KREP

2025

KARNATAKA RADIOLOGY EDUCATION PROGRAM

CASE PRESENTATION

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JJMMC,DAVANGERE

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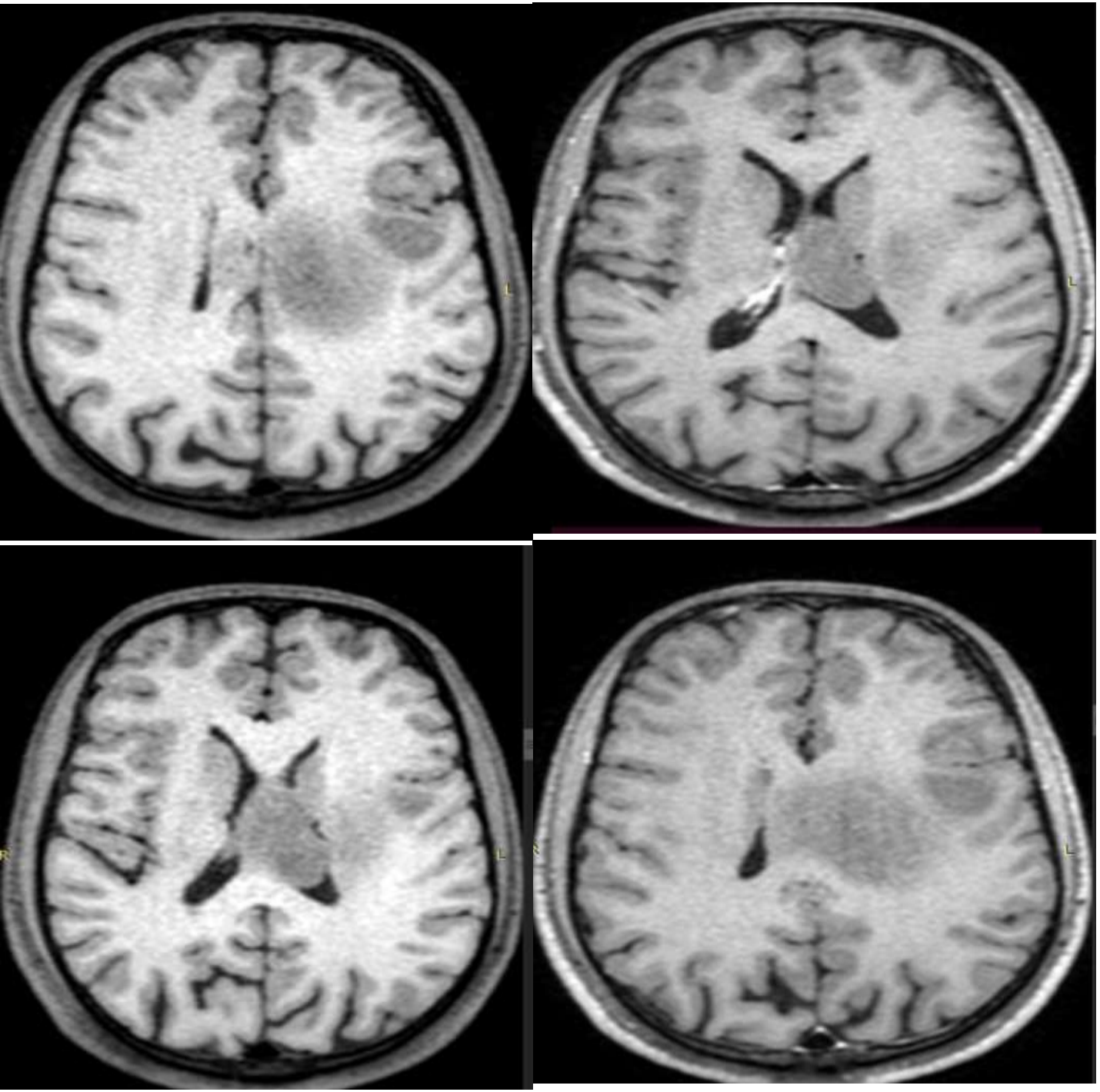
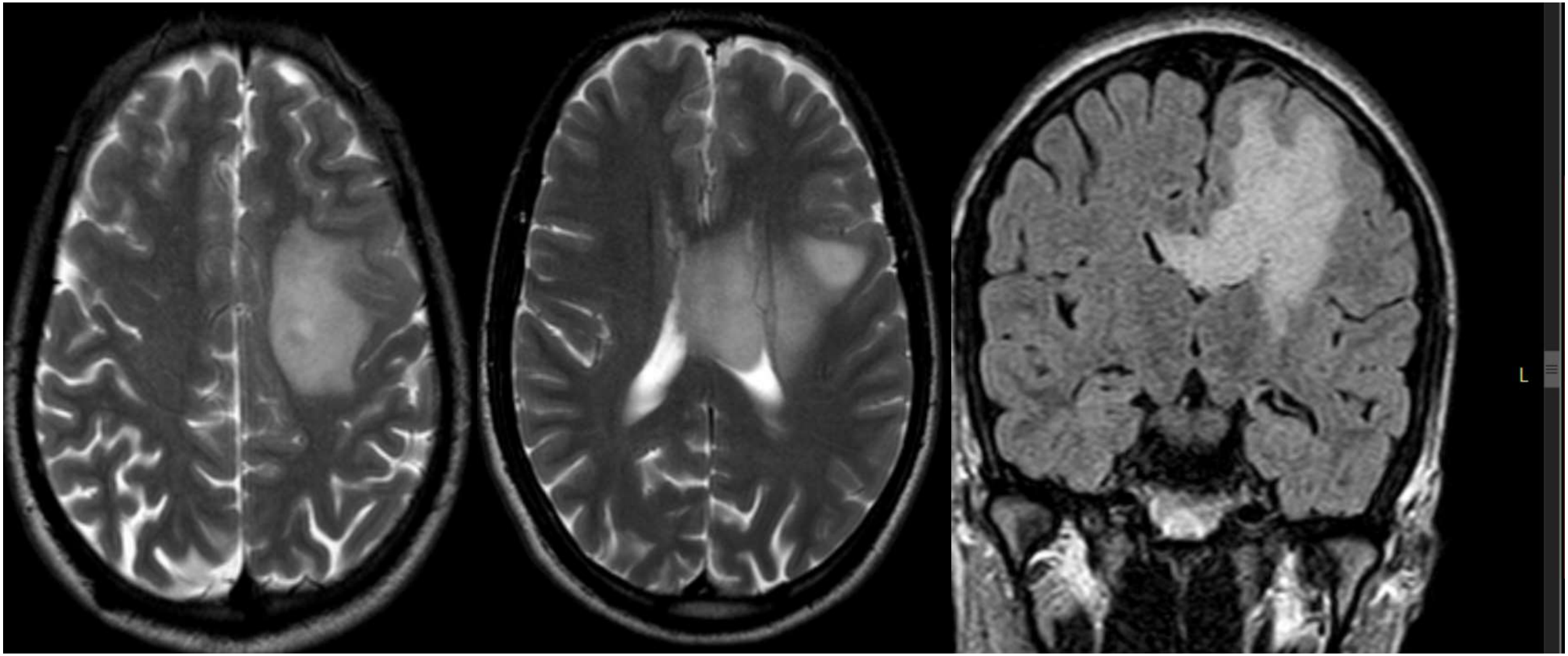
History

