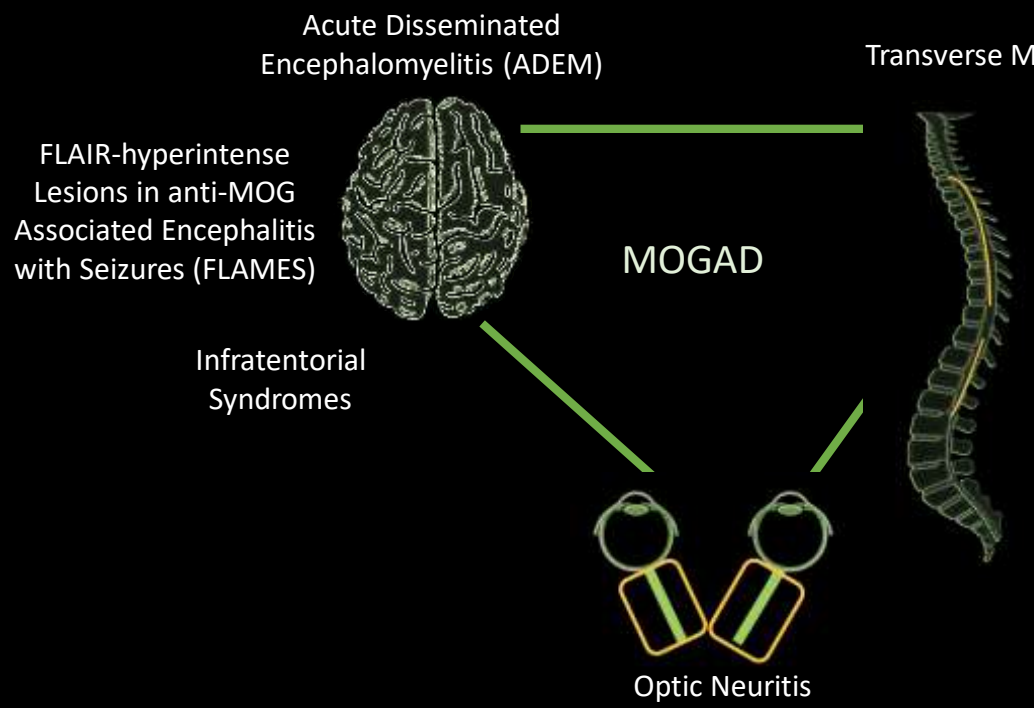
MYELIN OLIGODENDROCYTE GLYCOPROTEIN ANTIBODY-ASSOCIATED DISEASE (MOGAD) HOW TO RECOGNIZE NEUROIMAGING PATTERNS

The disease known as MOG antibody-associated disorder (MOGAD) is an additional inflammatory CNS. It is currently reported as a variable clinical presentation marked by episodes of demyelination driven by the immune system, mainly affecting the optic nerves, brain, and spinal cord

- There is a particular tendency for this disease to occur in children and young people
- MOGAD displays both overlapping features and significant differences from MS and aquaporin-4-IgG seropositive neuromyelitis optica spectrum disorder (AQP4-IgG NMOSD), especially in clinical, radiological, and cerebrospinal fluid analysis.
- It is crucial to radiologists to recognize MOGAD as a distinctive and newly established CNS demyelinating disease with several clinical presentations and overlaps:

