura ,better seen in convexity. Falx & tentorium also shows enhancement

ABNORMAL MENINGEAL ENHANCEMENT



Abnormal thick Dural enhancement





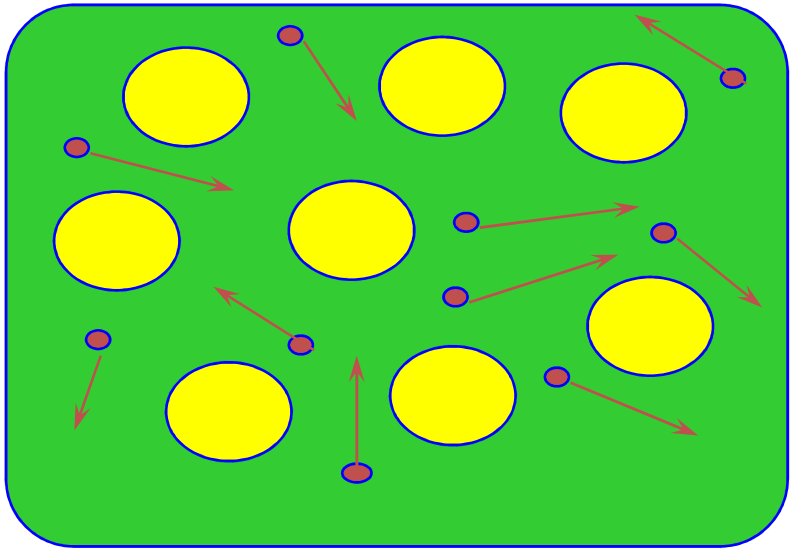
Abnormal leptomeningeal enhancement ..gyri,sulci & subarachnoid spaces. Enhancement is prolonged along spinal cord(Sagittal image)

COMMON CAUSES OF ABNORMAL MENINGEAL ENHANCEMENT:

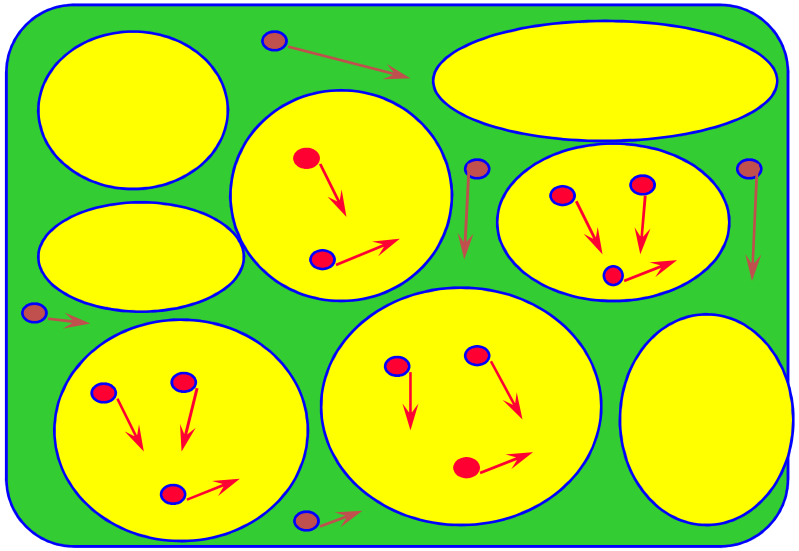
1. Post surgical changes.
2. Subarachnoid haemorrhage
3. Infection.
4. Neoplasm.

- Diffusion contrast -

- **Diffusion gradients sensitize MR Image to motion of water molecules**
- **More motion = Darker image =CSF,Vasogenic edema**
- **Less motion= Bright image= cellular tumor, Abscess, encephalitis,infarct**

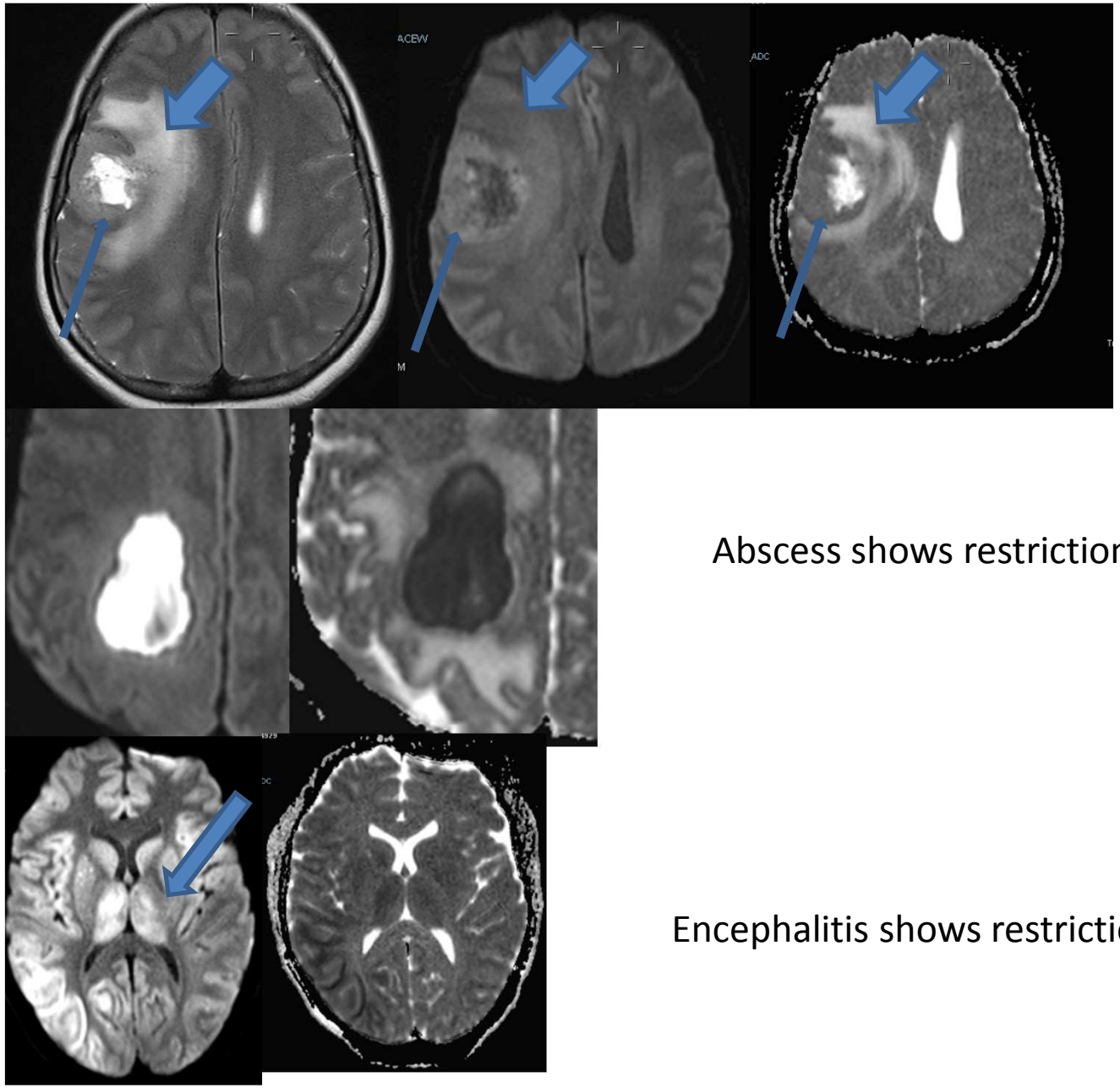


Freely Diffusing Water = Dark



Restricted Diffusion = Bright

VASOGENIC EDEMA Vs CYTOTXIC EDEMA



Vasogenic edema-
No restriction

Solid part of tumor
Shows restriction due to cellularity

Abscess shows restriction due to high viscosity

Encephalitis shows restriction due to cytotoxic edema

WHY THERE IS ENHANCEMENT OF SOL?

Nonneoplastic Astrocytic are required to induce BBB(Blood brain barrier) features of cerebral endothelial cells.

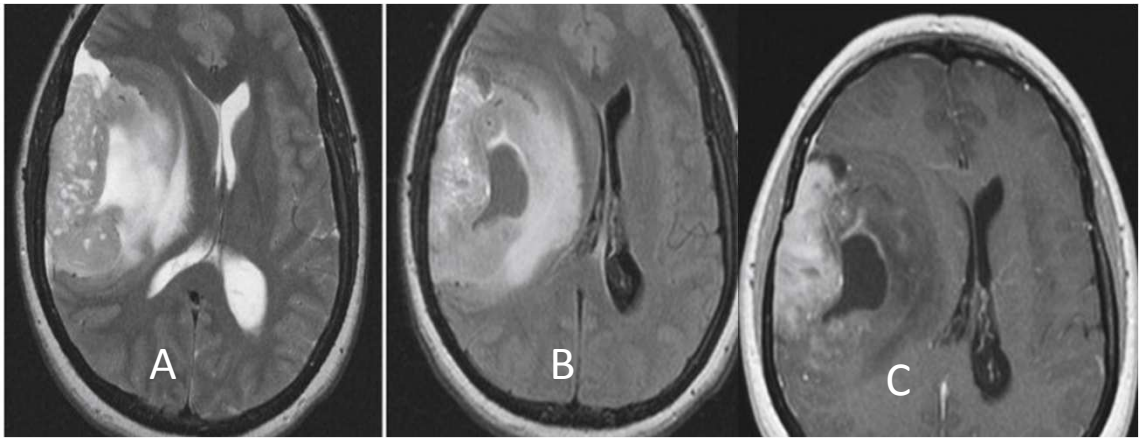
The enhancement seen in brain tumors is based on a disrupted blood–brain barrier (BBB), which can be compromised by revascularization and direct tumoral damage. Any lesion which Causes BBB disruption(Tumor, Radiation necrosis/ /infective/demyelinating lesion) will cause enhancement.

If a tumor or lesion is not causing disruption of BBB it will not cause an Enhancement.

Radiation necrosis cause disruption of blood brain barrier .
Recurrent high grade glioma may cause neoangiogenesis and no blood brain barrier disruption or both. It means they may or may not show enhancement in post contrast study

So at times it is difficult to differentiate on contrast enhancement images

1. Radiation necrosis from residual tumor/ recurrence.
2. Abscess from Tumor.
3. Primary from metastatic tumors unless there is a typical pattern
4. Tumor from Demyelinating lesion.



Typical enhancement pattern in a patient with malignant glioma. The more solid parts appear darker in T2 **(A)** and FLAIR **(B)** and present with a strong enhancement **(C)**. The surrounding lower tumor grade tissue still has an intact blood–brain barrier

PATTERNS OF GBM SPREAD.

"Brain to brain" metastases. because GBMs spread so quickly and viable tumor cells are available throughout much of the normal appearing brain, many neuropathologists and oncologists consider Glioblastoma a "whole-brain" disease.

White matter metastases of GBM spread is throughout the white matter. Tumor spreads directly into (and beyond) the peritumoral edema. Dissemination along compact white matter tracts such as the corpus callosum, fornices,anterior commissure, and corticospinal tract can result in tumor implantation in geographically remote areas such as the pons, cerebellum, medulla and the spinal cord.

CSF Dissemination: appearance of" carcinomatous meningitis" may be indistinguishable on imaging studies from pyogenic meningitis.