- A 25-year-old male
- Came with a history of acute-onset giddiness and weakness in the right upper limb that began 20 days ago. reportedly.
- The weakness was predominantly distal, characterized by difficulty gripping objects, holding a pen, performing fine motor tasks, and a noticeable loss of dexterity.
- No similar complaints in the past, as well as any systemic symptoms such as fever, headache, seizures, or visual disturbances.
- There was no history suggestive of cranial nerve involvement, gait disturbances, or bladder or bowel dysfunction.
- His past medical history was unremarkable, with no significant illnesses or surgeries
- No family history of neurological or hereditary disorders.
- no substance abuse, including alcohol or tobacco use.

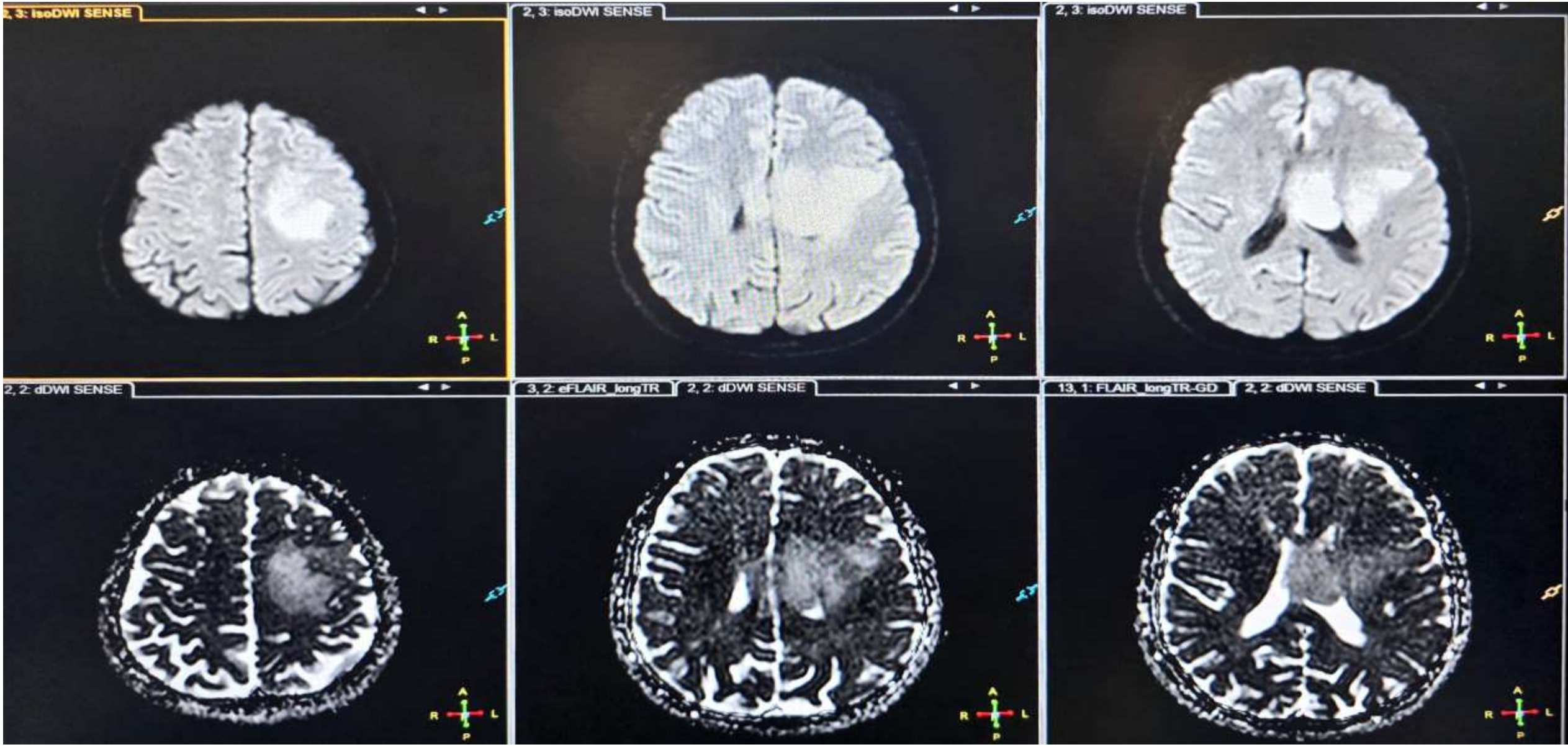


Fairly well defined intra-axial large mass lesion measuring approx. 5.3 x 4.4 x 5.8 cm (CCxAPxTR) noted epicentered in deep white matter of left frontal lobe with infiltration to the corpus callosum, left putamen, left posterior limb of internal capsule, left centrum-semiovale, left corona-radiata, sub-cortical region which is hypointense on T1