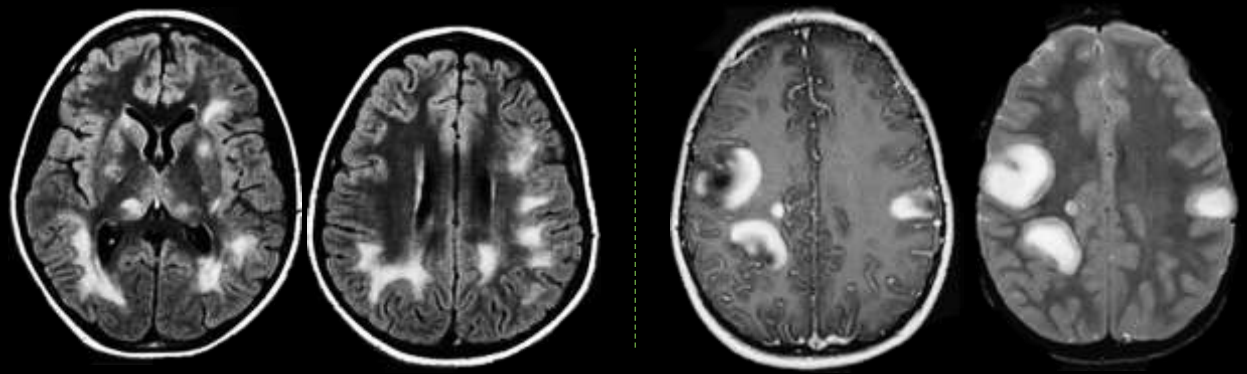


	Children	Adults
ADEM	✓	✓
Brain Stem Lesions	✓	✓
Optic Nerve	✓	✓
Cortical Lesions/FLAMES	✓	✓
Spinal Cord	✓	✓

ADEM

Acute Disseminated EncephaloMyelitis

- Acute inflammation and demyelination of white matter (criteria 2013)
- Monophasic: more common (> 70%);
  - Multiphasic: second episode after 3 months
- Typically follows a recent viral infection or vaccination
- Common in children or adolescents (usually <15 years)
  - Cases reported in all ages
- 10% of cases may relapse within three months

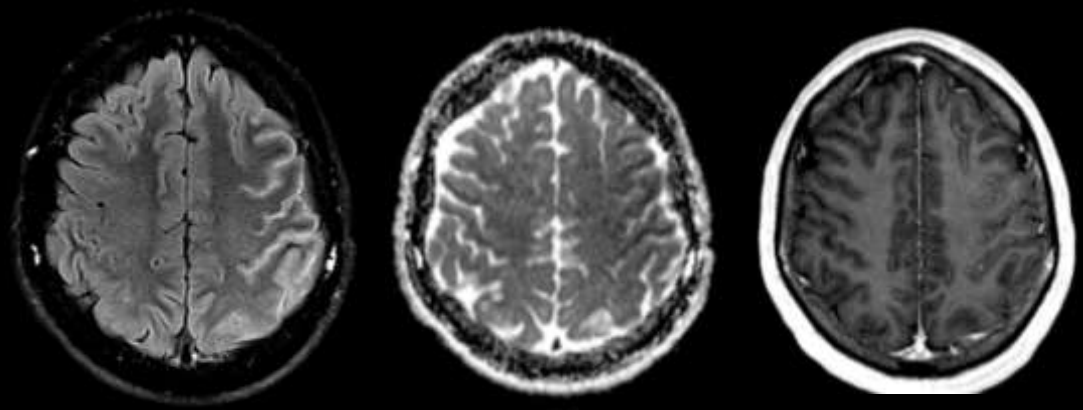


- In T2WI large regions of high signal intensity with surrounding edema are typically found in subcortical locations (affecting predominatly WM)
- Involvement of the thalami and brainstem is also possible
- In T1 C+ (Gd) enhancing lesions are variable including, punctate, ring, or arc enhancement (large lesions with incomplete enhancement) is often observed along the leading edge of inflammation (monomorphic patterrn)
- The absence of such enhancement does not exclude the diagnosis
- On DWI there can be peripherally restricted diffusion (particularly When Gd+)

