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MRI FEATURES OF DEMYELINATING DISEASE ASSOCIATED WITH ANTI-MOG ANTIBODIES IN ADULTS

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ABSTRACT:

The spectrum of Myelin Oligodendrocytes Glycoprotein (MOG) antibody disease constitutes a recently described challenging entity, referring to a relatively new spectrum of autoimmune disorders with antibodies against MOG predominantly involving the optic nerve and spinal cord.

The purpose of this article is to describe MRI features of MOG-AD involvement in the optic nerves, spinal cord and the brain of adults.

Keywords: MOG-IgG; MOG-AD; Neuromyelitis Optica; AQP4-IgG; NMOSD; MRI

Abbreviations: MOG = Myelin Oligodendrocytes Glycoprotein; MOG-SD = Myelin Oligodendrocytes Glycoprotein antibody-associated Spectrum Disorder; NMO = Neuromyelitis Optica; NMOSD = Neuro-Myelitis Optica Spectrum Disorder; Aquaporin-4 antibodies = AQP4-IgG; MOG-antibodies = MOG-IgG

BACKGROUND

MOG is a glycoprotein located on the myelin surface and found exclusively in the central nervous system (CNS). Although its exact role remains unclear, it is thought to act as a cellular adhesive molecule, as a regulator of oligodendrocyte microtubule stability and as a mediator of the complement activation cascade.

In recent years, a new diagnostic role has been discovered for MOG antibodies (MOG-IgG), particularly in adults. While MOG-IgGs were initially thought to play a role in typical cases of multiple sclerosis (MS), recent studies have excluded this correlation and highlight its occurrence among patients with isolated or relapsing optic neuritis (ON) and/or myelitis [1].

Clinically, MOG antibody disease is closely related to neuromyelitis optica spectrum disorder (NMOSD) in terms of the localisation of inflammatory attacks within the CNS. But while in the majority of NMOSD cases, the syndrome is associated with auto-antibodies to aquaporin-4 (AQP4-IgG), the most commonly expressed water channel located on astrocytes of the CNS, up to 40 % of NMOSD patients test negative for AQP4-IgG [2–4].

So, based on the fact that AQP4-IgGs are usually absent in MOG-IgG-positive (MOG+) patients [4–6], the histopathology of inflammatory CNS lesions differs between MOG-IgG- and AQP4-IgG-positive (AQP4+) patients [7,8] and since MOG-IgGs are pathogenic both *in vitro* and *in vivo*, MOG-IgG-related autoimmunity, also called MOG antibody disease (MOG-AD) [9], is now recognised as a distinct nosological entity with specific management and therapeutic requirements.

In this review, we will briefly outline the clinical presentation of MOG-SD in adults and discuss their MRI features in detail.

CLINICAL AND DEMOGRAPHIC CHARACTERISTICS

MOG-AD can occur in all decades of life, with a median age of onset in the early to mid-thirties, and it affects slightly more women than men (female: male ratio = 1,1:1) [5,9–13].

The most common presenting feature is optic neuritis (ON), occurring in 54–61% of patients, followed by myelitis. More rarely MOG-AD involves the brain, the phenotype in these cases is often similar to acute disseminated encephalomyelitis (ADEM) or an ADEM-like presentation (e.g., brainstem attack) [9–15] or even encephalitis possibly associated with seizures [16,17].

A relapse pattern has been reported in 44–83% of patients [10,11,13] and more commonly involves the optic nerve [11–13].

The impact of relapses on disability is variable : some studies report no difference between monophasic and relapsing disease courses [11] whilst others report worsening disability associated with higher relapse frequency [13]. In case-based series, residual disability develops in 50–80% of patients [10,12,13], with transverse myelitis at onset being the most significant predictor of long-term disability [13].

At presentation, MOG+ patients are thought to be at lower risk of further relapses than AQP4+ patients and have better visual and motor outcomes [11,13]. In comparison to children with AQP4+ NMOSD, those with MOG-AD tend to be younger, less likely to present with area postrema syndrome, but more likely to present with ADEM.

Although MOG+ patients often present with severe symptoms, they usually display a remarkable recovery after steroid treatment, and usually have better visual field outcomes compared to AQP4+ patient, although recurrence of ON is significantly more frequent [18,19].

Although in most cases demyelination associated with MOG antibodies occurs without any apparent inciting or predisposing event/illness, it has been associated with demyelinating N-methyl-d-aspartate receptor encephalitis [20], post-infectious demyelination following herpes simplex virus, Borrelia and Epstein–Barr virus infections and, more rarely, with typical relapsing MS [7,10,21–25].

Whether MOG-IgG play a pathogenic role in all these conditions, or if they represent a bystander effect or epiphenomenon, remains unclear.

Recent studies have also reported a higher relapse rate following an initial inflammatory demyelinating disorder in paediatric patients with persistent MOG-IgG1 seropositivity, with one study [26] even demonstrating the benefits of MOG-IgG1 serological testing in adult patients and highlighting that longitudinal serologic evaluation of MOG-IgG1 could help predict disease course and that immunotherapy may constitute a promising therapeutic approach.