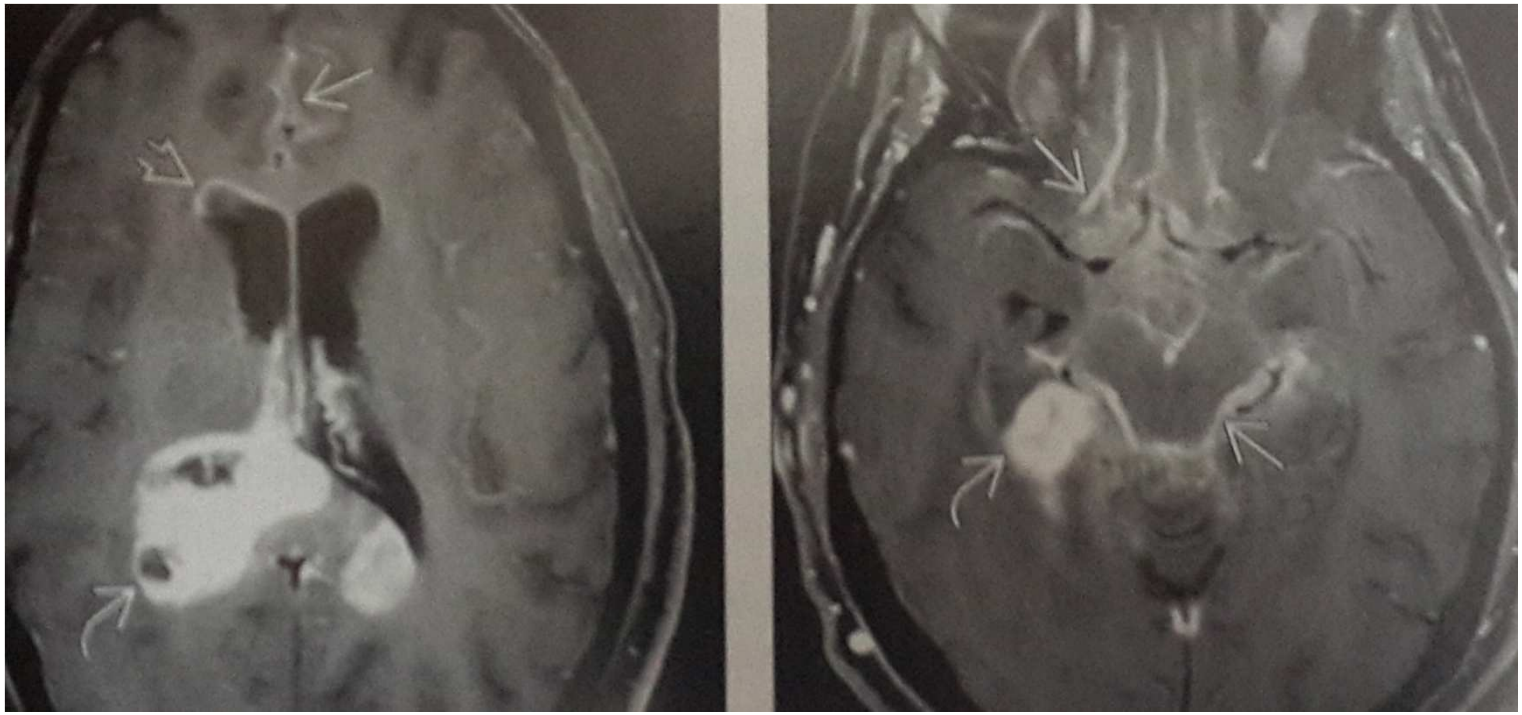
"Drop metastases" can extend inferiorly into the spinal canal,covering the spinal cord, thickening nerves, and causing focal mass- like deposits within the thecal sac.

PATTERNS OF GBM SPREAD.

Skull-dura invasion: Direct invasion of GBM through the pia and into the dura-arachnoid **is rare**. In exceptional cases, tumor erodes into and sometimes even through the calvaria, extending into the subgaleal soft tissues.

Extra-CNS metastases: Haematogenous spread of GBM to systemic sites occurs but **is rare**. Bone marrow(especially the vertebral bodies), liver, lung, and even lymph node metastases **can occur.**

TUMOR DISSIMINATION



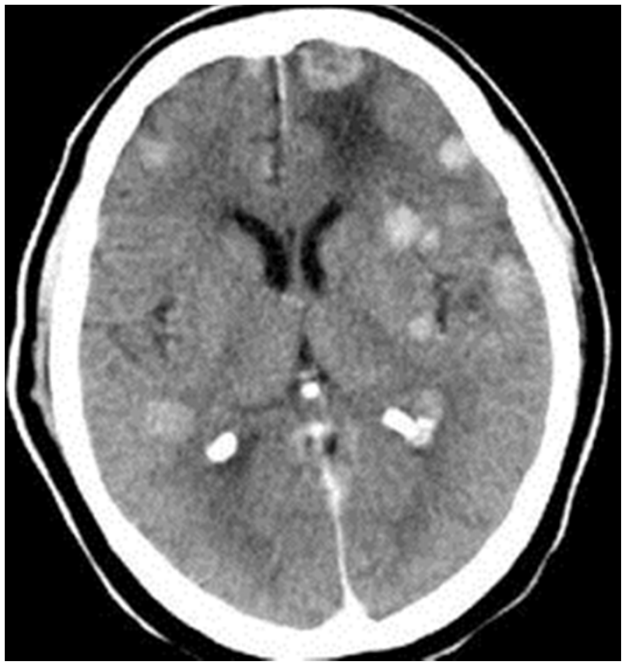
T1FS shows a nodular enhancing tumor with invasion of right ventricle together with a widespread CSF Dissemination..ependymal, cistern, sulcal enhancement, extending to olfactory sulcus

MULTIPLE RING/ NODULAR ENHANCING LESIONS :METASTATIC VS DD

cerebral metastases can mimic cerebral tumors. Intensive edema is present in large lesions but frequently is absent in small metastatic lesions.

Look for

1. Location
2. Shape
3. Multiplicity
4. Intensity/density & pattern of enhancement.
5. Presence of blood component.
6. Known history of malignancy.
7. Search extra axial location: skull,
   Leptomeninges: Arachnoid/subarachnoid
   Dural(patchymeningeal): less common: D/D Meningioma



METASTATIC

EVALUATION OF POST RT/CT TUMOURS

1.Contrast-enhanced MR is currently the imaging mainstay for monitoring treatment response in patients with GBMs.

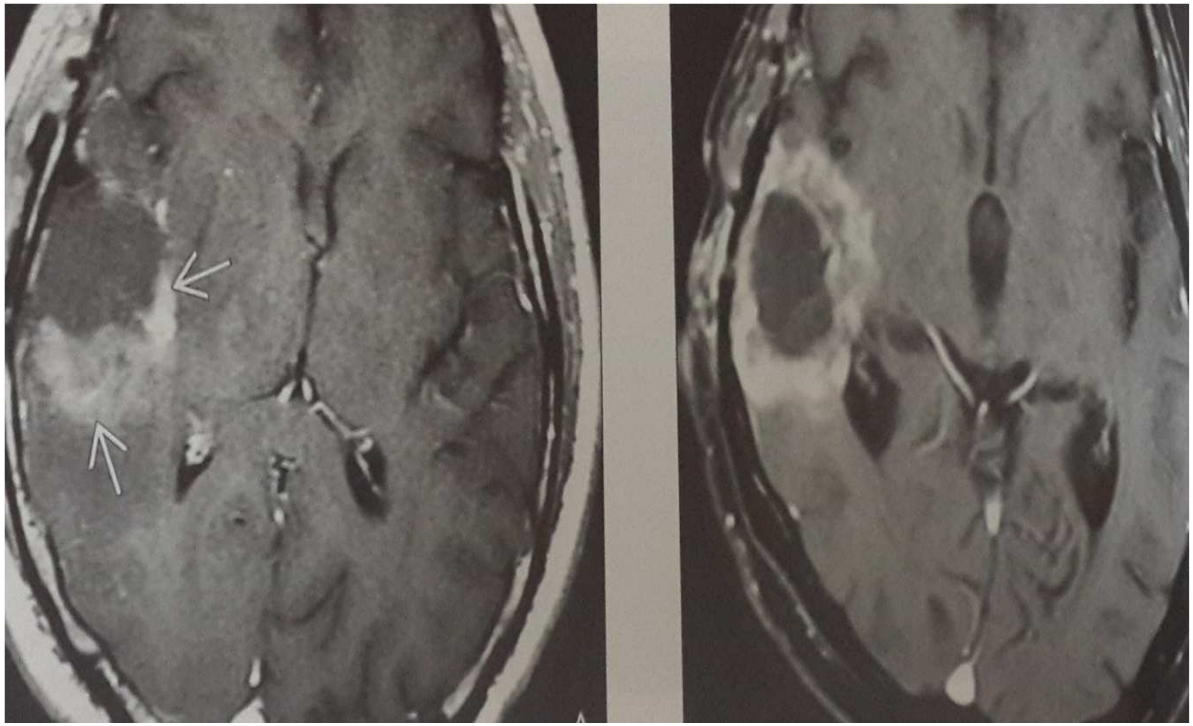
2.However, standard imaging can neither distinguish recurrent or progressive tumour from treatment induced parenchymal injury nor identify admixtures of tumour and parenchymal injury.

WHAT ARE MAJOR TYPES OF TREATMENT-RELATED EFFECTS THAT CAN MIMIC TUMOUR RECURRENCE

RADIATION NECROSIS AND PSEUDO PROGRESSION are the two major types of treatment-related effects that can mimic tumour recurrence. Radiation necrosis is a delayed response(from months to decades), but pseudo progression typically occurs within three months.

WHAT IS PSEUDOPROGRESSION

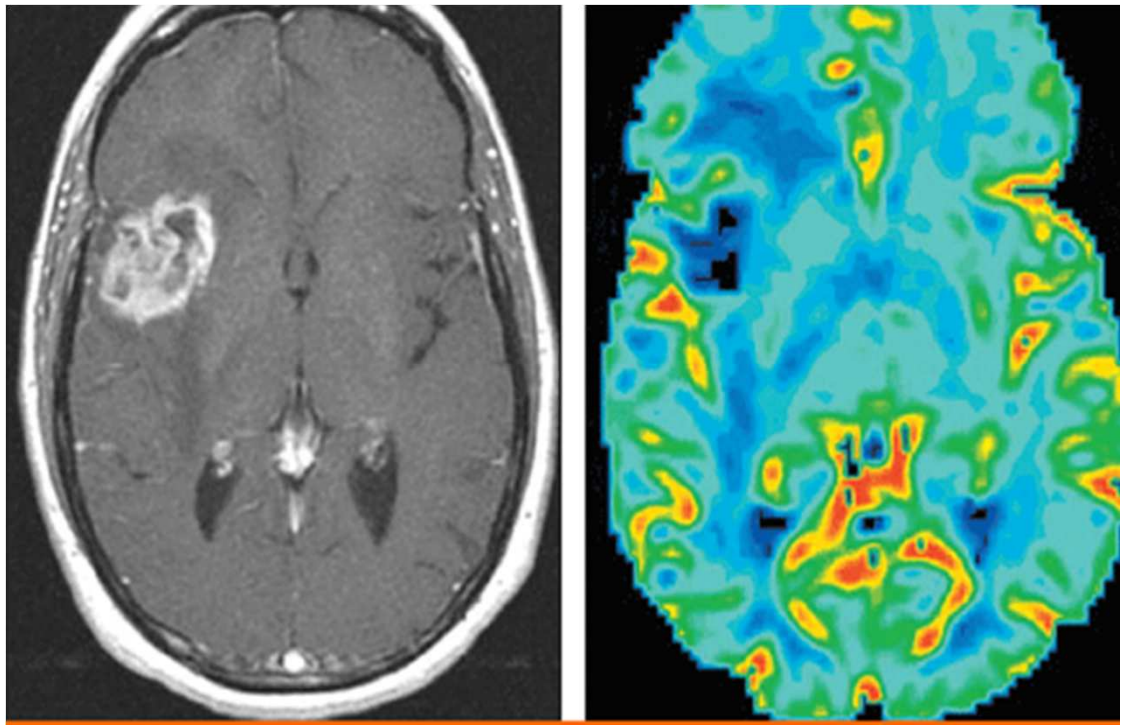
Pseudoprogression is a sub acute treatment-related reaction, usually associated with asymptomatic patients



POST SURGERY RECEIVED RT & CT

T1Contrast immediately following resection of right temporal lobe GBM. Residual enhancing Tumor outline the resection cavity. Patient received radiation & concurrent temozolomide. Five weeks later, thick enhancement Surrounds the resection bed, Biopsy ..mostly necrotic tumor. The enhancement represented Pseudo progression

PSEUDOPROGRESSION



AN AXIAL T1-WEIGHTED MR IMAGE. THE HETEROGENEOUS ENHANCING RIGHT INSULAR LESION WAS SLIGHTLY LARGER FOLLOWING RADIOTHERAPY.

THE RELATIVE CEREBRAL BLOOD VOLUME MAP SHOWS MARKED DECREASED BLOOD VOLUME IN THE REGION OF ENHANCEMENT, WHICH IS CONSISTENT WITH PSEUDOPROGRESSION AS THE DOMINANT UNDERLYING CAUSE OF ENHANCEMENT

Folowing surgical resection and radiotherapy with concurrent temozolomide, lesion enlargement on the first follow up MR is often observed. Almost half of all patients show increased mass effect and new areas of enhancement compared to immediate baseline postoperative imaging. Approximately 40% are secondary to pseudo progression rather than “true” tumor progression.

Distinguishing early “true” progression from pseudo progression is difficult .pMR with dynamic susceptibility-weighted contrast enhancement can be used to map rCBV and estimate tissue microvasculature across lesions. rCBCV canbe quantify tumor burden relative to components of pseudo progression and radiation necrosis.

