

Accurate and Timely MS Diagnosis

CAN DO
MULTIPLE SCLEROSIS



WEBINAR 
WEDNESDAYS

This activity is supported by independent educational grants from EMD Serono, TG Therapeutics, Inc., and The Kathleen C. Moore Foundation



Target Audience

This activity has been designed to meet the educational needs of physicians, PAs, NPs, nursing professionals and other members of the healthcare team involved in the management of patients with MS.

Accreditation Information

In support of improving patient care, this activity has been planned and implemented by the Consortium of Multiple Sclerosis Centers (CMSC), The Texas MS Consortium and Can Do Multiple Sclerosis. CMSC is jointly accredited by the Accreditation Council for Continuing Medical Education (ACCME), the Accreditation Council for Pharmacy Education (ACPE), and the American Nurses Credentialing Center (ANCC), to provide continuing education for the healthcare team.

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Faculty Disclosures

Mirla Avila MD, FAAN, has reported the following relevant financial relationships: Consultant: Biogen, Bristol Myers Squibb, and TG Therapeutics; Advisor: Amgen.

Léorah Freeman MD, has reported the following relevant financial relationships: Advisor: Genentech, Horizon Therapeutics, Sanofi, and TG Therapeutics; Consultant: Hoffman LaRoche and Merck EMD Serono; Contracted Research: Genentech, Merck EMD Serono, Sanofi and TG Therapeutics; Speaker: Genentech, Merck EMD Serono, and Sanofi.

Lyndsey Hale, MD, has disclosed no relevant relationships.

Staff Disclosures

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All other planners, reviewers and staff at the CMSC who are in a position to control content have disclosed no relevant financial relationships.

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Your Speakers



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Medical Director, MS And Neuroimmunology
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LEARNING OBJECTIVES



1

Incorporate the McDonald Diagnostic Criteria into diagnosis strategies in evaluating patients with suspected MS in order to make more accurate and timely MS diagnosis

2

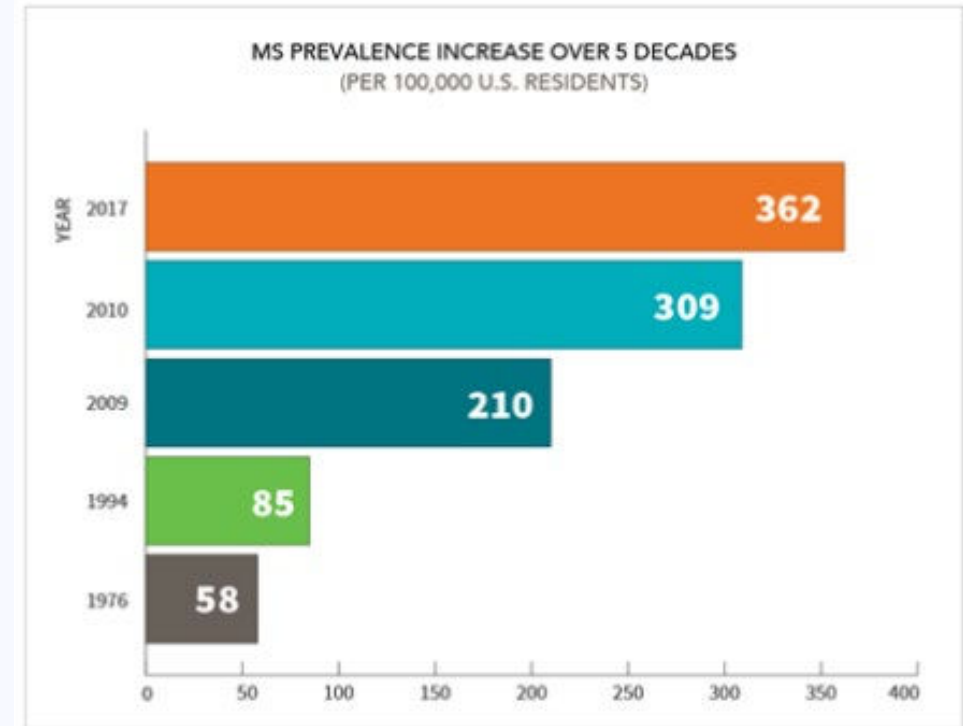
Identify red flags to MS misdiagnosis in order to recognize and address them when evaluating patients