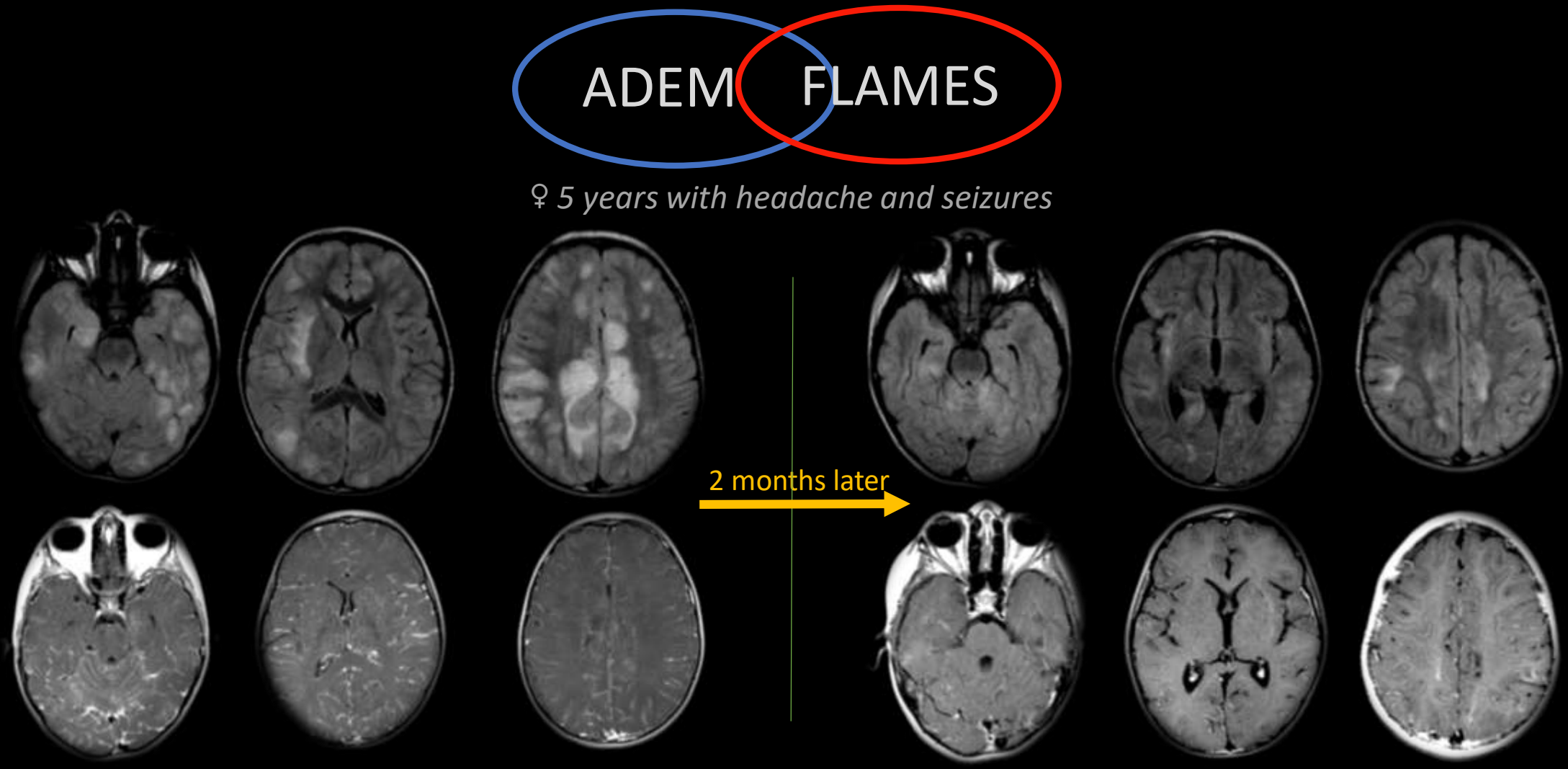
FLAMES

FLAIR-hyperintense Lesions in Anti-MOG associated Encephalitis with Seizures

- FLAIR-hyperintensities on MRI, particularly involving the cerebral cortex
- **Male** predominance (60%); mean age: **29** years old
- Clinical presentation: Seizures (85%), headache (70%), fever (65%), focal cortical symptoms (55%)