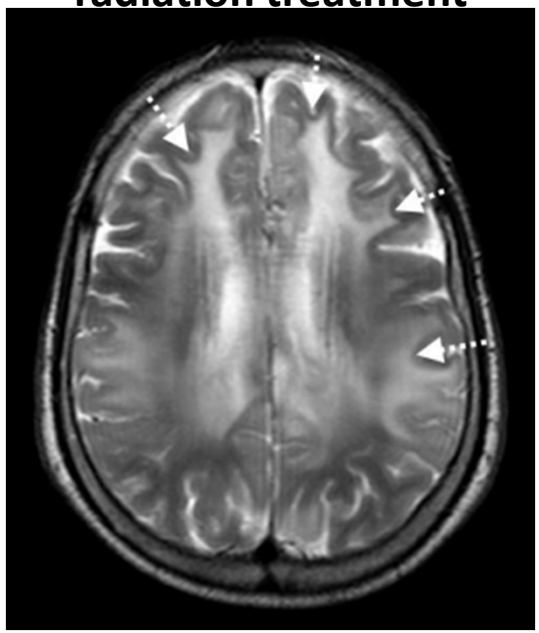
The use of biodegradable carmustine(Gliadel) wafers also complicates postoperative imaging. Ring enhancements occurs within one postoperative day and peaks at one month. Restricted diffusivity may last upto one year.

CENTRAL NERVOUS SYSTEM SYNDROMES SECONDARY TO RADIOTHERAPY

1.**Acute encephalopathy** occurs during and up to 1 month after radiotherapy. This is due to disruption of the blood-brain barrier.

2. **Early delayed complications occur 1-4 months after radiotherapy are demyelination and vasogenic edema**. produce a somnolence syndrome(drowsiness**)** in children, reappearance of the initial tumour's symptomatology, temporary decline in long-term memory, and encephalopathy.

3. **Radiation necrosis and diffuse cerebral atrophy** are considered long-term complications of radiotherapy that **occur from months to decades after radiation treatment**



DEMYELINATION OF WHITE MATTER: POST RT, PML,AIDS etc

RADIATION NECROSIS

Radiation necrosis in the brain occurs in three different clinical settings.

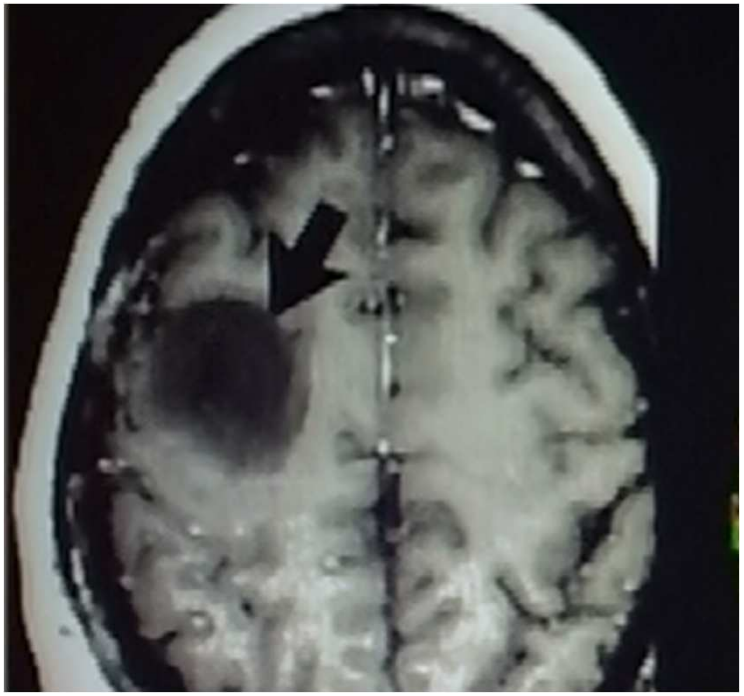
(a)A new brain enhancement or signal abnormality in a patient with a history of radiation therapy for extra cranial head and neck malignancy or intracranial extra axial tumor;
(b) enlargement of an enhancing lesion following stereotactic radiation therapy that includes radio surgery; and
(c) worsening signal abnormality or enhancement following fractionated radiation therapy for brain malignancy.

NOTE: Radiation necrosis can be suspected or diagnosed in the context of treatment for extra cranial head and neck malignancies or intracranial extra axial tumors on the basis of (a) location of primary malignancy, (b) extent of radiation port, (c) type of radiation, and (d) inclusion of normal structures (depending on the radiation treatment technique used and the amount of time elapsed since radiation therapy).

WHAT IS BEYOND CONTRAST ENHANCED IMAGES FOR TUMOUR INTERPRETATION

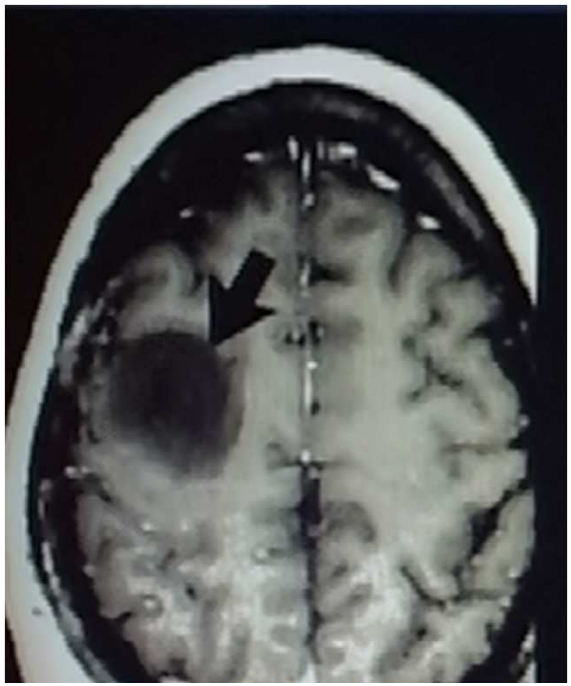
In the past few years, a number of advanced, no enhanced, and contrast-enhanced MRI techniques have been developed that provide new insights into the pathophysiology of brain tumors, mainly gliomas. They include MR spectroscopy (MRS), perfusion MRI, dynamic contrast-enhanced MRI, Diffusionand diffusion tensor(DTI) MRI.

What should we perceive if a lesion is not enhancing: It may still be a malignant lesion, Do a perfusion study, it's a few second study after contrast injection and than a 3D T1wt Contrast study,
As perfusion tells about neoangiogenesis

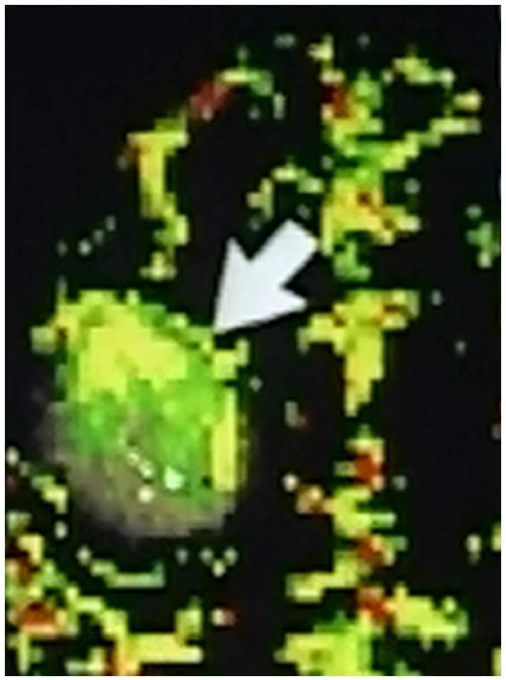


NO ENHANCEMENT IN POST CONTRAST STUDY

PERFUSION TELLS ABOUT NEOANGIOGENESIS



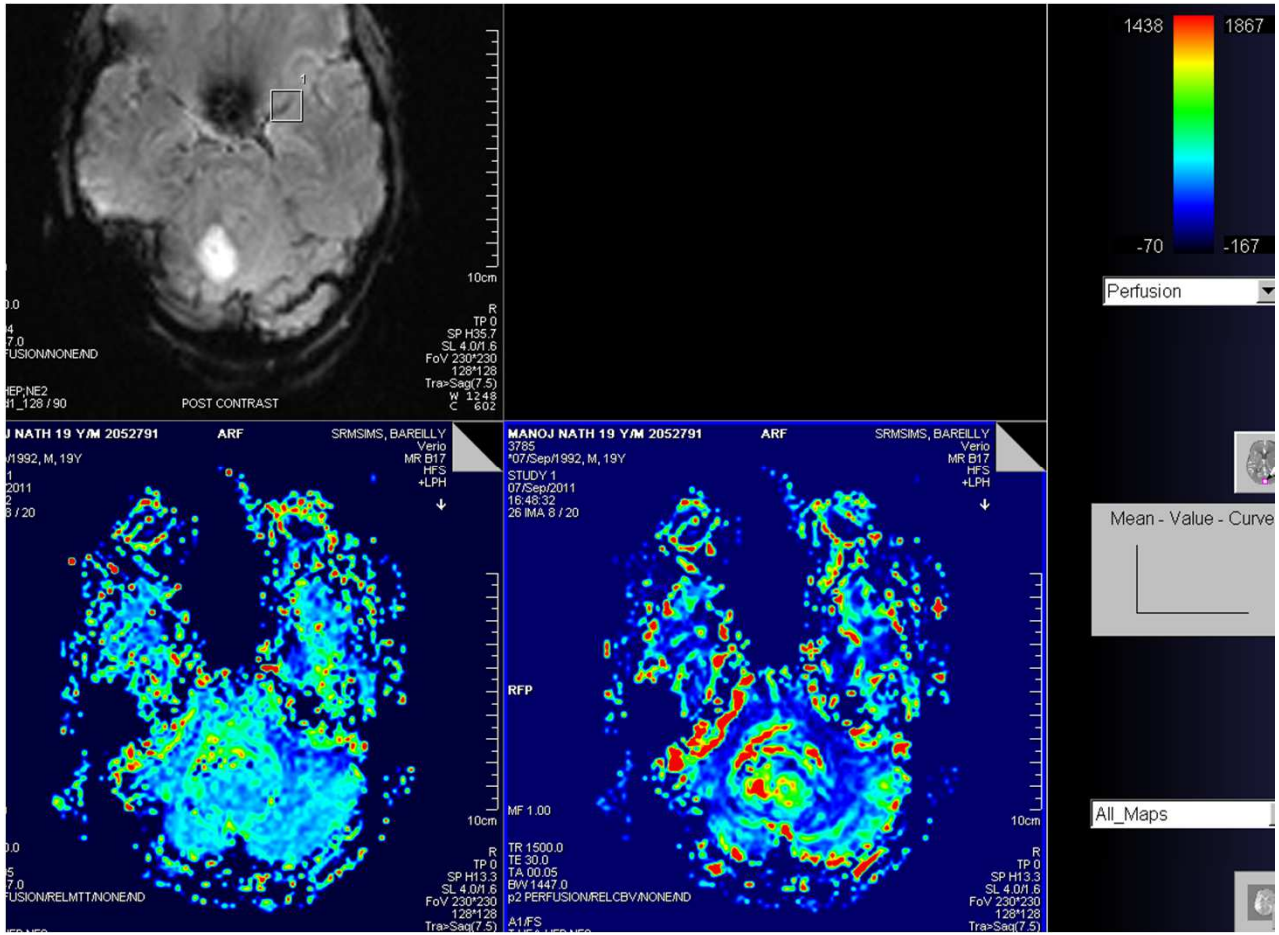
POST CONTRAST: NO ENHANCEMNT



PERFUSION: HIGH CBV
BIOPSY: Anaplastic Astrocytoma

TEACHING POINT: CONTRAST ENHANCEMENT IS NON SPECIFIC FINDING AND ONLY INDICATE BBB DISRUPTION

PERFUSION STUDY



CBV 1.5—1.75 high grade gliomas, rCBV <1.5 low grade gliomas

Tumor progression(Angiogenic Switch): If there is a increase in rCBV in a low grade Tumour….. Conversion to high grade….seen in the MR perfusion study

While conventional contrast MRI will show Contrast enhancement which is due to Disruption of blood brain barrier

