



Case Series on Imaging Patterns in Toxic and Metabolic Encephalopathies.

Category / Sub Specialty: Neuroimaging – Case series

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Aims and Objectives

1. To examine the spectrum of magnetic resonance imaging (MRI) characteristics of several common metabolic and a few uncommon toxic encephalopathies.
2. To explain very specific imaging results for a specific toxic and metabolic encephalopathy

Methods and Materials

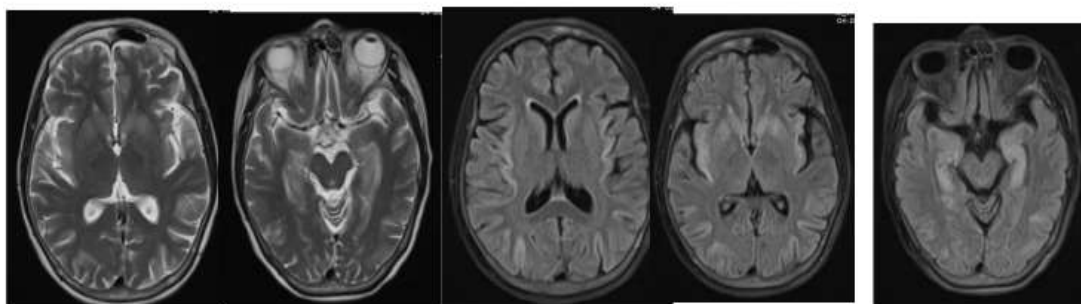
- A series of descriptive case studies was completed in retrospect. We looked at MR scans of individuals who had a clinical suspicion of toxic and metabolic encephalopathies and who had presented to the Department of Radiodiagnosis at AJ Institute Of Medical Sciences and Research Centre, Mangalore. From hospital records, a thorough

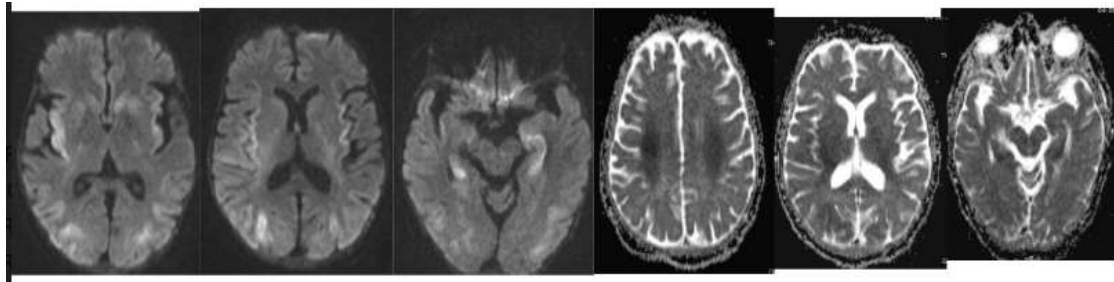
clinical history, physical examination, and laboratory work-up were obtained.

- Image analysis comprised:
- Gray matter sparing and symmetric periventricular white matter involvement.
- Involvement of the central pons, corpus callosum, and/or basal ganglia.
- Vasogenic edema of parieto-occipital subcortical area.