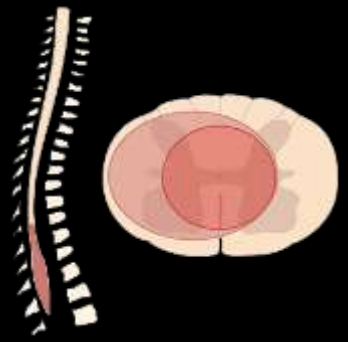
- Cortical hyperintense T2/FLAIR signal
- No juxtacortical white matter involvement
- Sulcal FLAIR-hyperintensity and leptomeningeal contrast enhancement
- There may be BILATERAL cortical involvement



First examination (on left): tumefactive lesions in the deep and juxtacortical WM of the both cerebral hemispheres, in a child (5y-o) that tulfilled ADEM criteria. A diffuse Gd enhancement is noticeable in the leptomeningeal lining, compatible with FLAMES.

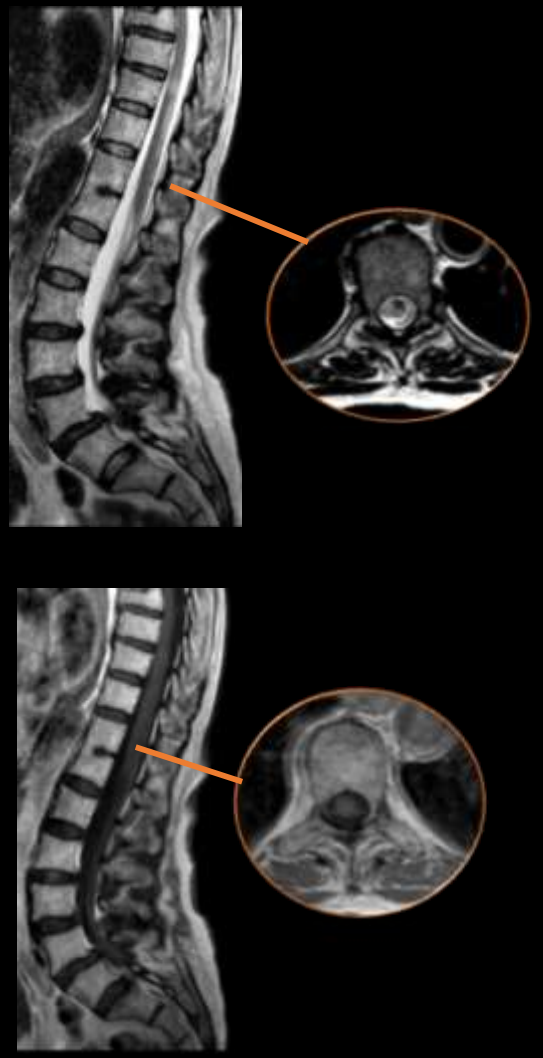
Control examination obtained 2 months later (on right) most of the lesions reduced, remaining cortical hyperintensity on FLAIR images compatible with FLAMES. Corticopial Gd enhancement remain, including new areas in the posterior left temporal lobe, configuring an overlap between ADEM and FLAMES.