PERFUSION

Evaluating the tumor grade

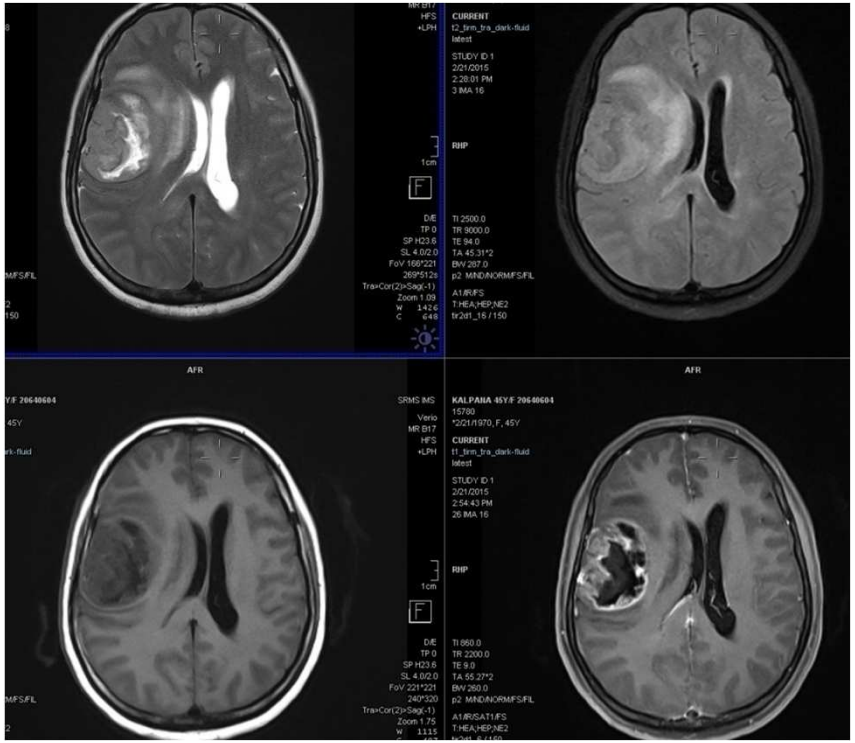
Stereotactic biopsy guidance

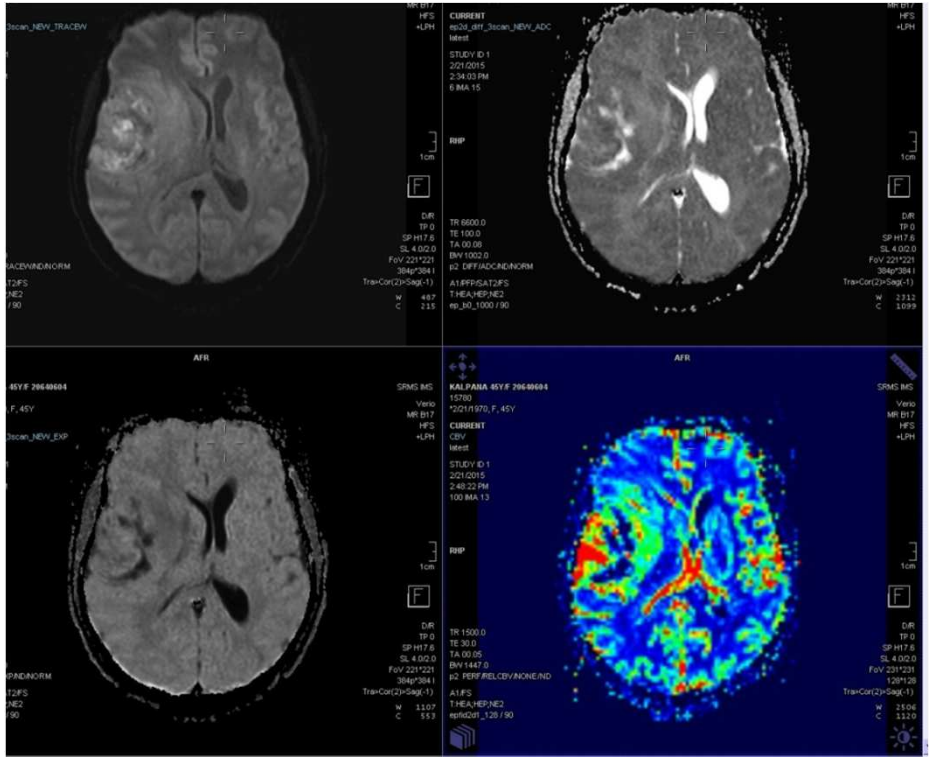
Assessment of response to therapy

Differentiation of radiation induced necrosis & recurrent tumor

Nonneoplastic lesions: infections, tumefactive demyelinating lesions

HIGH GRADE GLIOMA





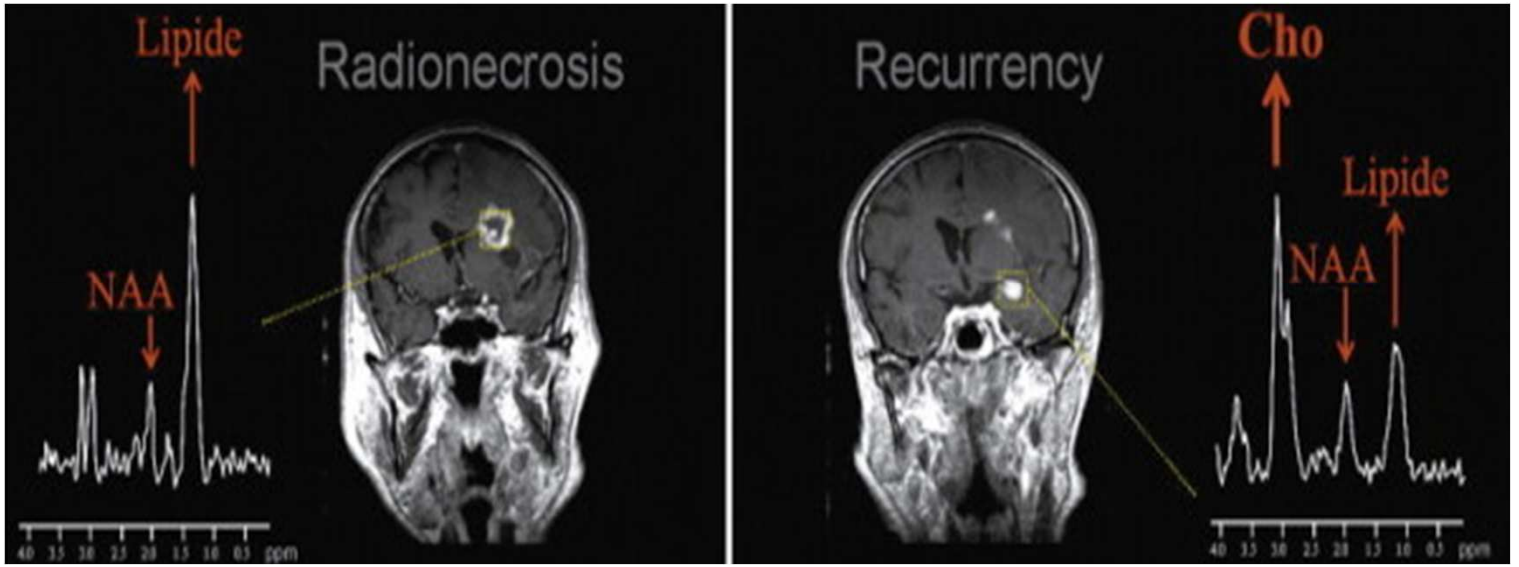
Conventional MR imaging is very limited in making the distinction from primary tumor Vs metastatic. Contrast enhancement on T1-weighted images reflects areas of blood-brain barrier breakdown regardless of the pathology. FLAIR imaging can depict a large portion of the tumor but also is nonspecific

Perfusion rCBV, which correlate with tumor vascularity which is present in the solid component of tumor as well as in the surrounding edema(In mets no perfusion in surrounding edema) and allow indirect assessment of tumor angiogenesis.

DIFFERENTIATING RECURRENT TUMOR FROM THERAPEUTIC-INDUCED CHANGES

After radiation or chemotherapy, BBB breakdown can occur and mimic tumor recurrence.

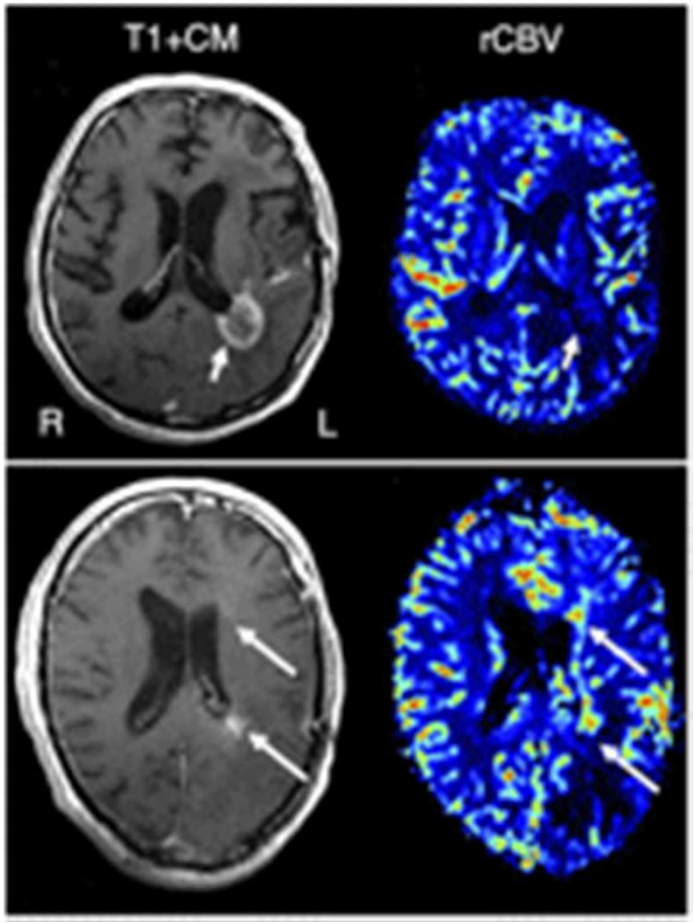
MRS has been shown to be useful in identifying tumoral tissue by the presence of choline and, in high-grade tumors, lactate. Choline, which is a marker of membrane turnover, is normally not present in necrosis or neoangiogenesis increasing the perfusion(r CBV=relative cerebral blood volume)



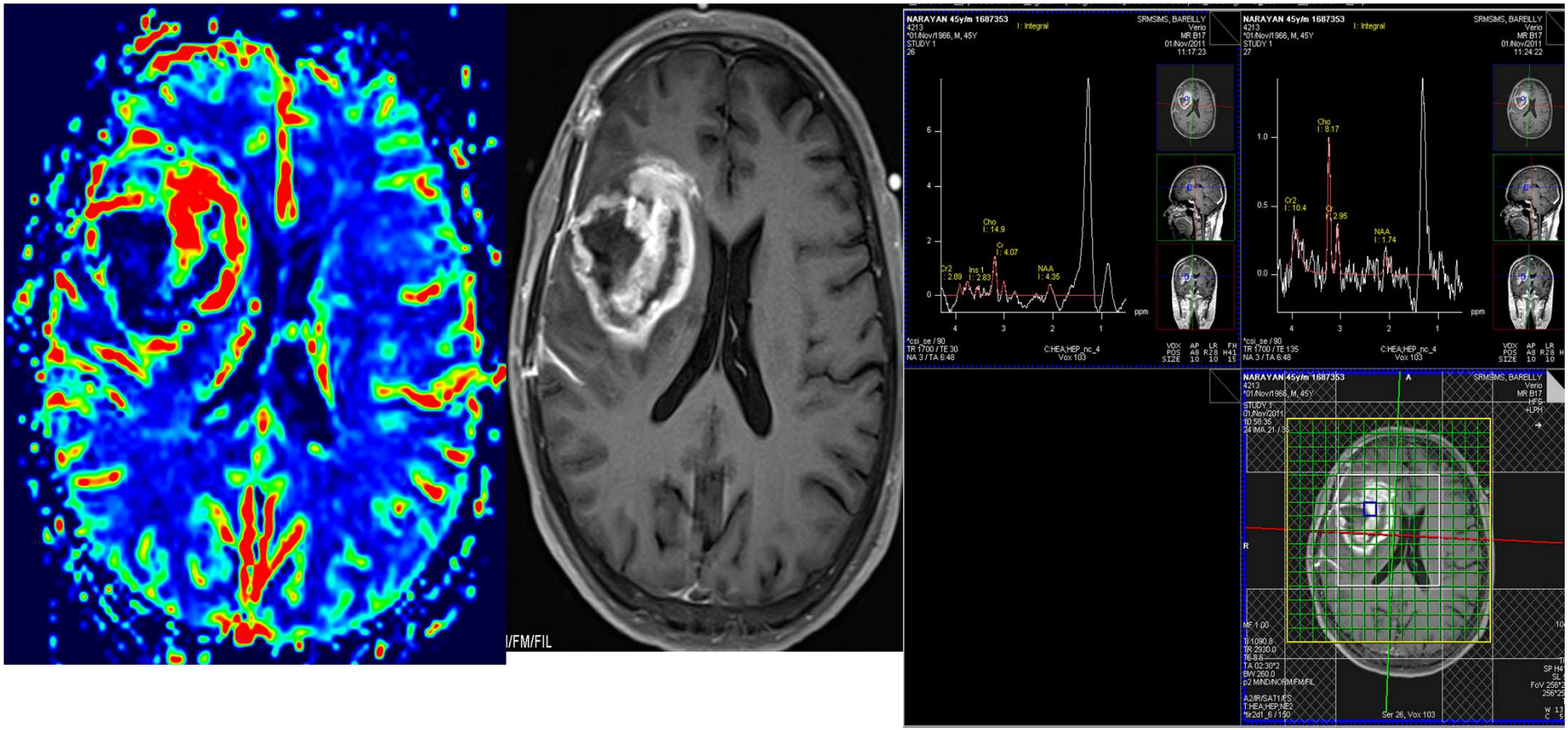
When Cho: Cr ratio from opposite normal range is > 1.3 suggest Tumor recurrence