Such outcomes reinforce the need for rapid identification and confirmation of MOG+ in order to initiate adequate treatment, and further justify an evaluation of MRI features associated with this disease.

MRI FEATURES

OPTIC NERVE MRI FINDINGS IN MOG-SD

MRI features of optical neuritis are highly reproducible among MOG+ patients. During optic neuritis attacks, orbital MRI (ideally acquired with coronal fat-suppressed T2- and post-contrast T1-weighted sequences) shows an extensive optic nerve T2 hyperintensity. This predominantly involves the anterior segments of the optic nerve, with almost routine inclusion of the intra-orbital segments, while chiasm and retro-chiasmatic pathways are generally spared [18,27]. The optic nerve becomes oedematous, enlarged and tortuous, and optic disc oedema can be observed (Figure 1). Additionally, restriction on diffusion-weighted imaging can be seen (Figure 2). Inflammation and enhancement of the peri-optic nerve sheath, partly extending into the surrounding orbital fat, is often demonstrated (1/3 of cases) [27] (Figure 2). This seems crucial as it is not apparent in MS or NMOSD AQP4+ patients. Optic nerve involvement is bilateral in 25% of cases (Figures 1,3). All of these characteristic features provide invaluable insight into the diagnosis of MOG+ ON.

Furthermore, the MRI appearance of MOG+ ON differs from AQP4+ ON since it is more anterior, stretched, oedematous, with extended inflammation, whereas the latter tends to involve the posterior segments, including the optic chiasm, more frequently. MS-optic neuritis is also different in that it is usually unilateral and occurs in a limited segment [28].

Orbital MRI performed during follow-up shows marked regression of the oedematous appearance observed in the acute phase and extinction of gadolinium enhancement. In most patients, residual T2 hyperintensity is evident, persisting over time, and can be associated with optic nerve atrophy (Figure 3).

SPINAL CORD MRI FINDINGS IN MOG-SD

During myelitis attacks, MRI can demonstrate spinal cord involvement in two different patterns:

- longitudinally extensive spinal cord lesions, also known as longitudinally extensive transverse myelitis (LETM), representing extensive involvement of the spinal cord, with abnormal hyperintense T2 signal crossing at least three vertebral body segments lengthwise and involving more than 50% of the axial section of the medullary cord;

- Also T2-hyperintense short lesions, that are smaller than 2 vertebral segments [5].

Regardless of their extent, such lesions typically involve both grey and white matter, covering more than 50% of the axial section of the medullary cord, and may be associated with cord swelling (Figure 6). Lesions can affect the whole spinal cord but conus involvement is considered a highly specific location for MOG-AD diagnosis [5,29,30] (Figure 5). Patchy, heterogeneous enhancement with blurred margins (“cloud-like enhancement”) is generally noted (Figures 4,5). Well-defined nodular enhancement or meningeal enhancement is also occasionally depicted. A “bright spotty lesion”, an intramedullary lesion with a higher T2 signal intensity than CSF, and a T1 “dark lesion” (Figure 6), an imaging feature known as indicative of NMOSD AQP4+ [31], seem to be quite rare in MOG+ myelitis.

Whilst small lesions can resemble MS and large lesions may appear similar to anti-AQP4-associated myelitis, some new radiological features of MOG-IgG+ related myelitis have been highlighted in the recently published paper by Dubey et al [32]. One of the most relevant radiological findings described was the presence of a sagittal line hyperintensity (SLH) surrounded by a cloudier T2-hyperintense signal, concurrent with a H-shaped hyperintensity (HSH) seen in axial sequences and due to grey matter involvement. Interestingly, on axial sequences this T2-hyperintensity appeared more pronounced around the central canal area. The authors suggest that this MRI feature is associated with MOG-IgG positivity, since it is found in 28% (15/51) of MOG-IgG+ patients, but only 8% (3/39, $p=0.007$) of AQP4-IgG+ patients and is never observed in patients with multiple sclerosis [32].

In our experience, this pattern of central, thin and linear T2 hyperintensity, superimposed on the main lesions along the ependymal canal, was observed in 50% of anti-MOG myelitis (Figures 4,5). We previously described it as a “pseudo dilatation” of the ependymal canal [33]. This specific imaging sign could prove extremely useful in identifying lesions indicative of MOG-AD, and it would be certainly interesting in future studies to focus on “pseudo-dilatation” of the central ependymal canal and analyse its specificity and sensitivity in NMOSD-MOG+ compared to NMOSD-AQP4+ and MS.

After gadolinium administration, linear enhancement of the ependymal canal (pencil-thin enhancement) can also be observed (Figure 4).

During follow-up, as in the case of optic neuritis, spinal MRI shows a marked regression of the signal abnormalities and of the gadolinium uptake of the spinal lesions. In most patients, discrete residual T2 hyperintensity, persistent over time, is associated with minimal focal medullary atrophy (Figure 7), which is less pronounced than in AQP4-IgG+ patients [34]. Interestingly, we note in some cases that SHS can persist weeks to months after the acute phase.