Hypoglycemic encephalopathy

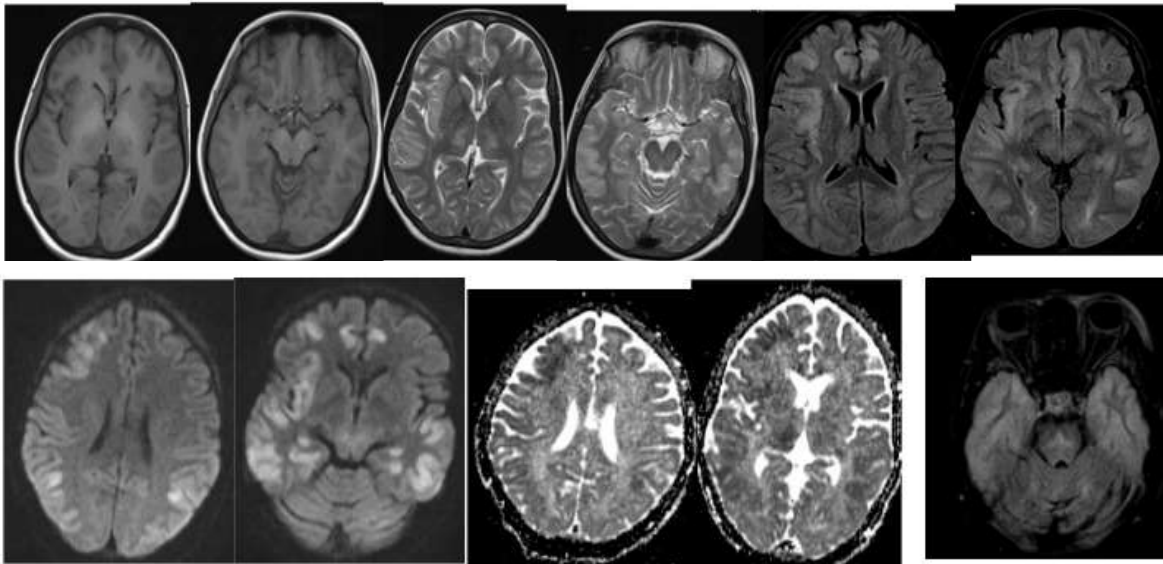
- A 43-year-old male patient comes with history of unresponsiveness since 2 days.
- On examination: Blood glucose level was 45mg/dl - s/o hypoglycemia.
- All other parameters were normal
- MRI brain scan was done following are the imaging features:





- Symmetric bilateral T2/FLAIR hyperintensities noted in parieto-occipital cortex, insular cortex, medial temporal lobes, hippocampus, head of caudate nuclei and putamen, showing diffusion restriction on DWI.
- T2/FLAIR hyperintensities noted in bilateral corona radiata and centrum semiovale, showing diffusion restriction on DWI.
- Features consistent with hypoglycemic encephalopathy.
- A known case of chronic liver disease presented with h/o altered sensorium and 2 episodes of GTCS seizures.
- On examination vitals were stable, GCS was E1V1M1.
- Blood investigations showed deranged liver function test, with very high levels of serum total bilirubin, direct bilirubin, indirect bilirubin levels, SGOT and SGPT levels.
- MRI brain scan was done following are the imaging features:

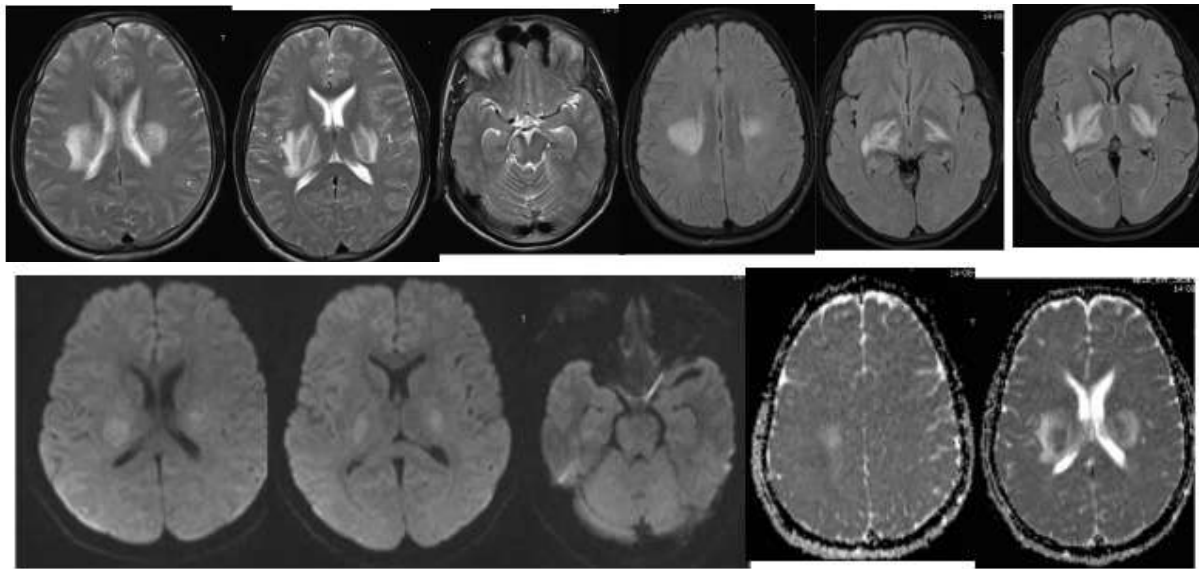
Hepatic encephalopathy



- T1 hyperintensity noted in globus pallidus and substantia nigra bilaterally
- T2/FLAIR hyperintensities noted in bilateral thalami, pons, bilateral cerebral gyri, bilateral insular cortex and bilateral hippocampi (R>L).
- Ill-defined T1 hypo and T2/FLAIR hyperintensity showing no diffusion restriction on DWI noted within the pons
- **Features consistent with hepatic encephalopathy.**