RADIATION INDUCED CNS CHANGES VS TUMOR RECURRENCE..DIAGNOSTIC CHALLENGE

Role of perfusion: If perfusion rCBV value> 2.6 suggest Tumor recurrence
< 0.6 suggest therapy related nonspecific changes
When rCBV is between 0.6 & 2.6 Do PET CT/MRI. Suspicious lesions on MR imaging
That show increase FDG uptake likely to represent Tumor recurrence

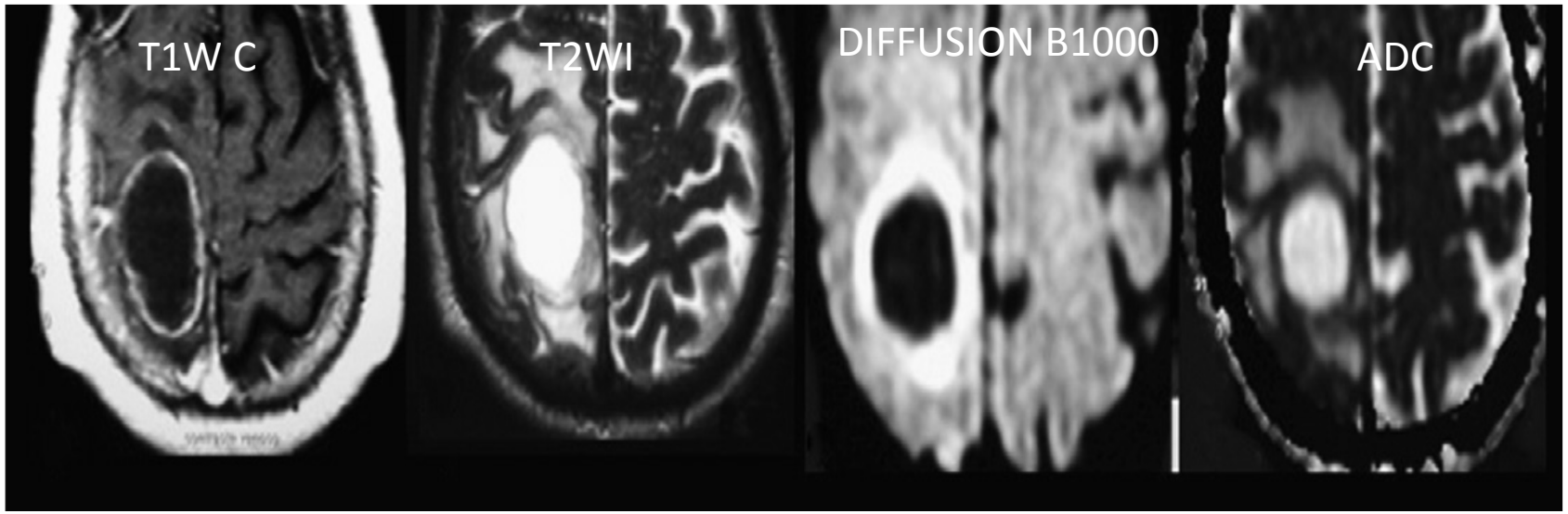


T1-weighted contrast-enhanced (T1+CM) and rCBV MR images showing radiation necrosis with low rCBV values (top row) and recurrent tumor with rCBV elevation along left ventricular wall (bottom row).

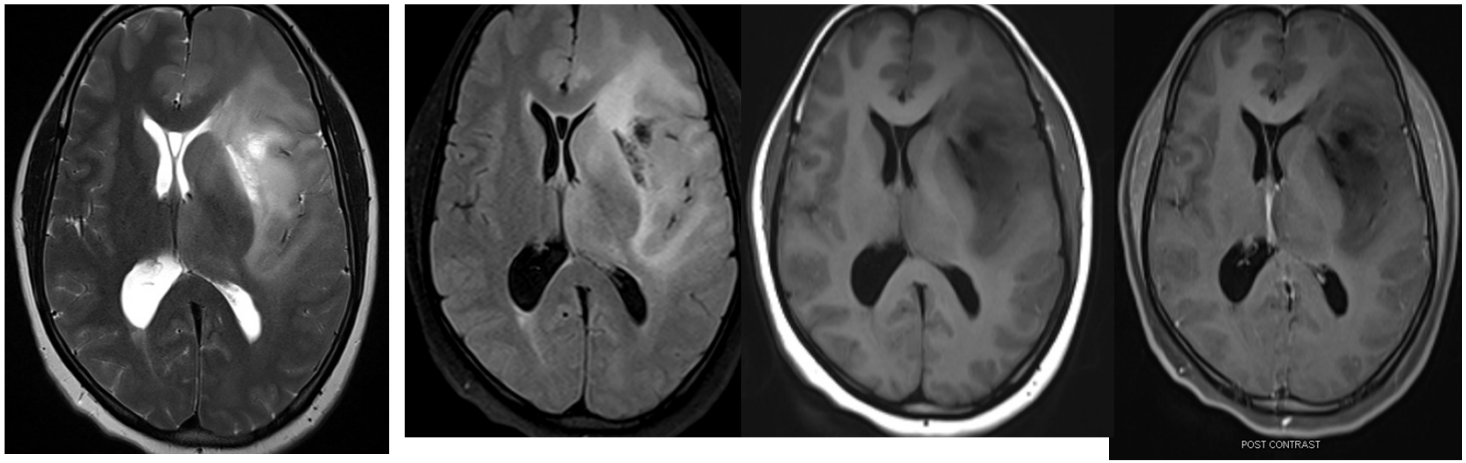


46 yrs old man with post op GBM for RT planning.
Perfusion shows high rCBV, MRS shows high choline ,low NAA &
High Lipid/Lactate peak ..A feature of High Grade Glioma

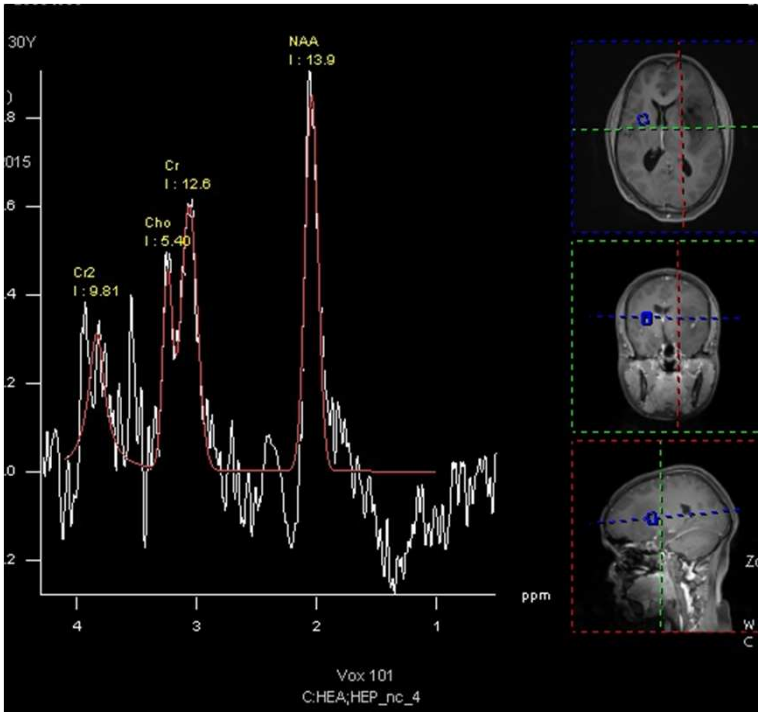
WHAT IS THIS?

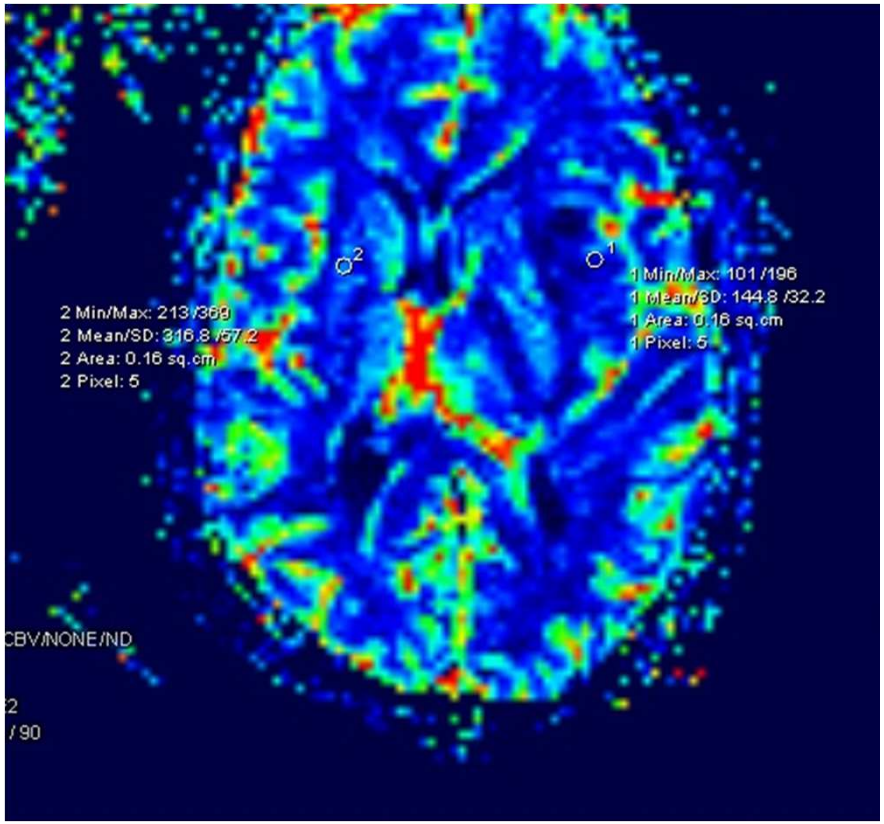


. Axial contrasted-enhanced T1-WI (A), T2-WI (B), DWI (C), and ADC mapping


POST CONTRAST

?