BRAIN MRI FINDINGS IN MOG-SD

Brain MRI is considered normal in 2/3 of cases or else reveals non-specific supratentorial sub-cortical or small, deep, white matter foci with hyperintensity on T2-weighted sequences [30]. These non-specific lesions are generally asymptomatic.

However, in rare cases, atypical T2 hyperintense lesions can be seen in the brain, with a predilection for the brainstem and infratentorial regions [27] such as the mesencephalon, pons, bulbar olives or the cerebellar peduncles (Figures 8,9). Cases of encephalomyelitis/encephalitis have been reported, and in these cases, MRI findings usually report an ADEM-like pattern with diffuse signal changes noted in the cortical grey matter (GM)/subcortical white matter (WM), deep WM and deep GM (basal ganglia and thalami) as seen on both T2-weighted and FLAIR [14].

Of particular note, use of the double inversion recovery sequence may be useful to potentially detect cortical lesions but have seldom been reported in cerebral MOG+ cases [35]. In this context, a recent

review proves that the brain lesion distribution criteria are helpful in distinguishing MS from NMOSD and MOG-associated encephalomyelitis [36].

These lesions can occasionally show gadolinium enhancement. Most of the gadolinium uptake is displayed in a poorly delineated, subtle pattern with multiple patches referred to as “cloud-like” enhancements [37] (Figure 9). Scattered linear and nodular enhancements are also apparent in active lesions with some cases demonstrating restricted diffusion [38] (Figure 8). These cloud-like enhancing lesions differ from the ovoid or ring/open-ring gadolinium-enhancing lesions with well-defined borders that are more typical of MS.

For now, no specific radiologic pattern has been identified to distinguish MOG antibody cases from non-MOG antibody cases, except that leptomeningeal enhancement and thalamic lesions were unique to the MOG+ cohort [11].

It has also been shown that cranial nerve involvement can coexist in patients with MOG antibody disease, with gadolinium enhancement of CNs at the transitional zone at the root level displayed on T1W postcontrast sequence, although the underlying pathophysiology remains elusive [39].

During follow-up, in the majority of cases, specific MOG+ brain lesions are known to substantially decrease after treatment [14,27,29], but there are exceptions.

CONCLUSION

MOG antibody disease is now considered a specific entity per se, predominantly affecting the optic nerves, spinal cord and, to some extent, the brain. Although it may resemble AQP4-IgG-mediated NMOSD, or even MS or ADEM from a clinical perspective, MRI could certainly help to highlight anti-MOG disease when key imaging features are demonstrated, such as bilateral extensive oedematous and inflammatory anterior optic neuritis or longitudinally extended myelitis with a predilection to the conus, associated with “pseudo-dilatation” of the ependymal canal, as illustrated in this review.

Captions

Figure 1: 29-year-old woman with bilateral MOG-optic neuritis. Coronal T2-weighted (A), axial FLAIR with fat suppression (B), coronal (C) and axial (D) T1-weighted with Gadolinium and fat saturation MRI. Bilateral and symmetrical optic nerve swelling (A) and Gadolinium enhancement (C), with longitudinally extensive bilateral involvement (B and D). Bilateral swelling of the optic nerve head is also present (arrows in B).

Figure 2: 27-year-old man with unilateral acute MOG-optic neuritis. DWI (A) image with corresponding ADC map (B), Coronal T2 STIR (C) and Coronal fat-suppressed contrast-enhanced T1 (D) MRI in the same patient. Bright signal diffusion intensity in the left optic nerve (arrow in A) in the acute phase of optic neuritis, with associated reduction in ADC, represented by the dark signal of the left optic nerve (arrow in B). T2 hyperintensity (C) of the left optic nerve and inflammation of the surrounding intra-orbital fat as well as contrast enhancement (D) of the intra-orbital left optic nerve and concurrent enhancement of the peri-optic nerve sheath, partly extending into the surrounding orbital fat.

Figure 3: 22-year-old woman with MOG-optic neuritis in acute phase (A, C) with MRI follow-up after 18 months (B, D). Axial (A, C) and coronal (C) FLAIR-weighted and fat-suppression, and coronal (D) T2 STIR images. In the acute phase, left optic nerve swelling and hyperintensity is visualised in A and C. 18 months later, atrophy of the optic nerve is visualised on the left (white arrow in B, white circle in D) with contralateral recurrence (red arrow in B, red circle in D). Of particular note, optic nerve head swelling visualised in the acute phase (orange arrow in A and B).

Figure 4: 22-year-old woman with acute MOG-myelitis. Spinal MRI with sagittal T2-weighted (A), sagittal T1-weighted (B), sagittal (C), and axial (D) T1 with Gadolinium images. During disease onset, a longitudinal extensive T2 hyperintense lesion (A) involving the cervical spinal cord is seen, as well as cord swelling. MOG-myelitis is isointense in T1 (B) with no T1 dark lesion, and shows pencil thin ependymal enhancement (arrows in C, confirmed in D) after Gadolinium administration.