Uremic encephalopathy

- A 34-year-old male patient was found unconscious in his home. He was a known case chronic kidney disease on regular dialysis, but patient has skipped 3 cycles of dialysis.
- On blood investigations, there was raised blood urea and creatinine level.
- MRI brain scan was done following are the imaging features:

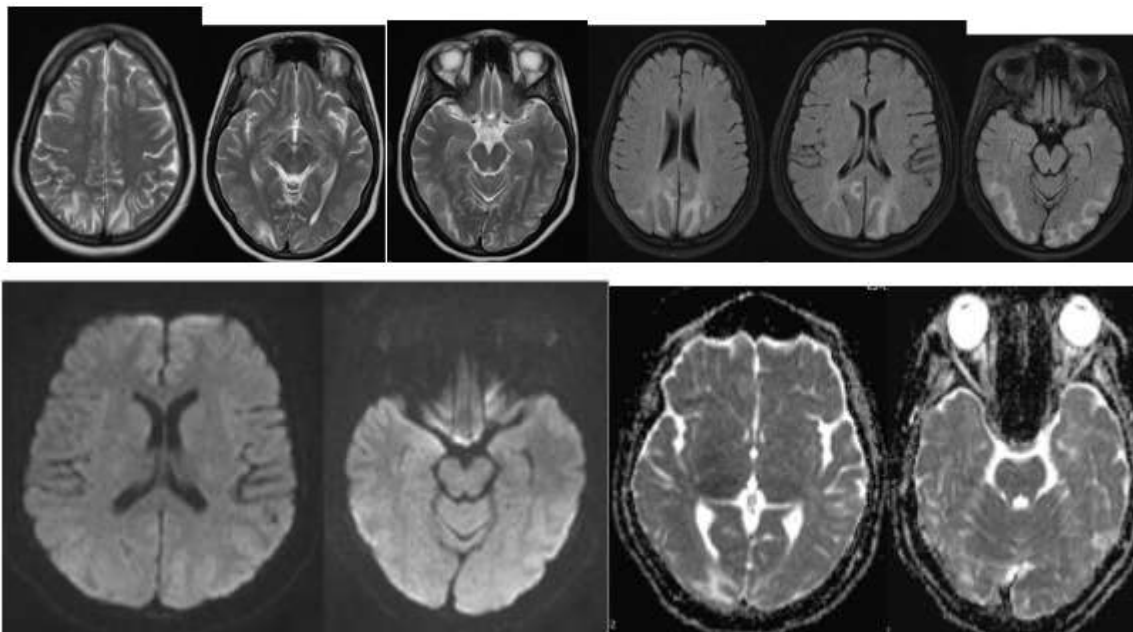


T2/FLAIR symmetric hyper intensities, T1 hypointense noted in bilateral corona radiata, capsuloganglionic region, thalamus, centrum semiovale, bilateral mesial temporal lobes, parahippocampal gyri, midbrain, right external capsule, right side of midbrain right cerebral peduncle and pons showing diffusion restriction on DWI in bilateral globus pallidus.

- **F/S/O Uremic encephalopathy.**

Posterior reversible encephalopathy syndrome (PRES)

- An 18-year old female patient P2L2 diagnosed as pre-eclampsia during her third trimester, now in post-natal day 2, she presented with severe headache for 2 days, not relieving on medication.
- On examination: Present blood pressure was 190/110 mm of hg.
- Patient referred to Department of Radiodiagnosis for plain MRI brain imaging features are as follows:



- Symmetrical T2/FLAIR hyperintensities noted in Bilateral parieto-occipital lobes and these

areas shows diffusion restriction on DWI images.