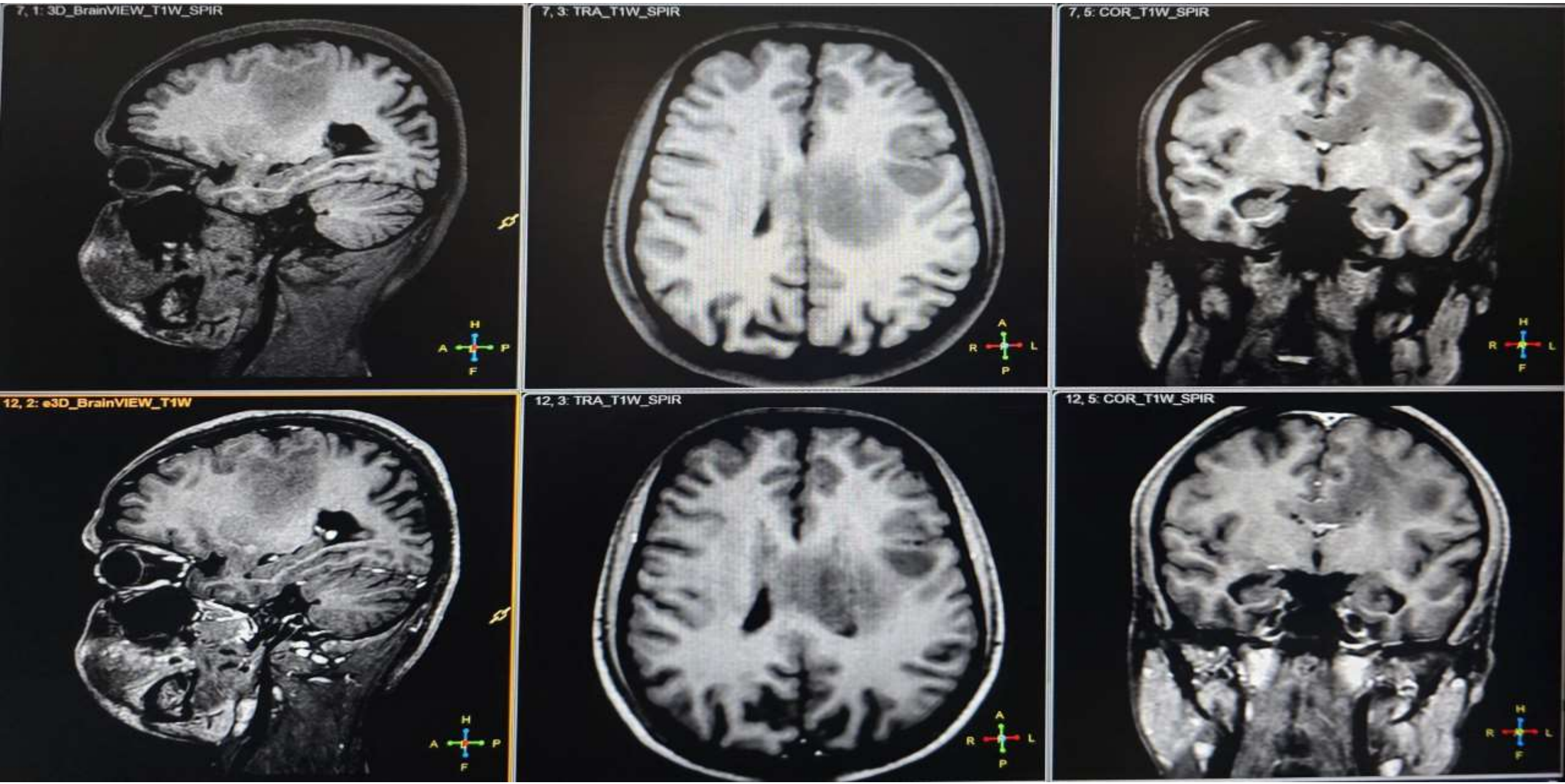
Lesion is showing mass effect in form of compression of body and frontal horn of ipsilateral lateral ventricle, adjacent cortical sulci, and left basal ganglia with midline shift of 4.2mm to right.