30Y
NAA
I : 13.9
Cr
I : 12.6
Cho
I : 5.40
Cr2
I : 9.81
ppm
4
3
2
1
Vox 101
C:HEA;HEP_nc_4


2 Min/Max: 213/369
2 Mean/SD: 316.8 /57.2
2 Area: 0.16 sq.cm
2 Pixel: 5
1 Min/Max: 101 /196
1 Mean/SD: 144.8 /32.2
1 Area: 0.16 sq.cm
1 Pixel: 5
CBV/NONE/ND
/ 90

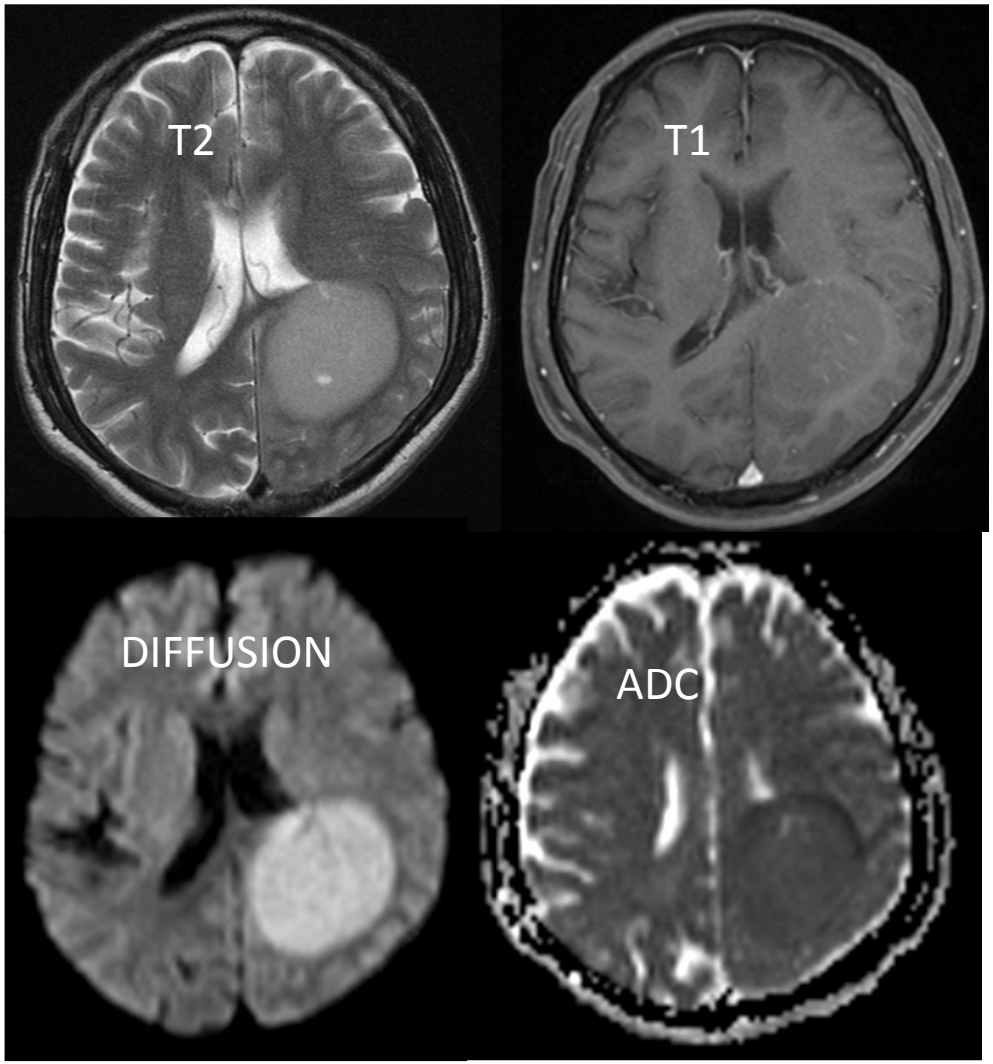
LYMPHOMA

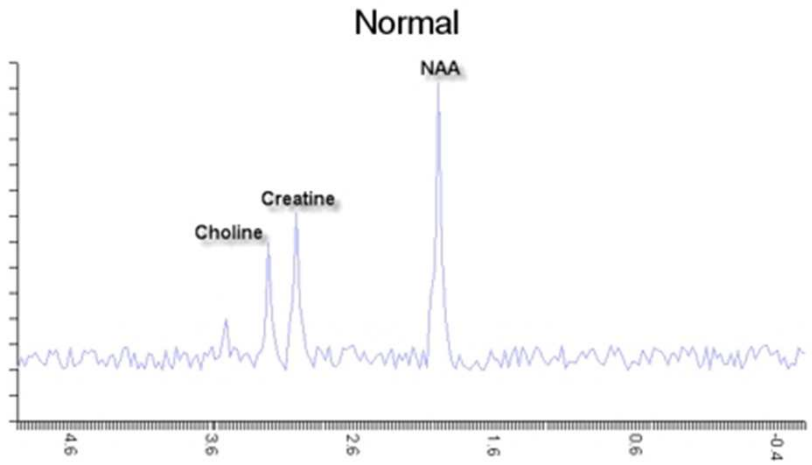
Lymphoma of the CNS consists of 2 major subtypes:

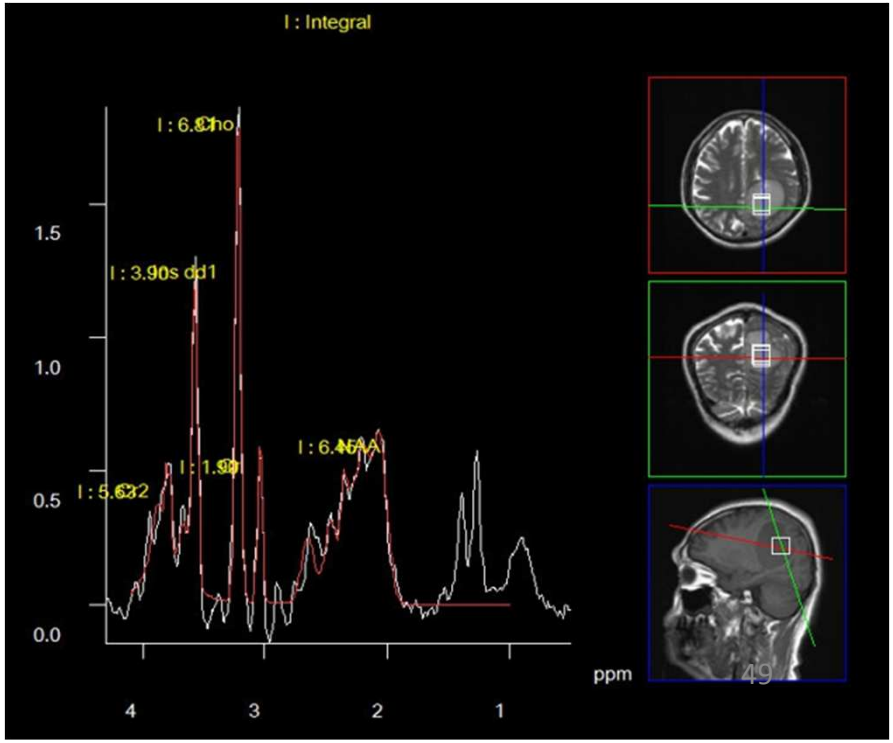
1.Secondary CNS involvement by systemic lymphoma (the most common) .

2.PCNSL(Primary CNS lymphoma), in which the lymphoma is restricted to the brain, leptomeninges, spinal cord, or eyes, without evidence of it outside the CNS at primary diagnosis

Maximum relative CBV measured in tumor tissue, calculated as a ratio to contra lateral normal-appearing white matter, is typically lower in lymphomas than in other brain tumors. This characteristic finding can help to differentiate Glioblastoma and metastases from lymphomas







LYMPHOMA AS SOLITARY MASS

LOW ADC—0.51 +/- 0.09

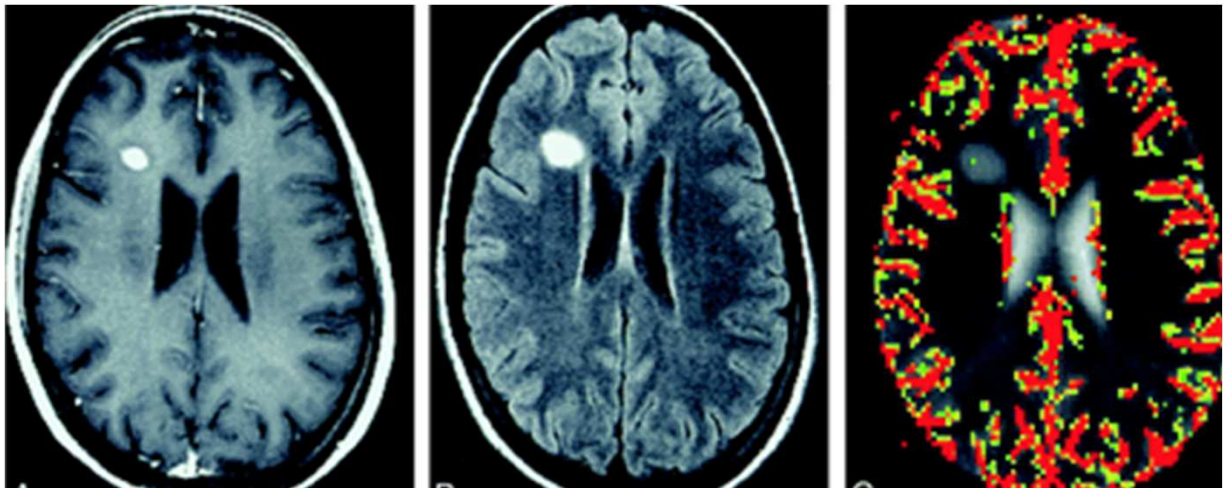
MRS- CHOLINE INCREASE, NAA- DEC, LIPID INCREASED

TUMEFACTIVE DEMYLINATING LESIOSN

The rCBV values of TDLs ranged from 0.22 to 1.79 (n = 12), with a mean of 0.88 ± 0.46 (SD).

The rCBV values of intracranial neoplasms ranged from 1.55 to 19.20 (n = 11), with a mean of 6.47 ± 6.52.

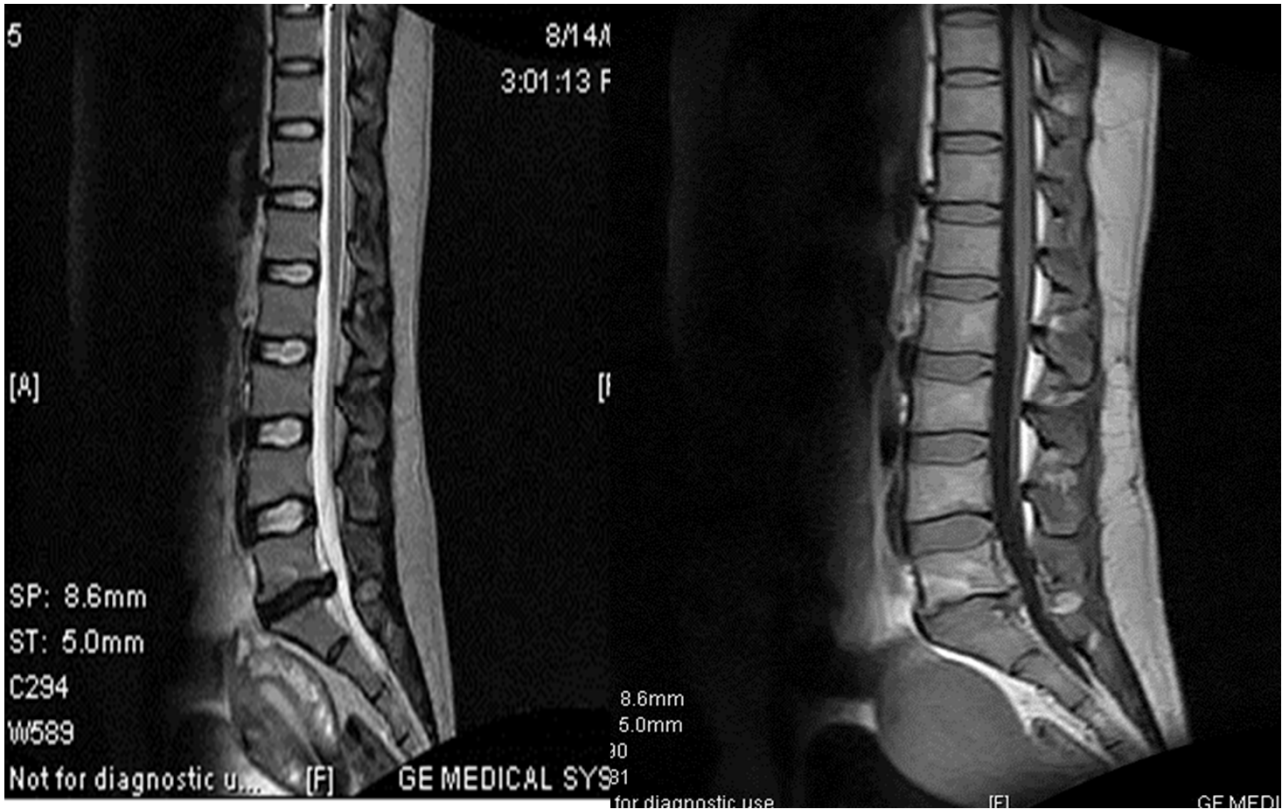
Proton MR spectrum shows an elevated Cho value, a decreased NAA value, and a Lac doublet.



T1C FLAIR rCBV Low

SPINE T1WT WITHOUT FAT PRE/POST CONTRAST

SEE
Vertebrae /posterior arches
Anterior/posterior epidural space
Cord & nerve roots
Pre/paravertebral soft tissues



SPINAL TUMORS

WHERE IS THE TUMOR:

- Within the canal?
- Outside the canal?