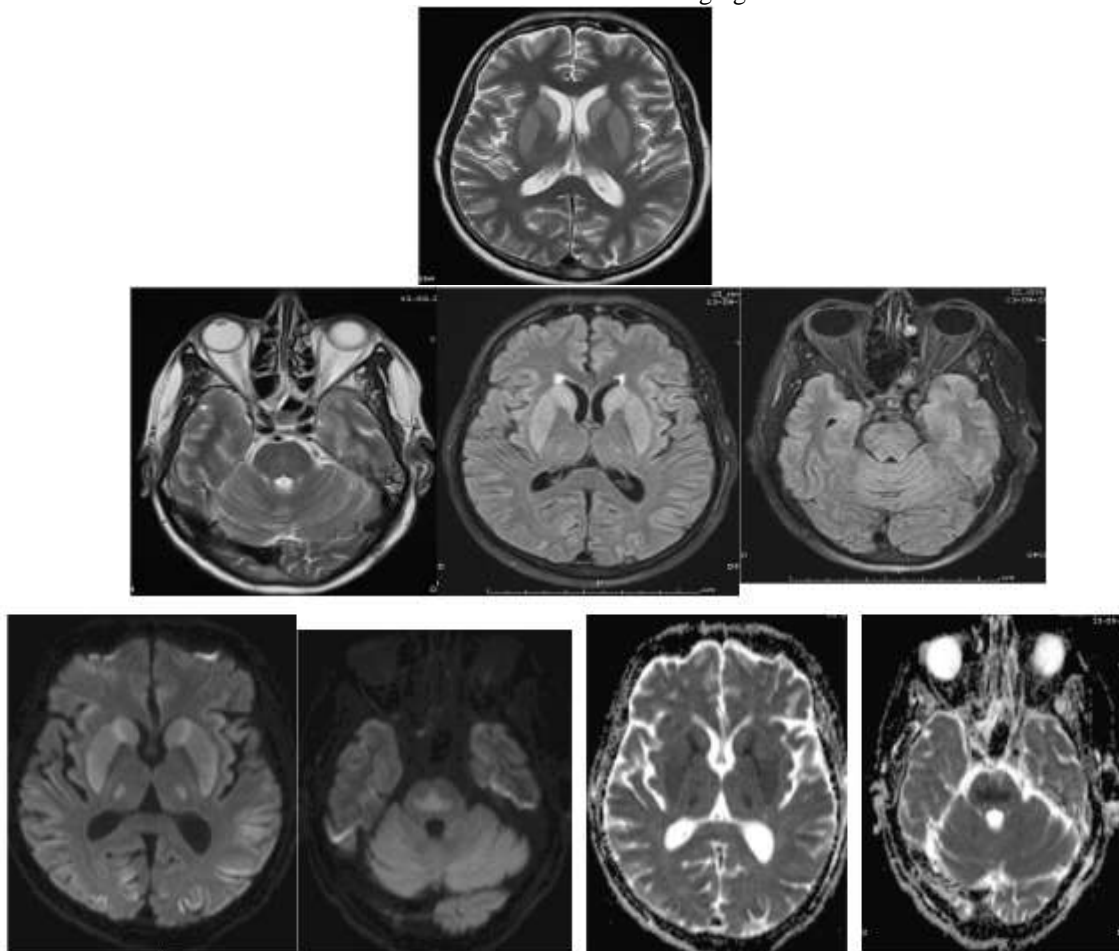


- Features are suggestive of **Posterior reversible encephalopathy syndrome (PRES)**

Pontine and extra pontine myelinolysis

- A 46-year-old male patient with history of hemicolectomy 20 days back for perforation of colon presented with altered sensorium.

- Serum electrolytes: Serum sodium level-49 mg/dl, serum potassium level: 4.1-Normal.
- Patient was diagnosed with hyponatremia.
- Patient is been subjected sodium correction, however there is deterioration of symptoms of altered sensorium, loss of consciousness and patient has been intubated.
- MRI brain scan was done following are the imaging features:



Symmetrical T2/FLAIR hyperintensities in central pons with relative sparing ventrolateral pons, bilateral

caudate nucleus, lentiform nucleus and ventrolateral thalamus, showing diffusion restriction on DWI