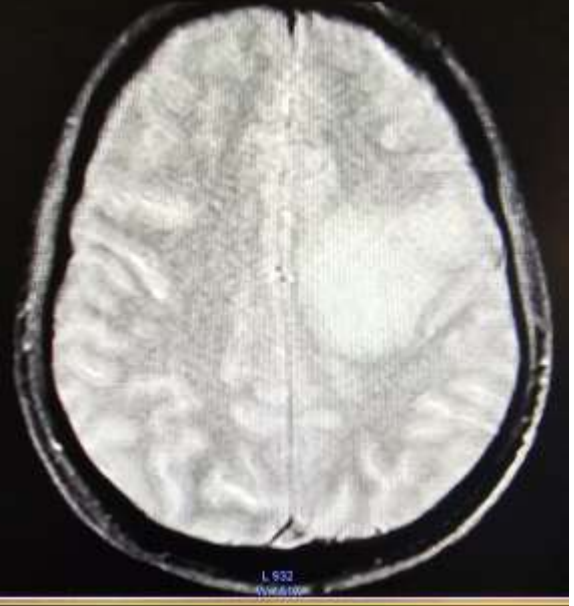
These are the corresponding sections of the T2 and flair images the lesion appears hyper intense on T2/FLAIR and showing perilesional edema.

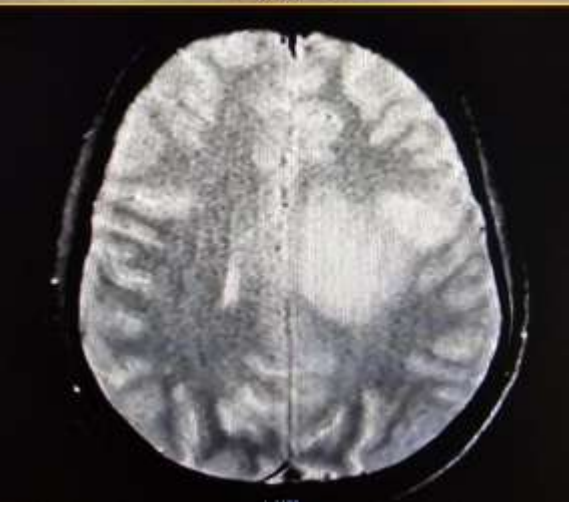


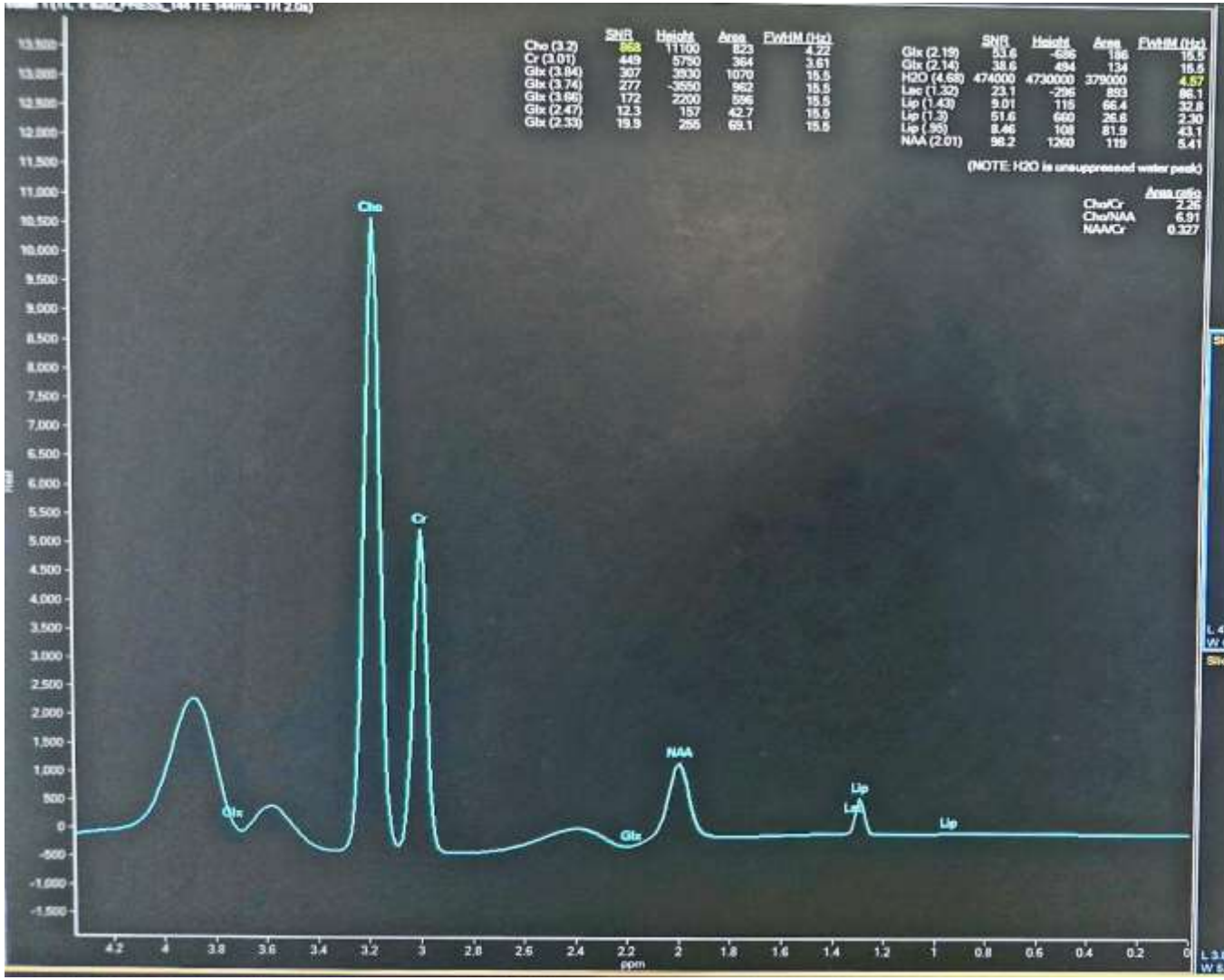
These are the corresponding sections of DWI and ADC imaging no true diffusion restriction