IF IT IS INSIDE THE CANAL:

- In the cord?
- outside the cord?

Adult or Child.

SPINAL TUMOR

INTRADURAL:

- Intramedullary:
- Extramedullary

EXTRADURAL:

- Epidural
- Vertebral

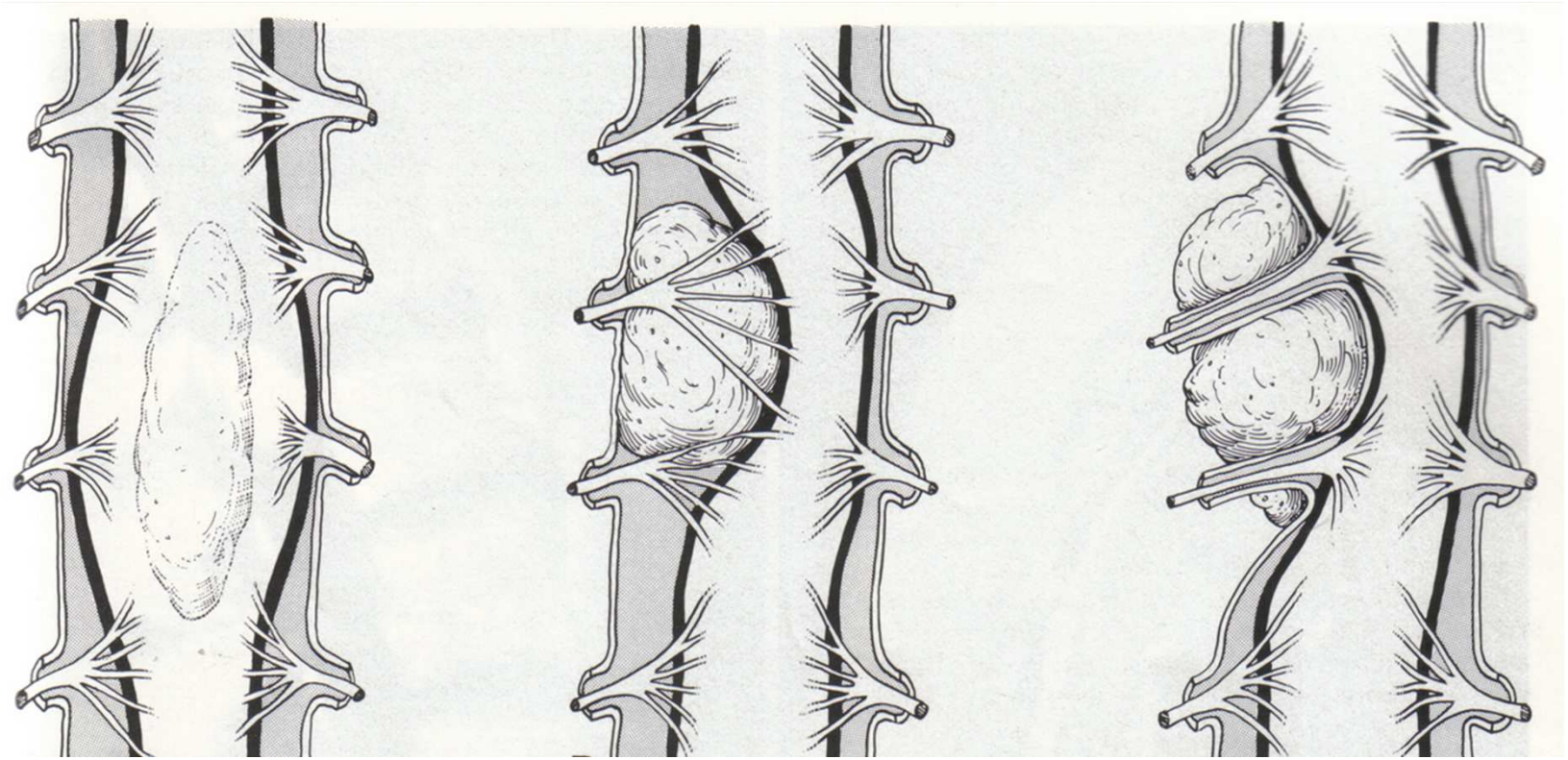
SPINAL TUMOR

INTRAMEDULLARY:

- Gliomas(Ependymoma,Astrocytoms)
- Hemangioblastoma

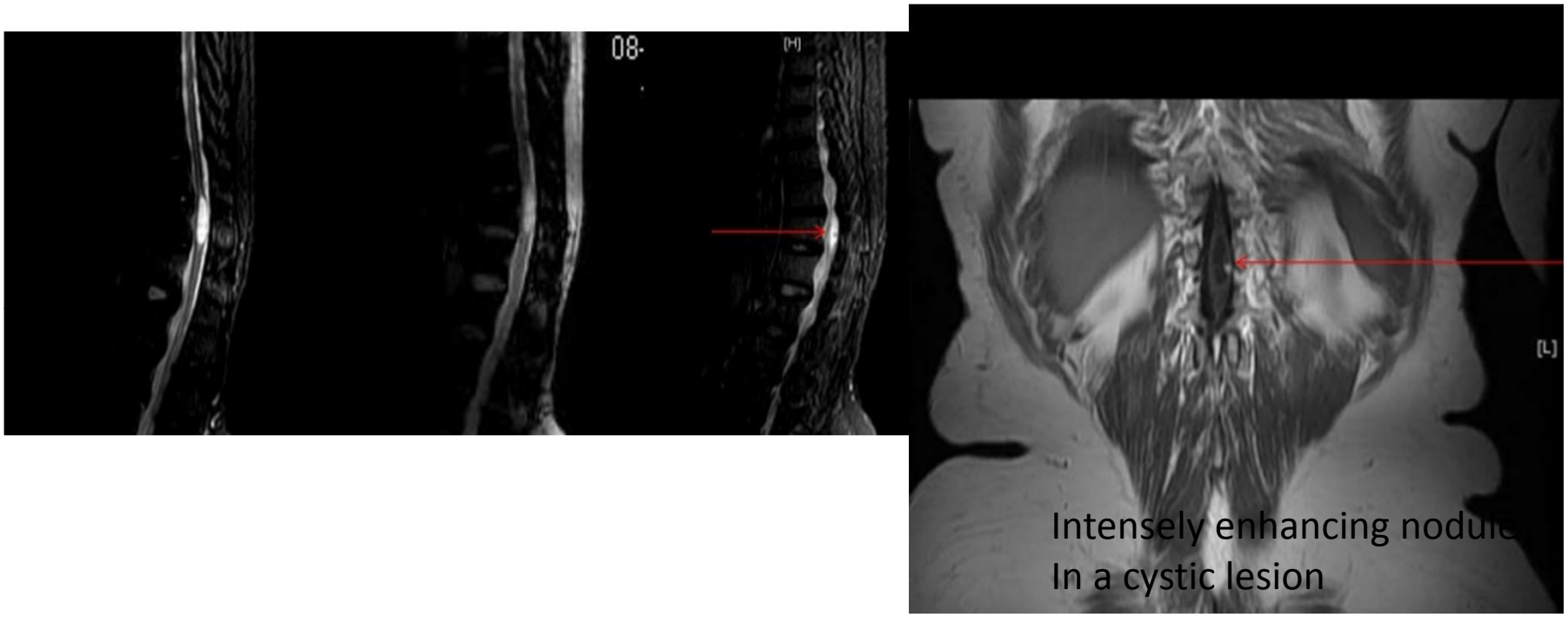
EXTRAMEDULLARY:

- Meningioma
- Neurinoma

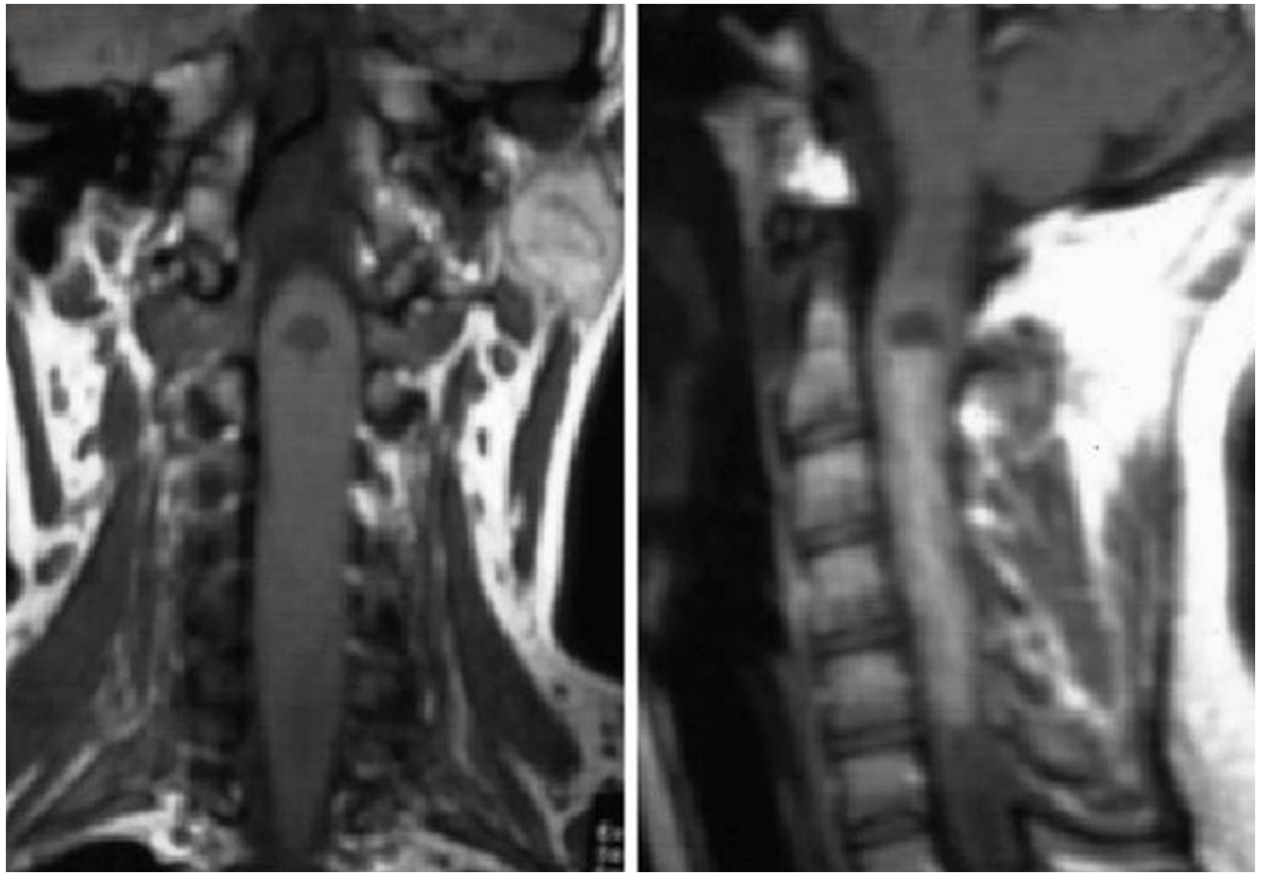


INTRAMEDULLARY **INTRADURAL EXTRAMEDULLARY** **EXTRADURAL**

HEMANGIOBLASTOMA

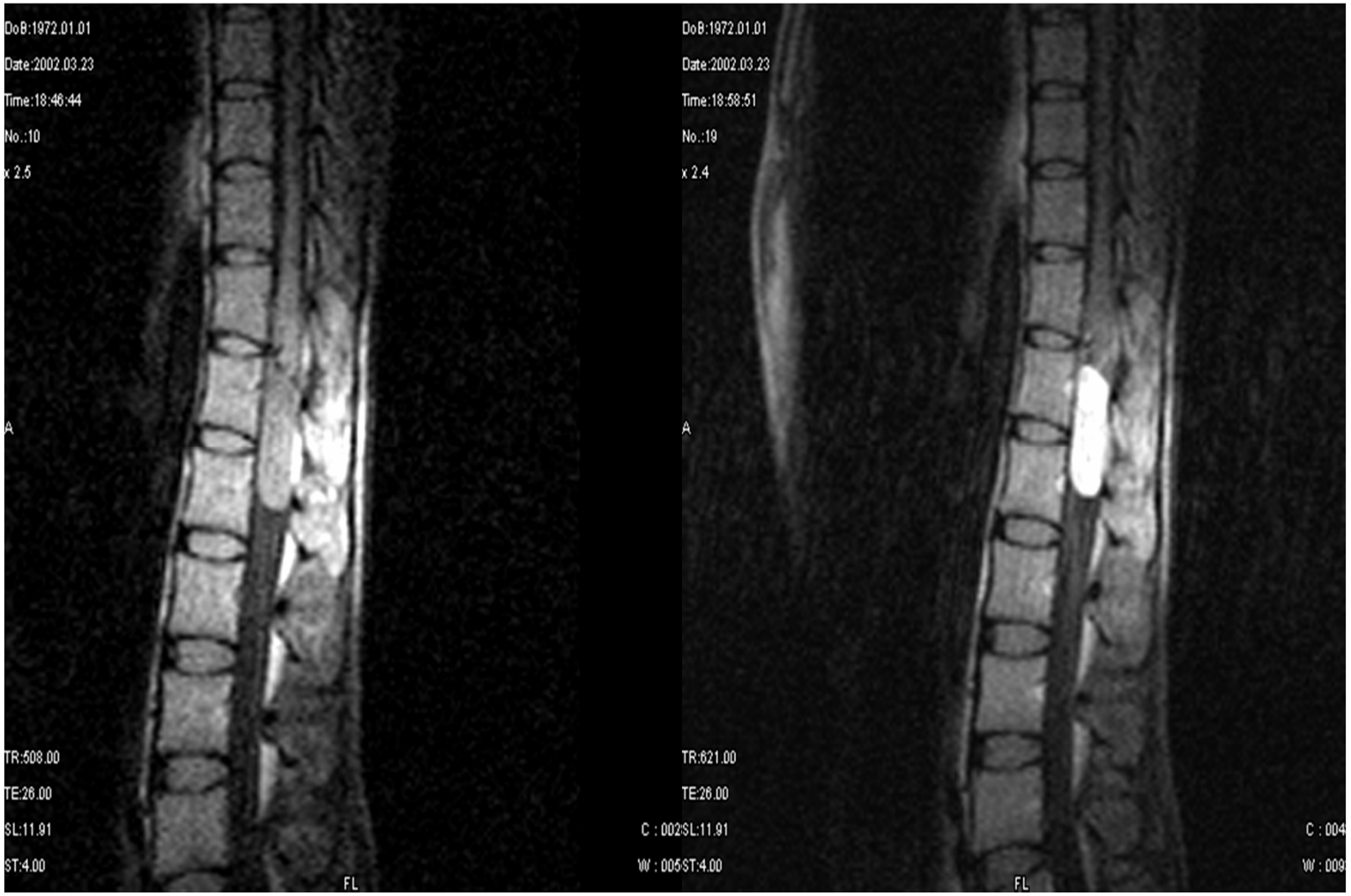


1Highly vascular tumor,Usually intramedullay, can be intradural, Extradural.
2.Common in dorsal spine followed by cervical spine
3 common post fossa tumor of adult but relatively
Rare in spinal cord.
4.Lesion is unlikely to be hemangioblastoma if it is larger than 25 mm and there are no associated flow voids



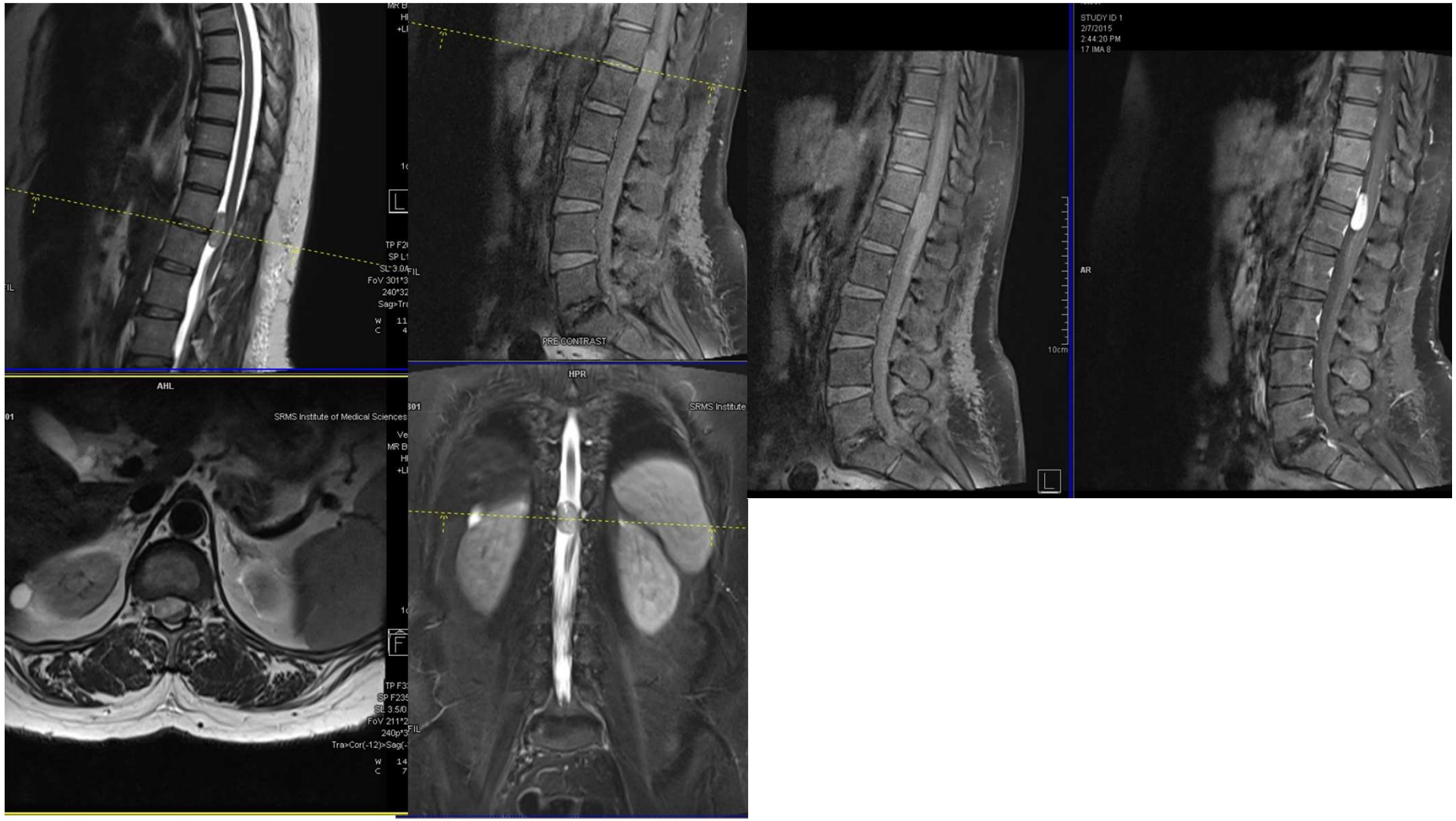
Intramedullary long segment lesion:
Ependymoma Vs Astrocytoma

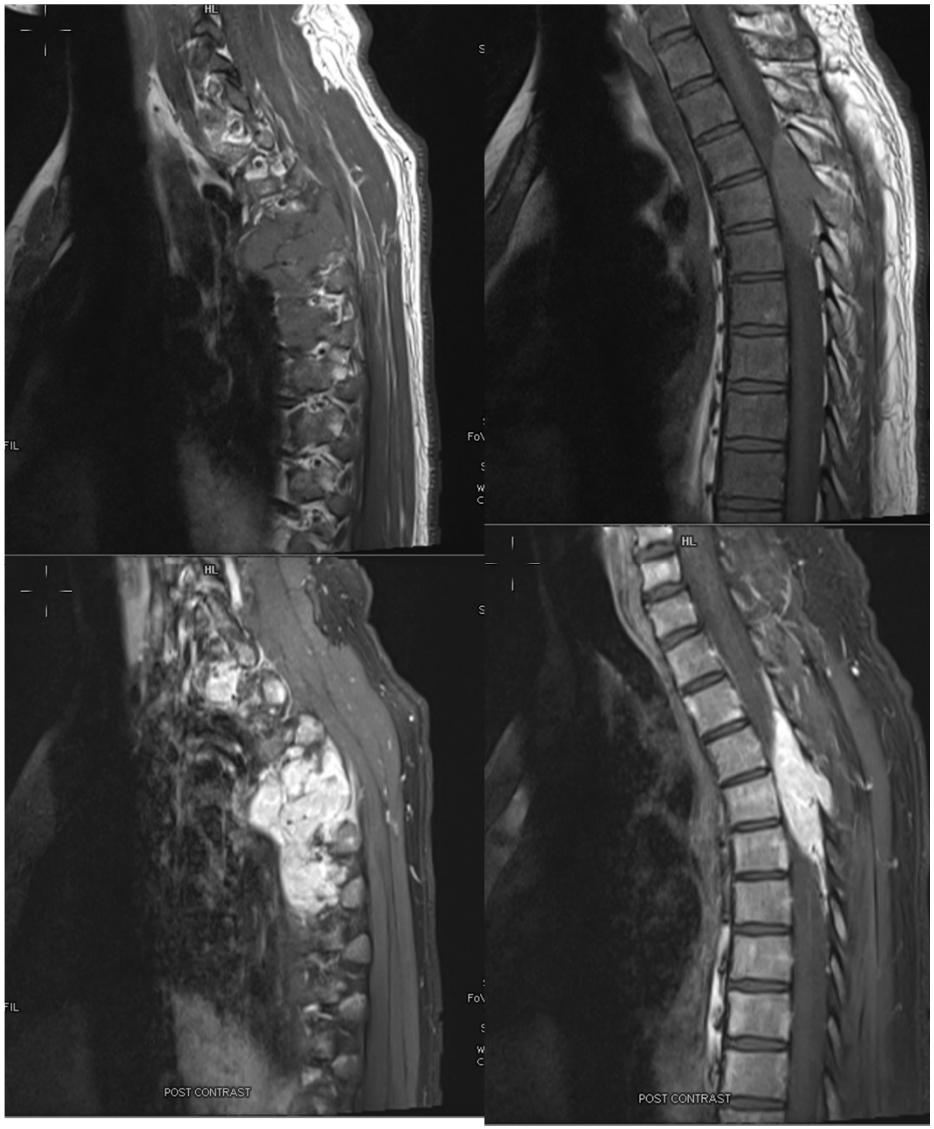
MYXOPAPPILARY EPENDYMOMA

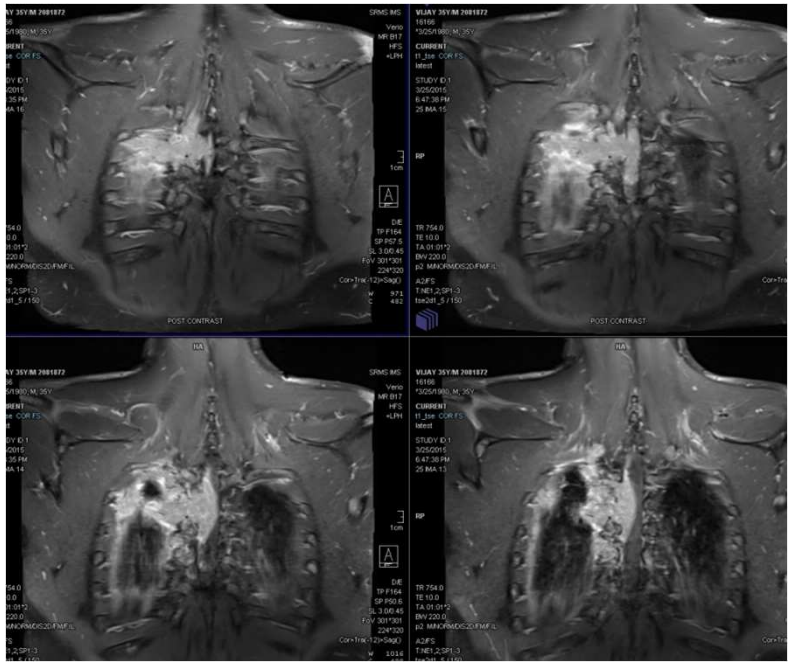


THE EXTENT OF LESION IS CLEAR IN POST CONTRAST STUDY.
INTENSE ENHANCEMENT MAKES POSSIBLE TO ARIVE D/D EPENDYMOMA/SCHWANOMMA

INTRADURAL SOL.. 55Yr female, DD: MENINGIOMA /SCHWANNOMA







EXTRADURAL LESION-35 YRS OLD MALE

D/D: LYMPHOMA
METS
PLASMACYTOMA

GO FOR FNAC/BIOPSY

THANKS