Figure 5: 22-year-old woman with acute MOG-myelitis. Spinal cord MRI with sagittal T2-weighted (A), sagittal T1 fat-suppressed with Gadolinium (B) and axial T2-weighted (C) images. MOG-myelitis short lesions involving lumbar cord and conus. Of particular note, pseudo dilatation of the ependymal canal (A) as a characteristic feature. After Gadolinium administration, the conus lesion shows a patchy, cloud-like contrast enhancement (arrow in B). On axial section, MOG-myelitis highlights the involvement of grey and white matter, >50% of the spinal cord section (C).

Figure 6: Different aspects of MOG-myelitis lesions on axial T2-weighted images. In A, combined lesion involving the white and grey matter of the medullary cord; in B, H-shaped lesion and in C, "bright spotty lesion". Preservation of the peripheral T2 hypointensity is apparent.

Figure 7: Changes in spinal cord highlighted during follow-up in an 18-year-old woman with MOG-myelitis. Sagittal T2-weighted or T2/STIR images in serial exams. Cervical longitudinal extensive transverse MOG-myelitis on initial MRI (A) with cord expansion. Regression of signal abnormalities and oedema in 1 month (B) and in 3 months (C). Recurrent remote cervico-thoracic myelitis after 7 months (D). Finally, regression of lesion with discrete T2 hyperintensity and minimal atrophy after 3 years (E).

Figure 8: Rhombencephalitis as a type of brain involvement in MOG-SD in a 62-year-old woman. Brain MRI with axial diffusion (A), ADC (B), axial (C) and sagittal (D) FLAIR-weighted images, axial T1-weighted before (E) and after Gadolinium admission (F), perfusion sequence (G) with K2 and rCBV maps and monovoxel TE 135 spectroscopy (E) images. Confluent lesion in the mesencephalon, very bright on FLAIR (C, D) and very dark on T1 (E), showing patchy enhancement

with blurred margins (F), and diffusion restriction (A, B). Perfusion sequence (G) shows rupture of the blood-brain barrier. Spectroscopy (E) is not specific, but is not indicative of tumour aetiology.

Figure 9: An additional case of rhombencephalitis in MOG-SD in a 37-year-old man presenting right facial hypoaesthesia for a duration of 1 month, followed by rotating vertigo. Axial FLAIR-weighted (A), ADC cartography (B) and T1-weighted with Gadolinium (C) images. Dynamic perfusion curve (D) and monovoxel TE135 spectroscopy (E). A large, confluent lesion in the right cerebellar peduncle (A-C), showing patchy enhancement (C) with blurred margins, without restricted diffusion (B). The lesion is associated with rupture of blood-brain barrier, but is not hypervascular (D). Spectroscopy (E) is highly suggestive of an inflammatory lesion: high choline to creatine ratio (1,70), N-acetylaspartate (NAA) peak reduced (choline to NAA ratio:1,23), and lactate peak.

