



KARNATAKA RADIOLOGY EDUCATION PROGRAM



Case of Bilateral Cerebral Abscess

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DEPT OF RADIODIAGNOSIS

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INTRODUCTION

- ▶ A **cerebral abscess** is a focal area of necrosis starting in an area of cerebritis surrounded by a membrane.
- ▶ It is a potentially life-threatening condition requiring prompt radiological identification and rapid treatment.
- ▶ Fortunately, MRI is usually able to convincingly make the diagnosis, distinguishing abscesses from other ring-enhancing lesions.

Mnemonics for the causes of cerebral ring-enhancing lesions are:

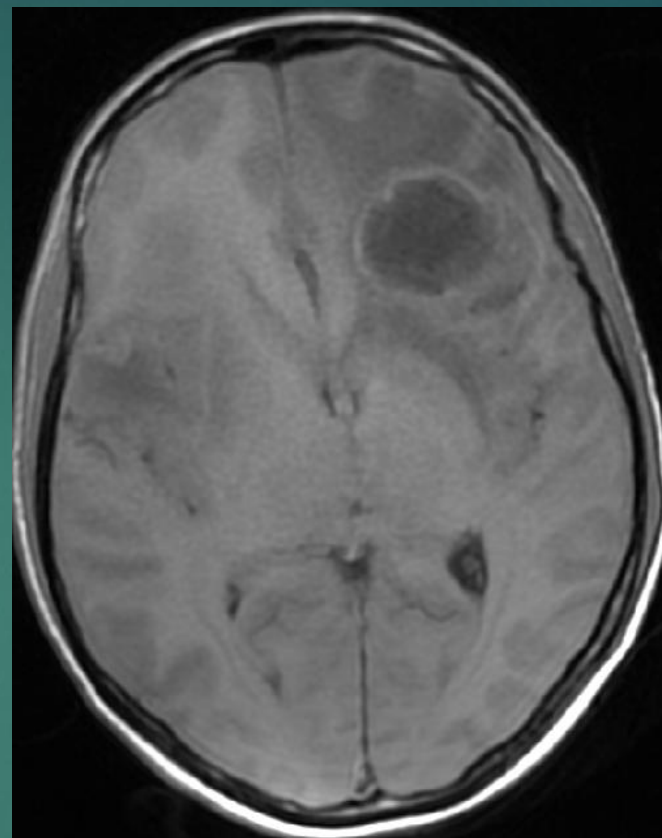
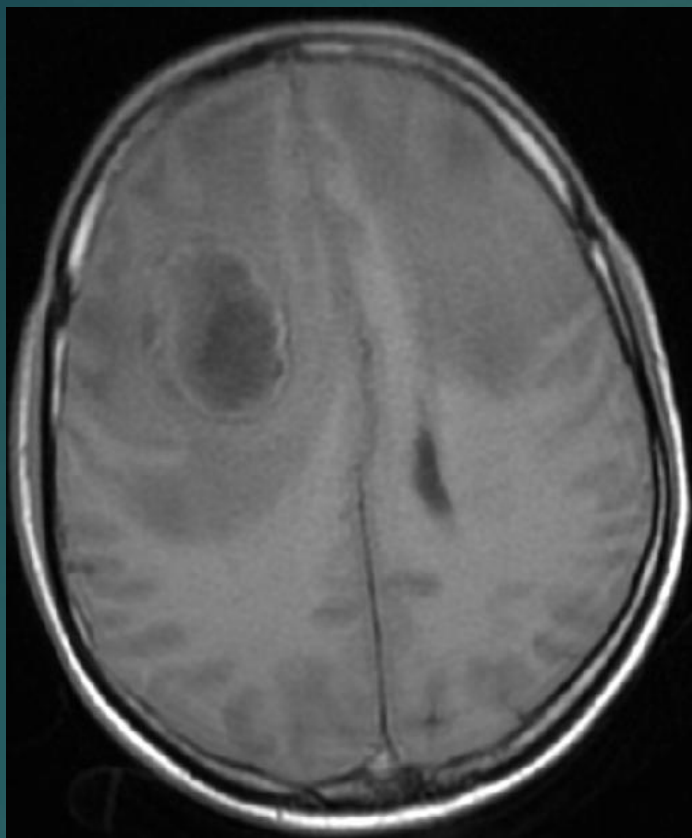
- **M:** metastasis
- **A:** abscess
- **G:** glioblastoma
- **I:** infarct (subacute phase) or inflammatory (neurocysticercosis, tuberculoma)
- **C:** contusion
- **D:** demyelinating disease (classically incomplete rim of enhancement)
- **R:** radiation necrosis or resolving hematoma