TRANSVERSE MYELITIS



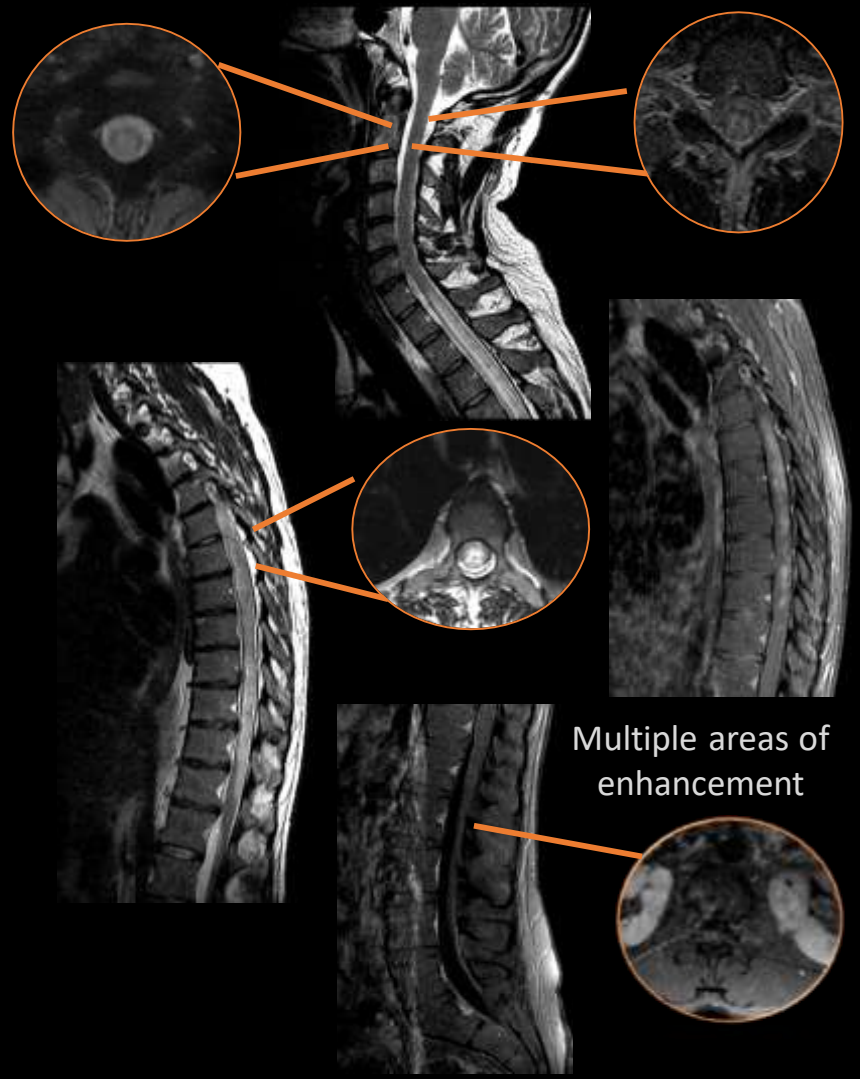
- The disease primarily affects the lower portion of the spinal cord, especially the conus or thoracolumbar region
- Clinically, it presents as transverse myelitis, which can be longitudinally extensive (Longitudinally Extensive Transverse Myelitis - LETM) or short-segment myelitis

- The "H sign" is particularly relevant in MOGAD as it indicates spinal cord involvement with a distinct emphasis on the gray matter. This is a notable feature since MOGAD often presents with spinal cord lesions that predominantly affect the gray matter, which is less common in other demyelinating diseases like Multiple Sclerosis

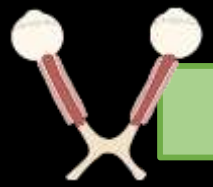


♂23 years with paraplegia

♂29 years with sudden paraplegia



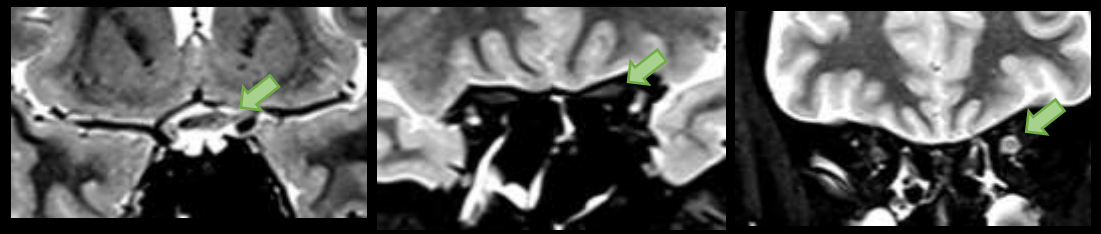
Longitudinally extensive signal change predominantly affecting the central aspect (medular “H”) of the cervicothoracic transition from C7 to T12, including the conus medullaris with thumephal effect→ multiple areas of nodular enhancement