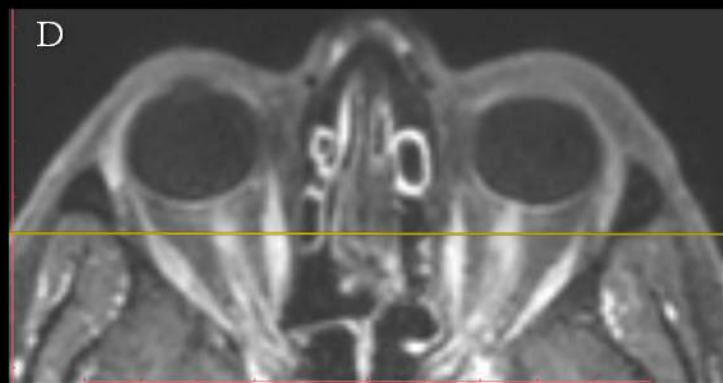
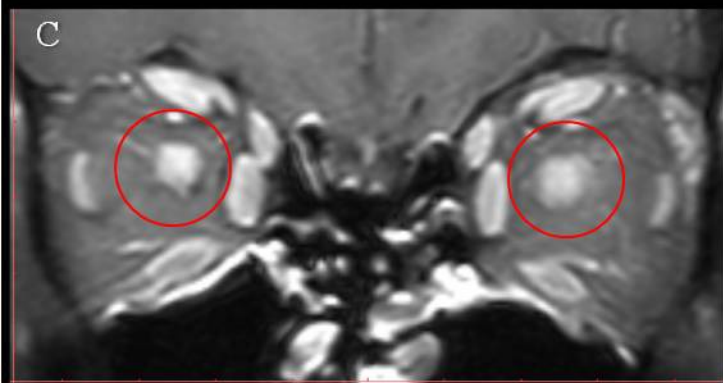
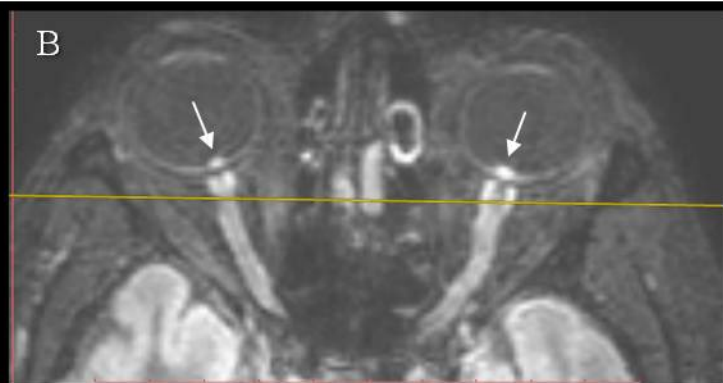
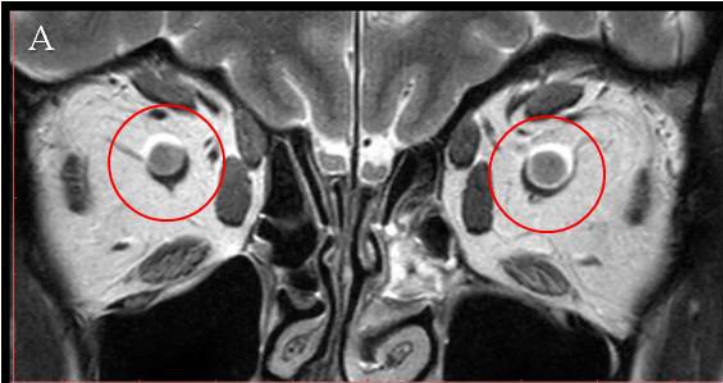
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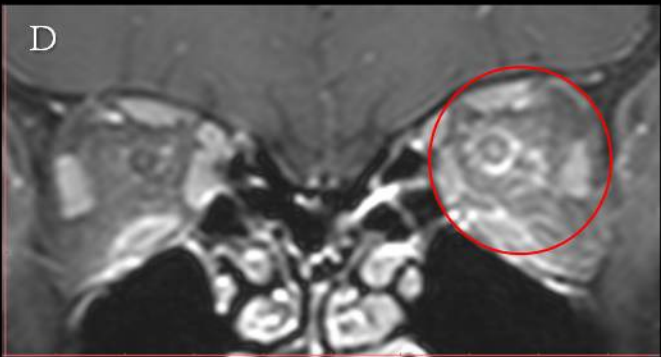
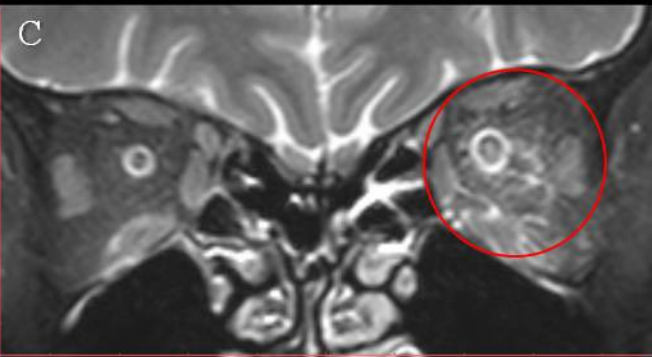
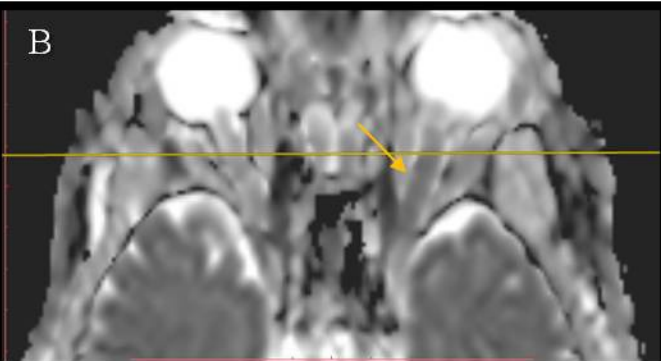
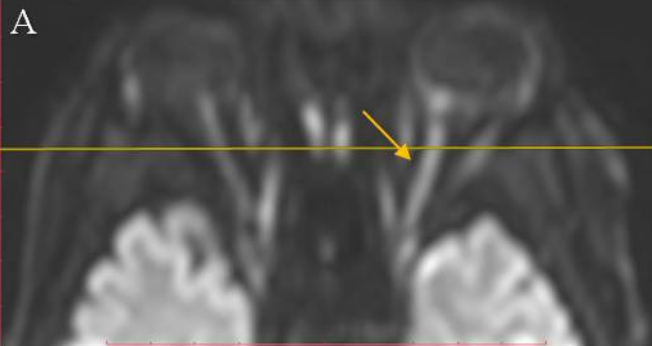
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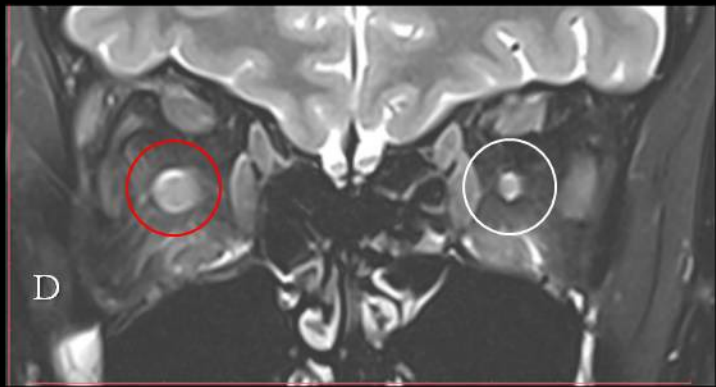