Clinical details

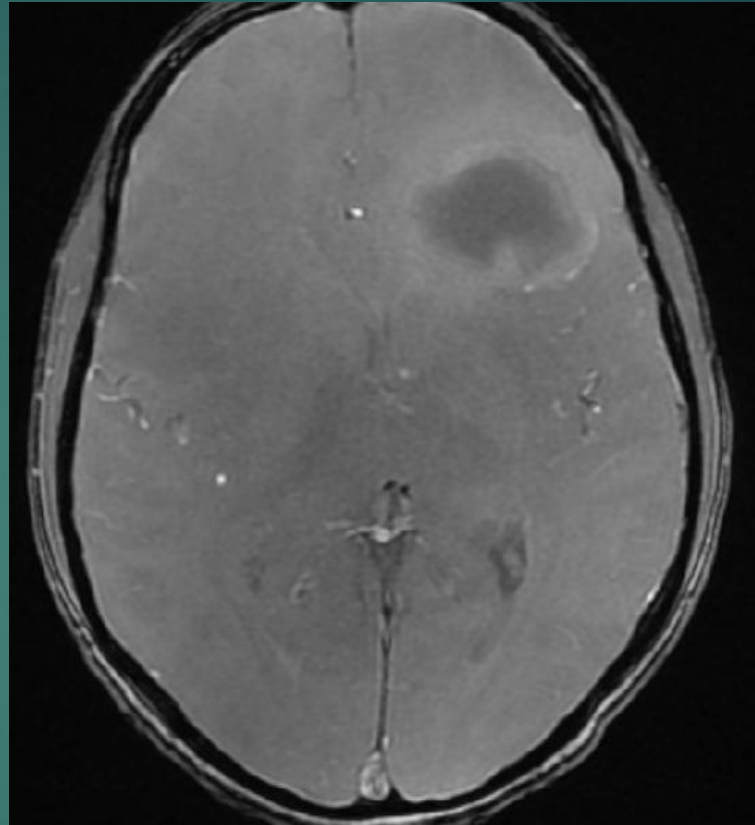
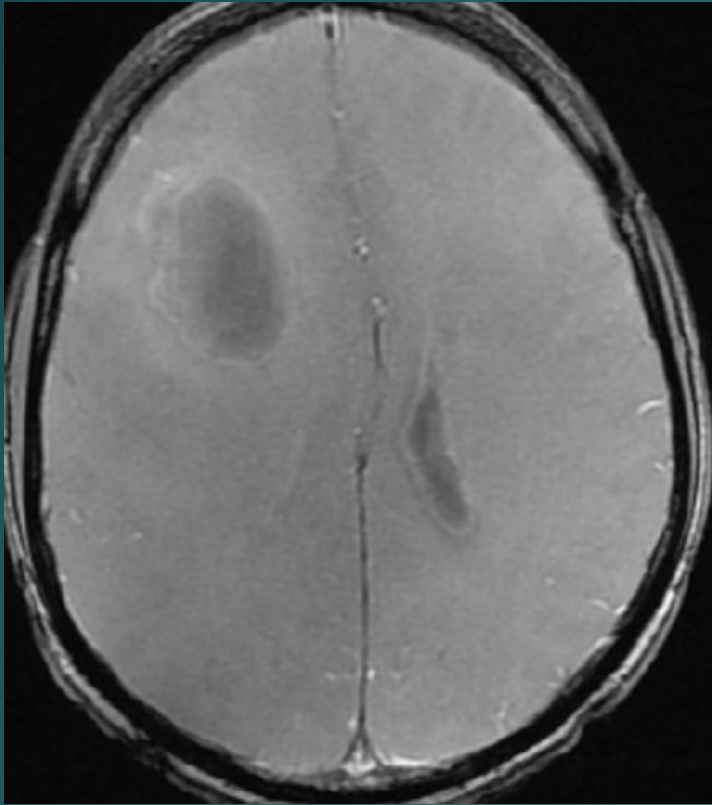
- ▶ A 49 year old man who is a K/C/O RVD, came to emergency with complaints of headache, decreased speech output and difficulty in walking.
- ▶ MRI Brain was done for further evaluation.