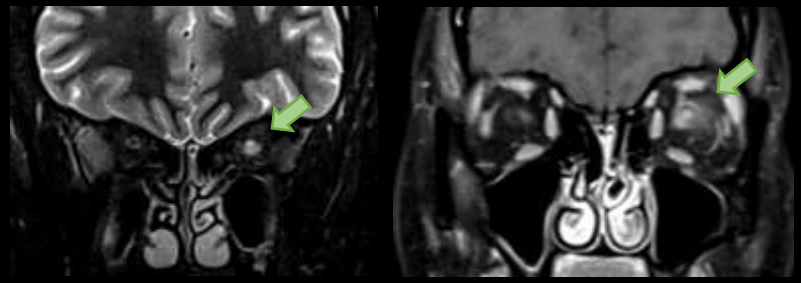
OPTIC NEURITIS

- The optic neuritis typically affects both optic nerves in MOGAD
- The inflammation often extends over a long segment of the optic nerves
- Predominantly involves the anterior portion of the optic nerves
- Swelling of the optic disc is a frequent feature, indicating inflammation
- Inflammation of the sheath surrounding the optic nerve (optic perineuritis) is noted

♀ 45 years with orbit pain and visual loss

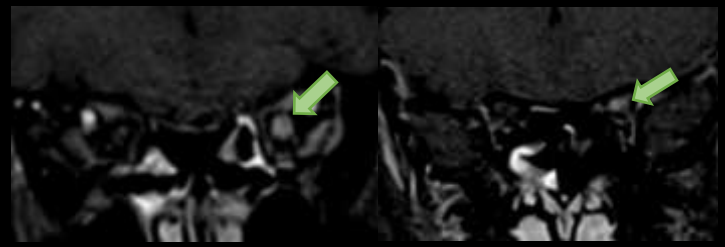


Hipersignal change in coronal T2 weighted sequences compromising the intracranial, intracanalicular and orbital segments of the left optic nerve



♀ 20 years with headache pain and visual loss

The T2-weighted image shows blurring of the optic nerves and adjacent fat, more pronounced on the left. T1 post-contrast imaging reveals enhancement of both the nerve and fat.

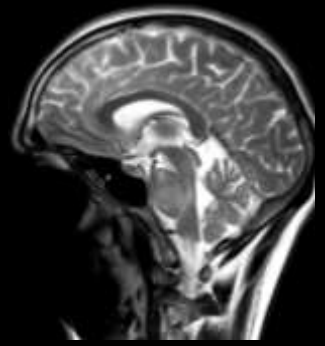


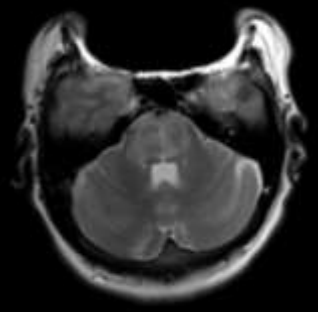
T1 coronal post-contrast illustrating the enhancement of orbital and intracanalicular left segments

INFRATENTORIAL SYNDROMES

- Predominant involvement of the pons and middle cerebellar peduncles

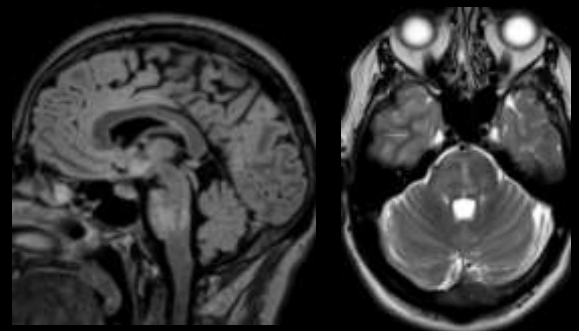
♂23 years with headaches associated with nausea and vomiting and transient diplopia





T2 hyperintensity affecting the midbrain, pons, and right middle cerebellar peduncle, without gadolinium enhancement or diffusion restriction.

1 week →



There was a reduction in the areas of signal change previously characterized.