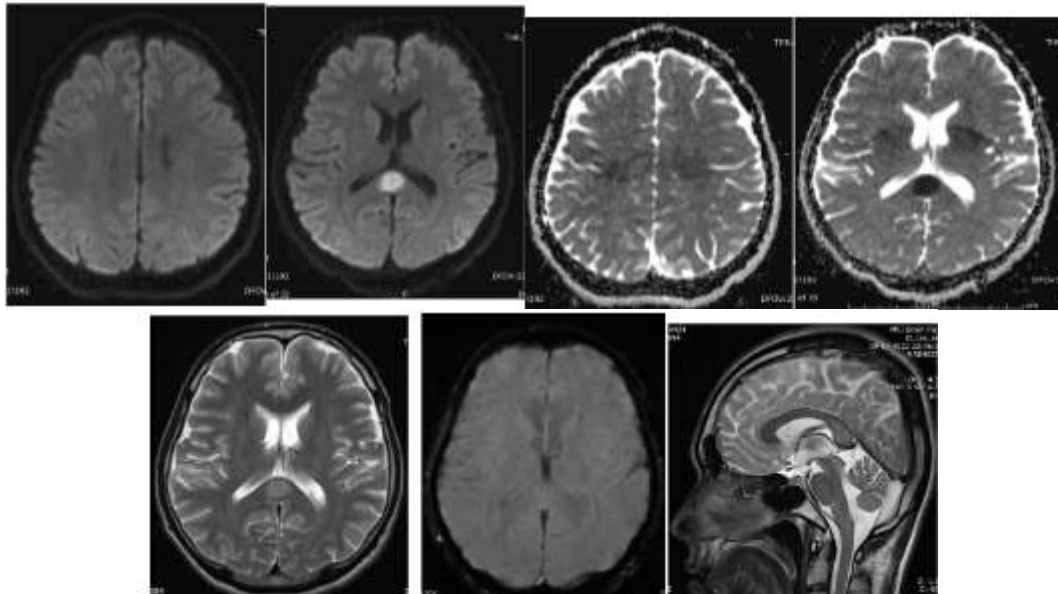
- Gyriiform cortical diffusion restriction in bilateral frontal, parietal and occipital lobes.

-Overall features suggestive of osmotic demyelination syndrome-Pontine and extra pontine myelinolysis with possibility of Superadded Hypoxic insult to be considered.

Cytotoxic Lesion Of Corpus Callosum (CLOCC)

(Reversible Splenial Lesion)

- A 28-year-old male patient with history of 15 tablets of phenytoin intake comes with history of altered sensorium followed by loss of consciousness.
- All blood parameters were normal.
- Patient is diagnosed with phenytoin toxicity.
- Patient referred to department of Radiodiagnosis for plain MRI brain imaging features are as follows



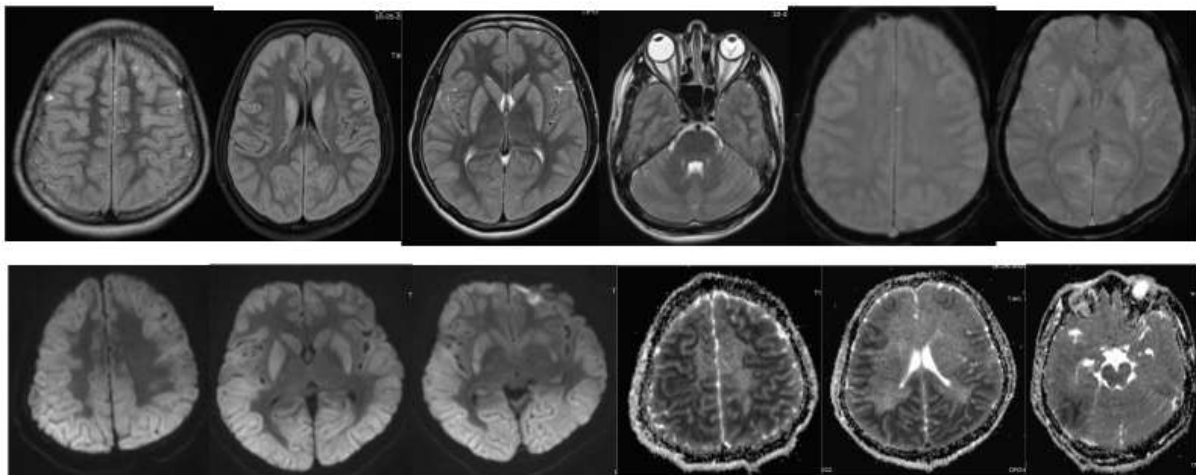
T2/FLAIR hyperintensity showing restriction on DWI with low ADC values and no blooming on SWI sequence noted in splenium of corpus callosum. – **Likely cytotoxic lesion of corpus callosum (CLOCC)*.**

Snake Bite Induced leukoencephalopathy

• A 51-year-old male with no known comorbidity presented to ER with alleged history of snakebite to right foot. At the time of presentation patient was in altered sensorium with GCS E1V1M1, pupils were equal, sluggishly reactive, other vitals were normal. Swelling of left dorsum of foot

present. Deep tendon reflexes were absent and plantars were mute. Patient was intubated and was given 20 vials of anti-snake venom, 5 cycles of atropine and neostigmine, intravenous antibiotics and other supportive treatment. However, patient's condition did not improve. Over the course patient developed shock and was started on inotropes. Brainstem reflexes were absent after 4 days of admission and patient succumbed after 10 days.