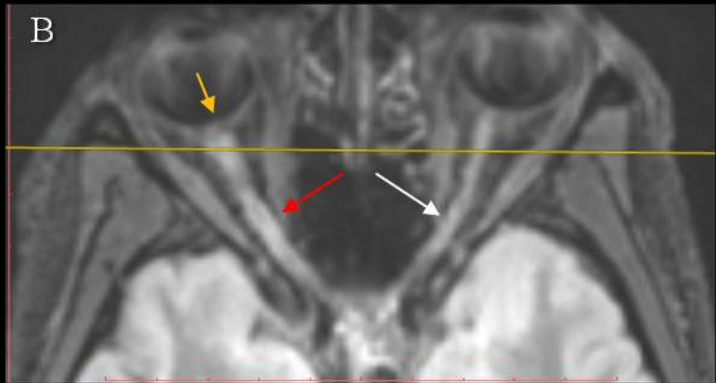
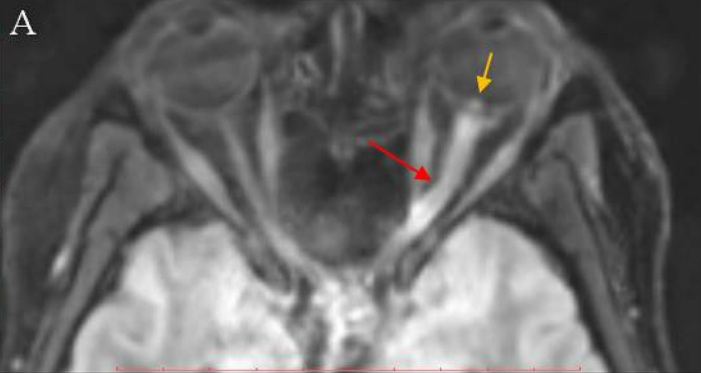
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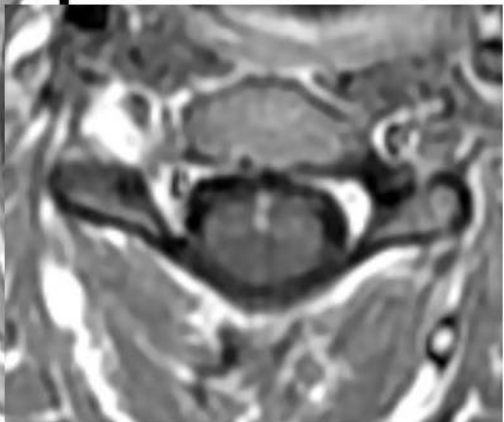
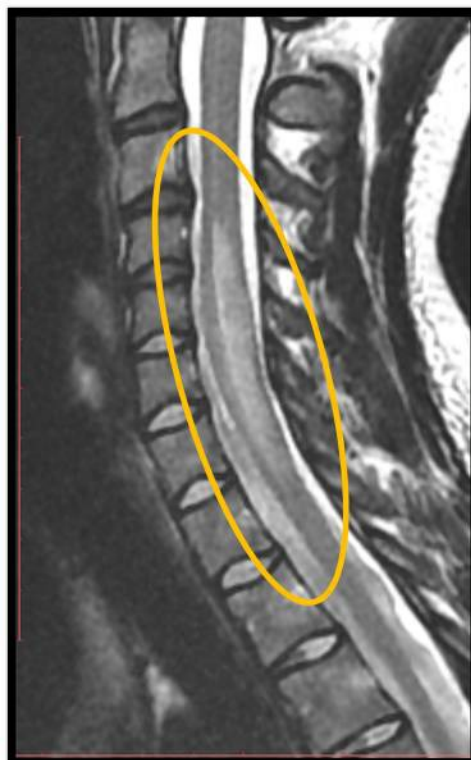
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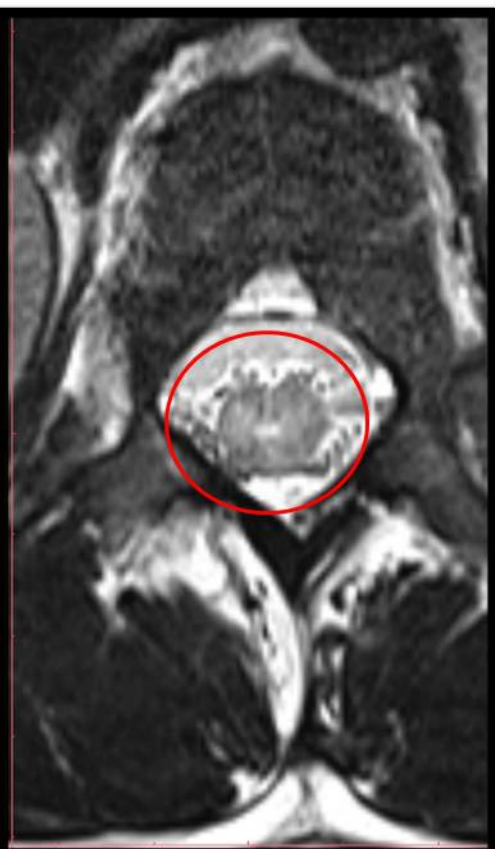
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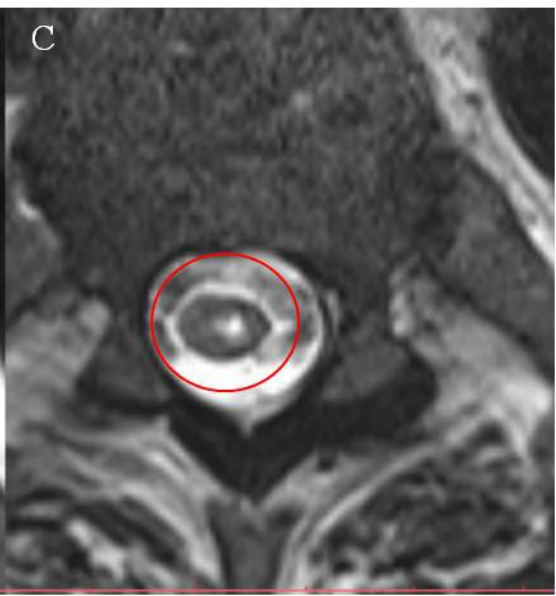
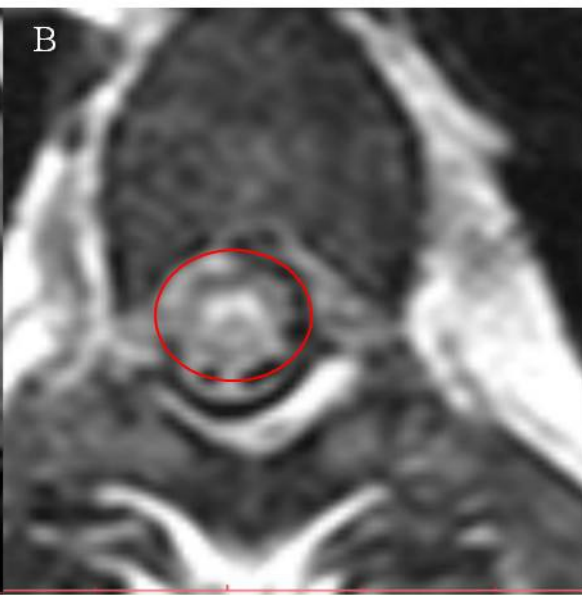