Imaging findings

- Two well-defined T1 hypointense, T2/FLAIR heterogeneous iso-hyperintense lesions in bilateral frontal lobes.
- (Right side – involving frontal lobe and basal ganglia & left side - involving anterior and basi-frontal lobe), measuring 4.2 x 3.2 x 3.3cms on right side and 3.4 x 4.2 x 4.7cms on left side.
- The lesions show thick irregular wall which appear hypointense on T2 and hyperintense on T1 & MT T1 sequence.
- The lesions show central true diffusion restriction.
- On post contrast study thick irregular complete rim enhancement noted (wall irregularity is maximum at lateral wall of the lesions).
- Spectroscopy reveals Lipid lactate peak.
- Significant T2/FAIR hyperintense perilesional edema noted causing mass effect in the form of effacement of frontal horns of bilateral lateral ventricles noted.
- Sulci in bilateral cerebral hemisphere, sylvain fissure and basal cisterns are effaced - Diffuse cerebral edema.

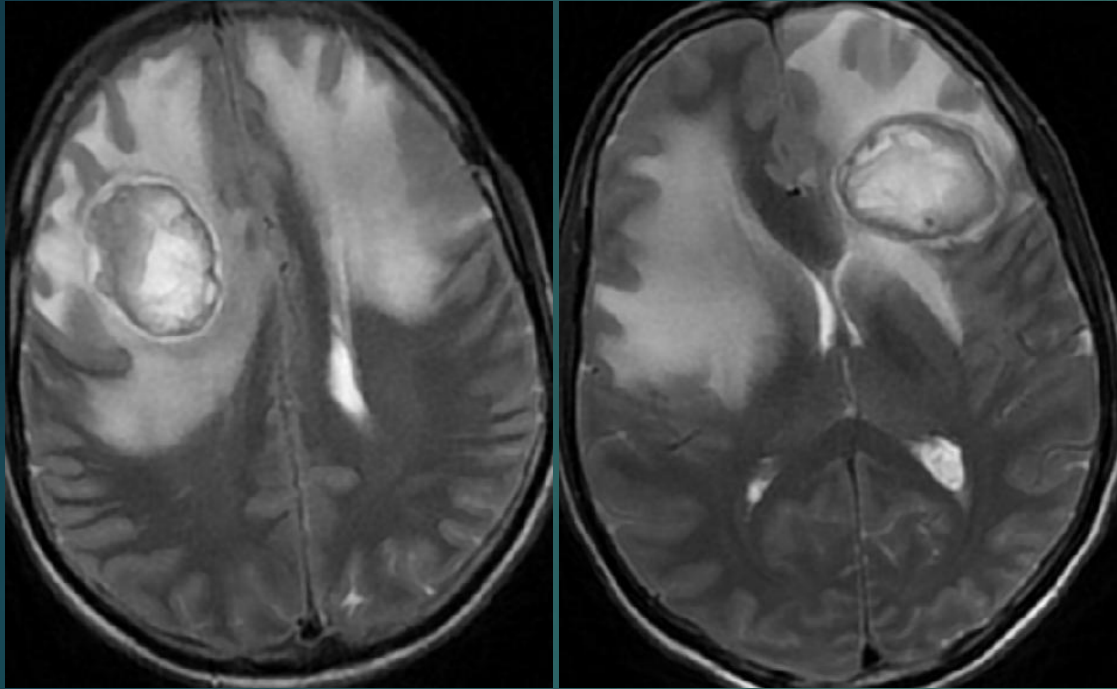


T1- hypointense

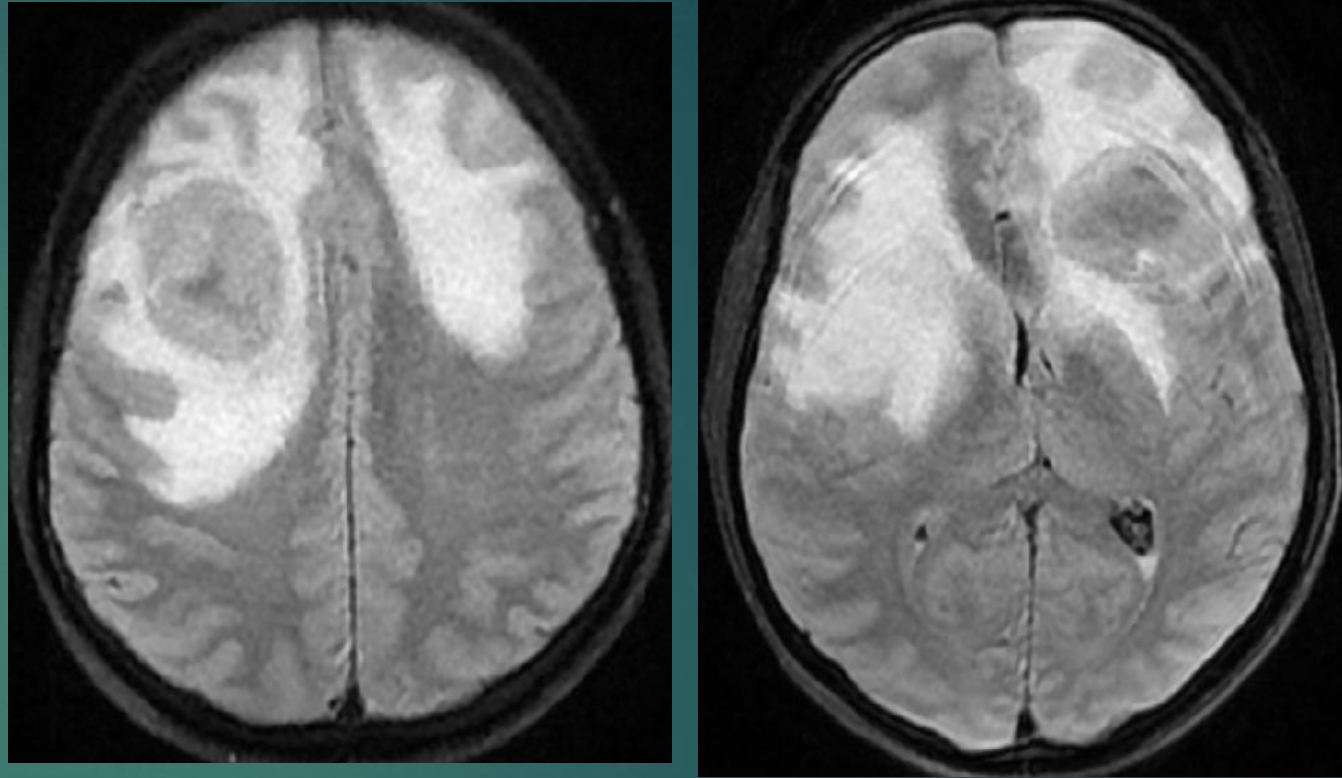


T1MT

T2



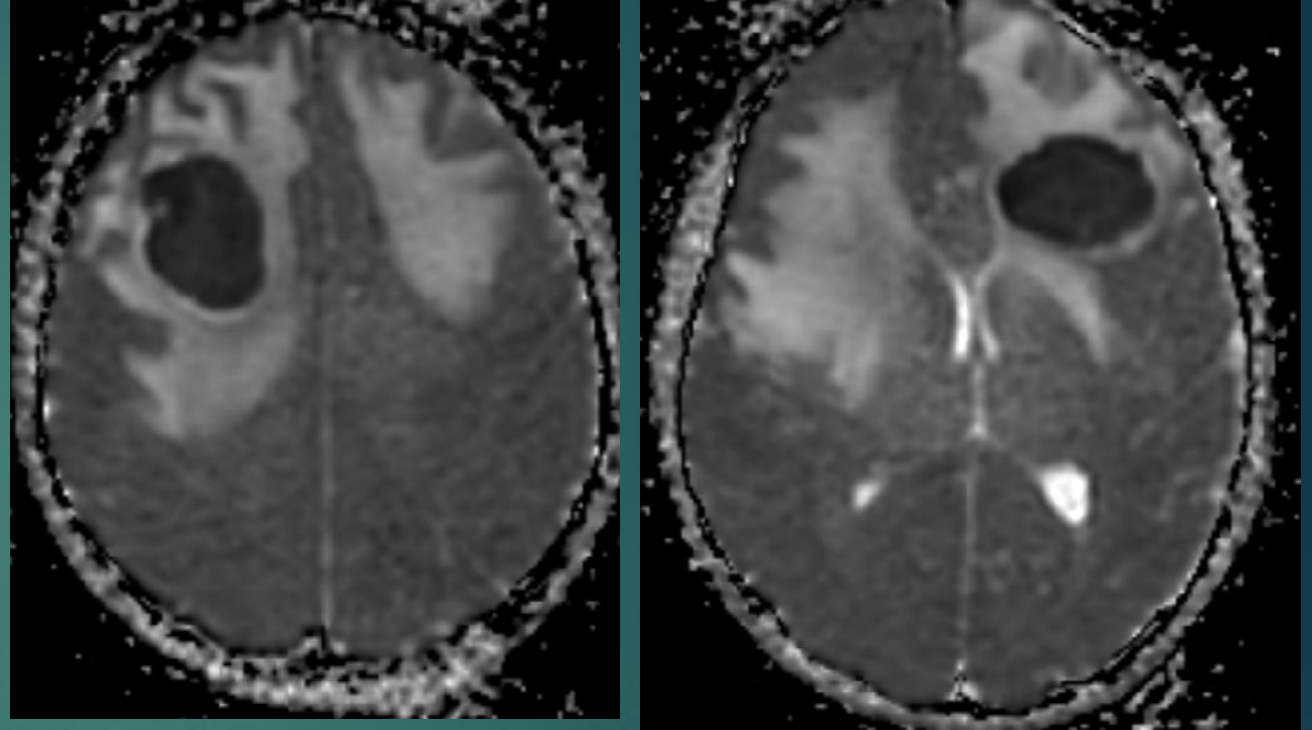
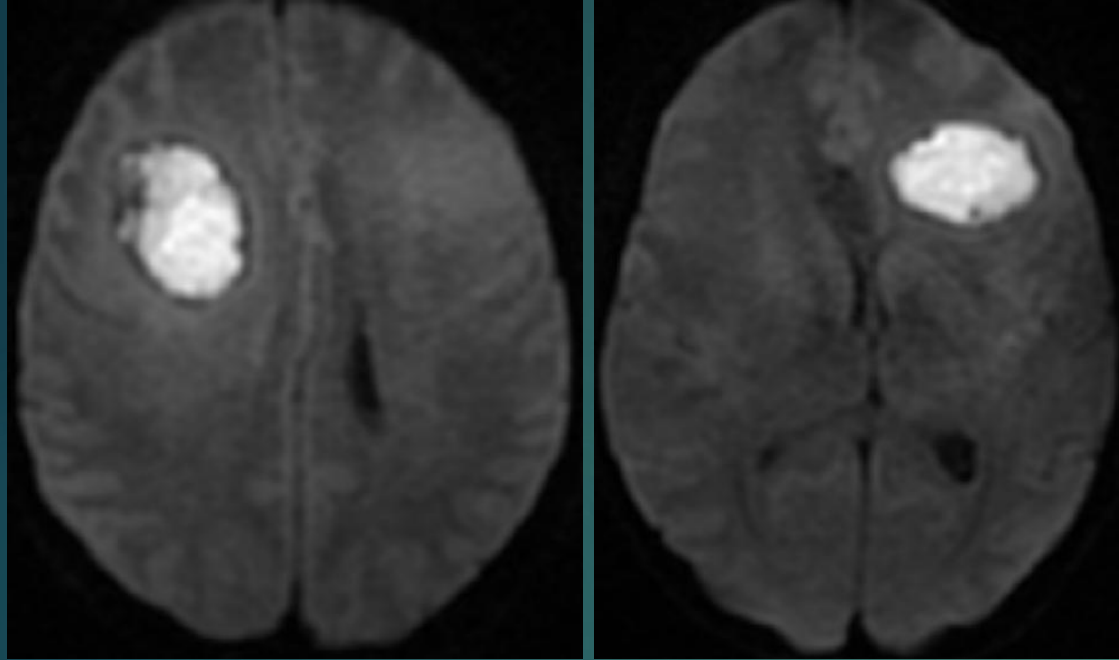
FLAIR