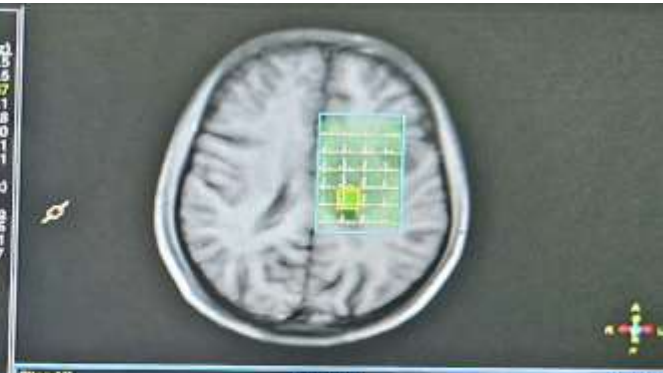


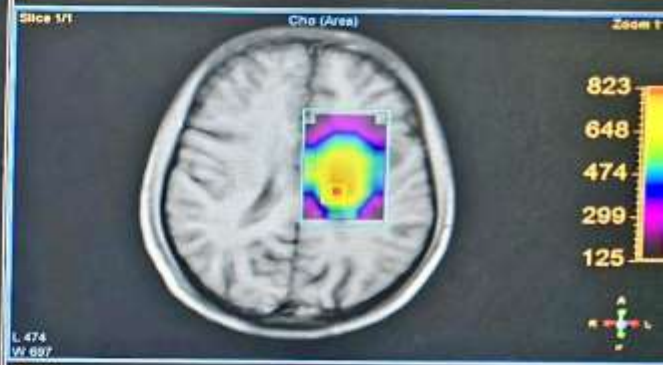
These are the post contrast images not showing enhancement on post contrast T1FS.

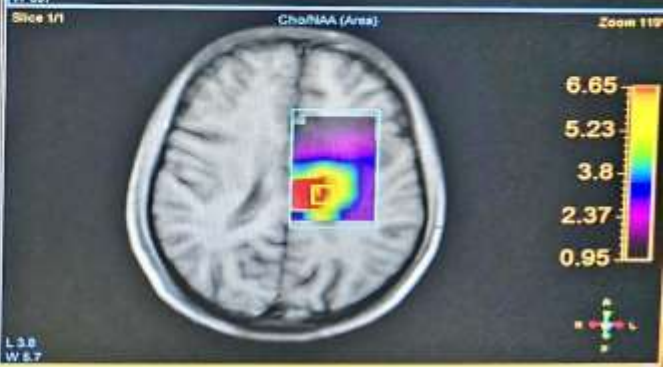












No blooming is noted on the GRE

- **<u>On MRS:</u> Choline peak noted at 3.2ppm. Cho/Cr ratio: 2 . Decreased NAA peak at 2ppm.**