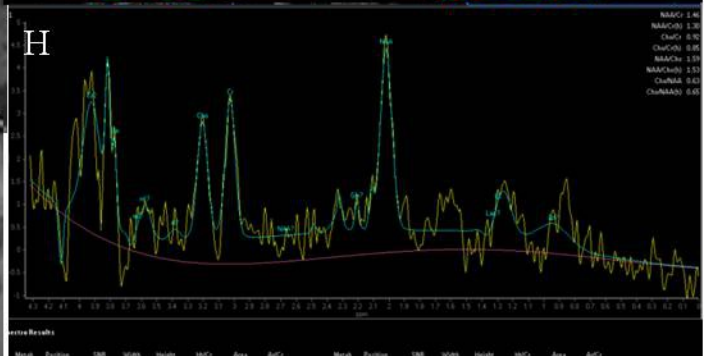
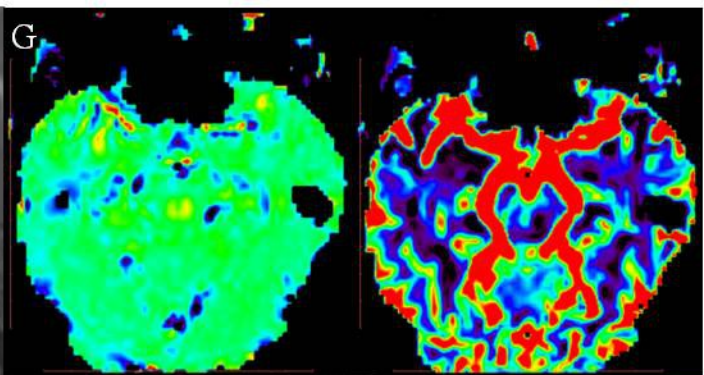
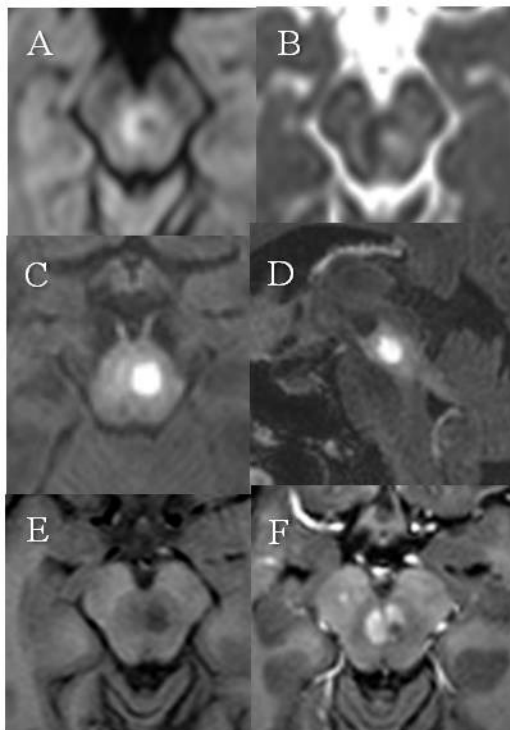