- WBCT clotted at 20minutes.
- aPTT, PT/INR-normal
- CSF analysis-within normal limits.



MRI brain-Diffuse symmetrical T2/FLAIR hyperintensity with areas of diffusion restriction noted in bilateral cerebral cortex, right half of

midbrain, corpus callosum, bilateral caudate nucleus and putamen.

- This rare case of encephalopathy following snake bite.



- Leukoencephalopathy is a rare complication of snake bite. Very few cases have been reported. Mechanism of leukoencephalopathy is not fully known, it is thought to be because of direct effects of toxin, however further studies are required.

DISCUSSION

- Toxic and metabolic disorders affecting the CNS usually manifest imaging characteristics and topographic distributions that should raise suspicion for such diagnoses when the clinical context is compatible.
- Bilateral and symmetric lesions with restricted diffusion, no or mild mass effects, and no enhancement are often depicted. In addition, sites with higher susceptibility include the cortical gray matter, deep gray nuclei, thalami, periventricular white matter, and corpus callosum. However, such manifestations are unspecific without adequate clinical context and can represent other conditions. Thus, correlations with clinical history are of particular importance in guiding imaging analysis.

• Major Causes of Toxic and Metabolic Disorders

• Most common endogenous metabolic derangements related to CNS involvement

Hypertensive encephalopathies
Glucose disorders
Parathyroid disorders
Hepatic encephalopathy (manganese and/or ammonia levels)
Uremic encephalopathy
Osmotic demyelination syndrome (ODS)
Cobalamin deficiency

• Major exogenous causes of toxic encephalopathy

Alcohol-related disorders (Wernicke encephalopathy, Marchiafava-Bignami disease)
Industrial agents (methanol, toluene)
Inhaled gases (carbon monoxide, pesticides)
Illicit drug use (heroin, cocaine)
Chemotherapeutic agents (methotrexate, fludarabine, 5-fluorouracil)
Immunosuppressive agents (TNF- α blockers, cyclosporine)
Other potentially neurotoxic medications (metronidazole, vigabatrin)

- Toxic and metabolic disorders are closely related to excitotoxic brain injury, as they often induce intense glutamate release. Although receptors related to excitotoxic injury are widely distributed in the brain, there

are classic CNS sites that are particularly susceptible to this mechanism, such as the basal ganglia and thalami, cortical gray matter, periventricular white matter, and the corpus callosum. This differential susceptibility is important because it indicates some possible characteristic imaging patterns that could lead to the consideration of toxic and metabolic causes during diagnosis.

• The most important patterns are as follows

1. Basal ganglia and/or thalami involvement. The periventricular white matter and the cortical gray matter may be also involved. This pattern is usually related to cytotoxic brain edema, poor outcomes, and irreversibility.
2. Dentate nuclei involvement.
3. Prominent cortical gray matter involvement. Although cortical lesions can coexist with basal ganglia- and white matter-associated involvement, they are the most distinguishing feature.
4. Symmetric periventricular white matter involvement with gray matter sparing. This is a pattern that includes ATL causes and is more related to intramyelinic edema and higher possibilities of reversibility and better outcomes.
5. Corticospinal tract region involvement.
6. Corpus callosum involvement.
7. Asymmetric white matter involvement in a demyelinating disease pattern.
8. Parieto-occipital subcortical vasogenic edema.
9. Central pons involvement.

CONCLUSION

- Diagnosing toxic and metabolic brain disorders remains challenging and refers to a heterogeneous group of diseases. Although many imaging manifestations are unspecific, some imaging findings can be quite specific for a few conditions.
- Approaching these conditions by considering the patterns at imaging can add to the radiologist's daily routine.
- Depending on the pattern, pathophysiologic information can be inferred and a reduced number of conditions in the differential diagnosis can be considered.
- One should always look for the accessible clinical history as it points to subjacent toxic and metabolic causes over other groups of diseases that can show the same imaging characteristics. Combining clinical issues with imaging findings can make it possible to reach a specific diagnosis.



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