T2/FLAIR – heterogenous iso-hyperintense
with significant perilesional edema,
Mass effect on bilateral frontal horns of lateral ventricles
Diffuse cerebral edema - Sulci in bilateral cerebral hemisphere, sylvain
fissure and basal cisterns are effaced

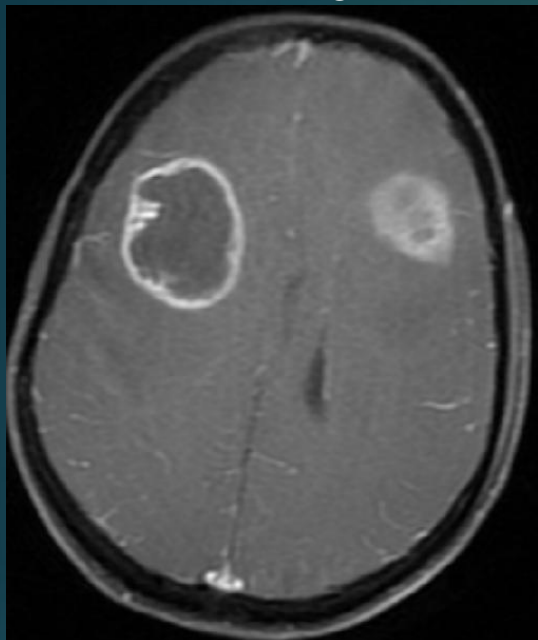
DWI

ADC



True diffusion restriction

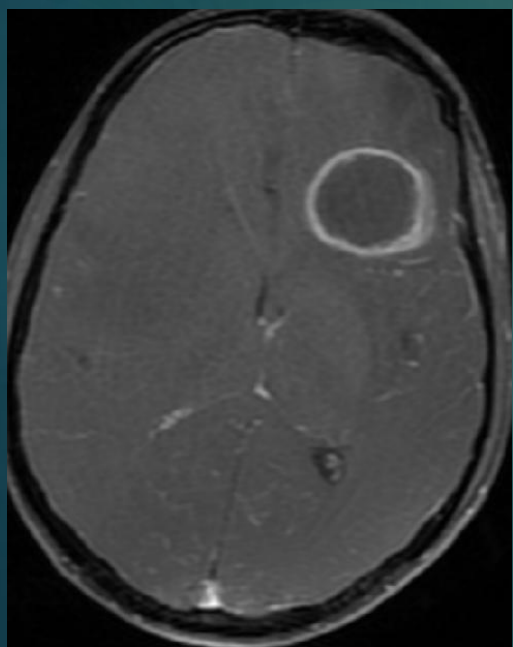
Axial



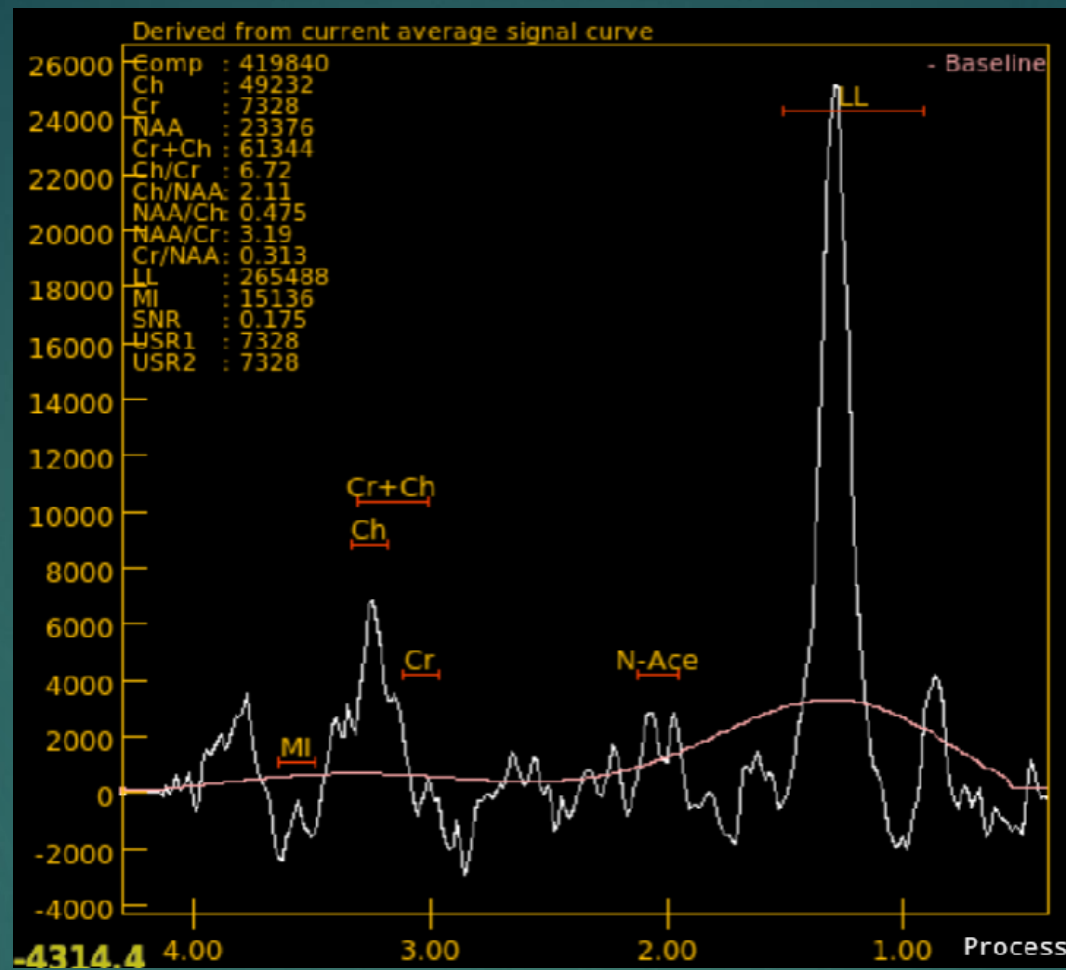
Sagittal



Coronal



T1 post contrast – complete rim enhancement



MR Spectroscopy - **Lipid lactate peak**

Discussion

- ▶ Cerebral abscesses result from pathogens growing within the brain parenchyma. Initial parenchymal infection is known as cerebritis, which may progress into a cerebral abscess.
- ▶ Historically direct extension from sinus or scalp infections was the most common source.
- ▶ More recently, hematological spread has become most common. Direct introduction by trauma or surgery accounts for only a small minority of cases.
- ▶ Cerebral infection is commonly divided into four stages with distinct imaging and histopathologic features:
 1. **early cerebritis** (a focal infection without a capsule or pus formation, can resolve or develop into frank abscess).
 2. **late cerebritis**.
 3. **early abscess/encapsulation** - may occur 10 days after infection.
 4. **late abscess/encapsulation** - may occur >14 days after infection.

Thank You.