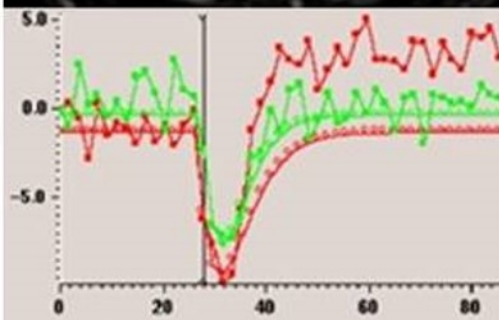
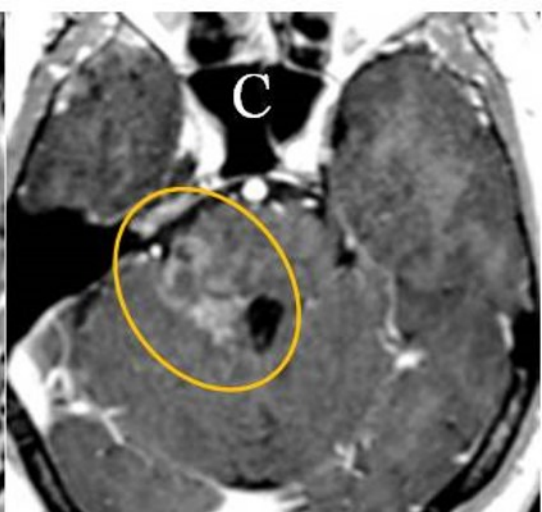
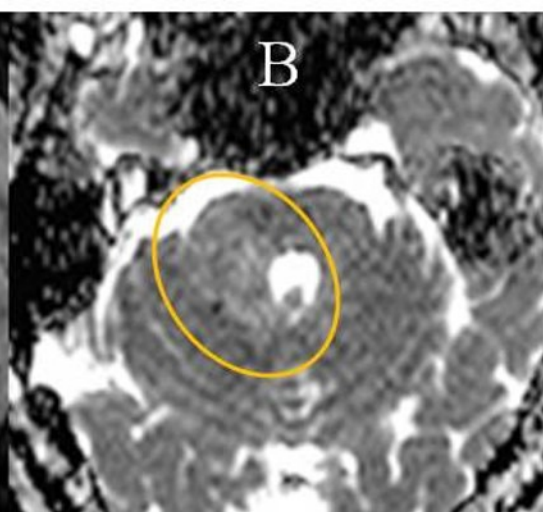
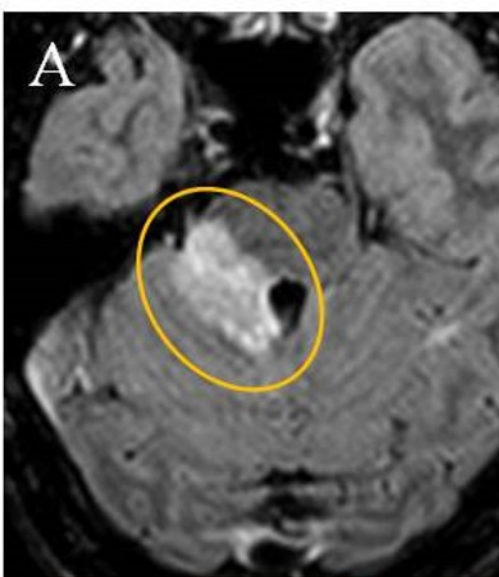












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- 'Dynamic' en cours
- 'Dynamic' de référence
- L1 (Adapté)
- L2 (Adapté)