IMPRESSION

- **Fairly well defined intra-axial large mass lesion noted epicentered in deep white matter of left frontal lobe with infiltration to the corpus callosum, left putamen, left posterior limb of internal capsule, left centrum-semiovale, left corona-radiata, sub-cortical and middle frontal gyrus of left frontal lobe which is hypointense on T1 and hyperintense on T2/FLAIR and no true diffusion restriction with no enhancement on post contrast T1FS. No blooming on GRE sequence.**
- **The lesion is associated with extensive perilesional edema. There is associated mild mass effect in form of compression of body and frontal horn of ipsilateral lateral ventricle, adjacent cortical sulci, and left basal ganglia with midline shift of 4.2mm to right.**

➔ F/S/O ***Diffuse/Anaplastic astrocytoma of left frontal lobe – WHO grade II/III, IDH mutant.***

Adult Astrocytomas

In contrast to astrocytomas in children, astrocytomas in patients over the age of 30 years are mostly supratentorial and occur primarily in the cerebral hemispheres **(17-4)**.

PAs are rare in adults. Most adult astrocytomas are IDH-mutant astrocytomas. In general, the older the patient, the higher the tumor grade. **IDH-mutant diffuse astrocytoma** (WHO grade II) predominates in young adults, whereas **anaplastic astrocytoma** (WHO grade III) and **glioblastoma** (WHO grade IV) are much more common in middle-aged and older adults.

IDH-MUTANT DIFFUSE ASTROCYTOMAS

Terminology
- Formerly known as low-grade astrocytoma or diffuse astrocytoma

Etiology
- Glioma cancer stem cell
- Molecular genetics
  - *IDH1/2* mutated
  - *ATRX* loss, *TP53* mutated
  - 1p/19q intact (not codeleted)

Pathology
- Supratentorial (frontal lobe most common)
- Infiltrating, ill-defined borders
- WHO grade II
- Inherent tendency to undergo malignant degeneration

Clinical Issues
- 10-15% of astrocytomas
- Age = 20-45 years (mean = 38 years)
- Overall survival = 11 years
  - Not significantly different from IDH+ anaplastic astrocytoma

IMAGING OF IDH-MUTANT DIFFUSE ASTROCYTOMAS

Imaging Features
- Hypodense on NECT
- No enhancement on CECT
- Hypointense on T1, hyperintense on T2/FLAIR
- No enhancement, hemorrhage
- MRS shows 2-HG peak at 2.25 ppm
- Low rCBV
  - Foci of ↑ rCBV suspicious for malignant degeneration

Differential Diagnosis
- IDH-mutant anaplastic astrocytoma
- Pilocytic astrocytoma
- Oligodendroglioma
- Nonneoplastic
  - Acute cerebral ischemia-infarction
  - Encephalitis

IDH-MUTANT ANAPLASTIC ASTROCYTOMA

Etiology
- Begins as IDH-mutant diffuse (grade II) astrocytoma
  - Undergoes malignant degeneration to anaplastic astrocytoma
  - Maintains IDH-mutant status throughout life

Pathology
- WHO grade III
  - IDH-mutant, *ATRX* loss
- Hypercellular
- Anaplasia
- Increased mitoses
  - Ki-67 5-10%
- Lacks necrosis, microvascular proliferation

Clinical Issues
- Overall survival not statistically different from IDH-mutant diffuse astrocytoma (DA)

Imaging
- Similar to IDH-mutant DA
- Enhancing focus, any areas of ↑ rCBV likely represent malignant degeneration

ANAPLASTIC ASTROCYTOMA, IDH-WILD-TYPE

Etiology, Pathology
- Same glial progenitor line as IDH-DA, GBM
- Can exhibit widespread infiltrative pattern
  - Most cases of "gliomatosis cerebri" are IDH-AAs
  - No longer separate diagnosis
- Degeneration into IDH-wild-type GBM, often in < 2y

Imaging
- Widespread, infiltrative T2/FLAIR hyperintense
- Often exhibits foci of contrast enhancement