3

Review key imaging features to help with the differential diagnosis

Epidemiology of Multiple Sclerosis

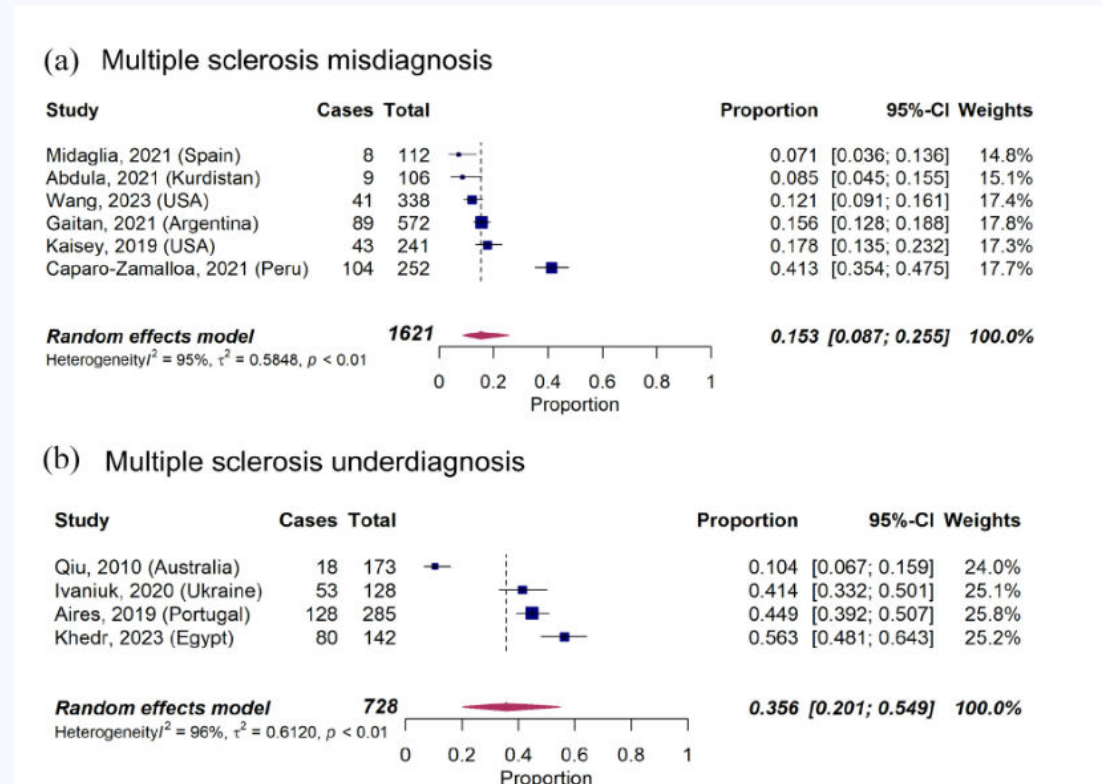
- The prevalence of MS in the US was estimated to have reached **362/100,000** in **2017**, or a total of 913,925 individuals living with the disease.
- 3:1 female-to-male ratio.
- MS is more common in areas inhabited by people of northern European Ancestry with a significant North-South gradient.
- **MS occurs in most racial or ethnic groups, including Blacks, Hispanics, and Asians.**
- **Findings suggest that Black women have a higher risk of developing MS than previously reported.**



Source: Wallin, Mitchell T. "The prevalence of MS in the United States: A population-based estimate using health claims data." *Neurology*. February 2019. *Neurology Journal Web*. <http://n.neurology.org/lookup/doi/10.1212/WNL.0000000000007035>
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Misdiagnosis and Underdiagnosis of Multiple Sclerosis

- A recent meta-analysis shows that MS is misdiagnosed in 15% of cases and underdiagnosed in 36% of cases.
- Women were more likely misdiagnosed with MS compared to men. Migraine, cerebrovascular diseases, and psychiatric disorders were the most commonly mistaken conditions.
- The time delays to correct diagnoses have also been found to be extensive.
- One study showed that in the US even in recent years, the mean time from first symptoms to diagnosis remains above 1 year, at 13.8 months.



Importance of Recognizing Presentations of MS

Typical Presentations

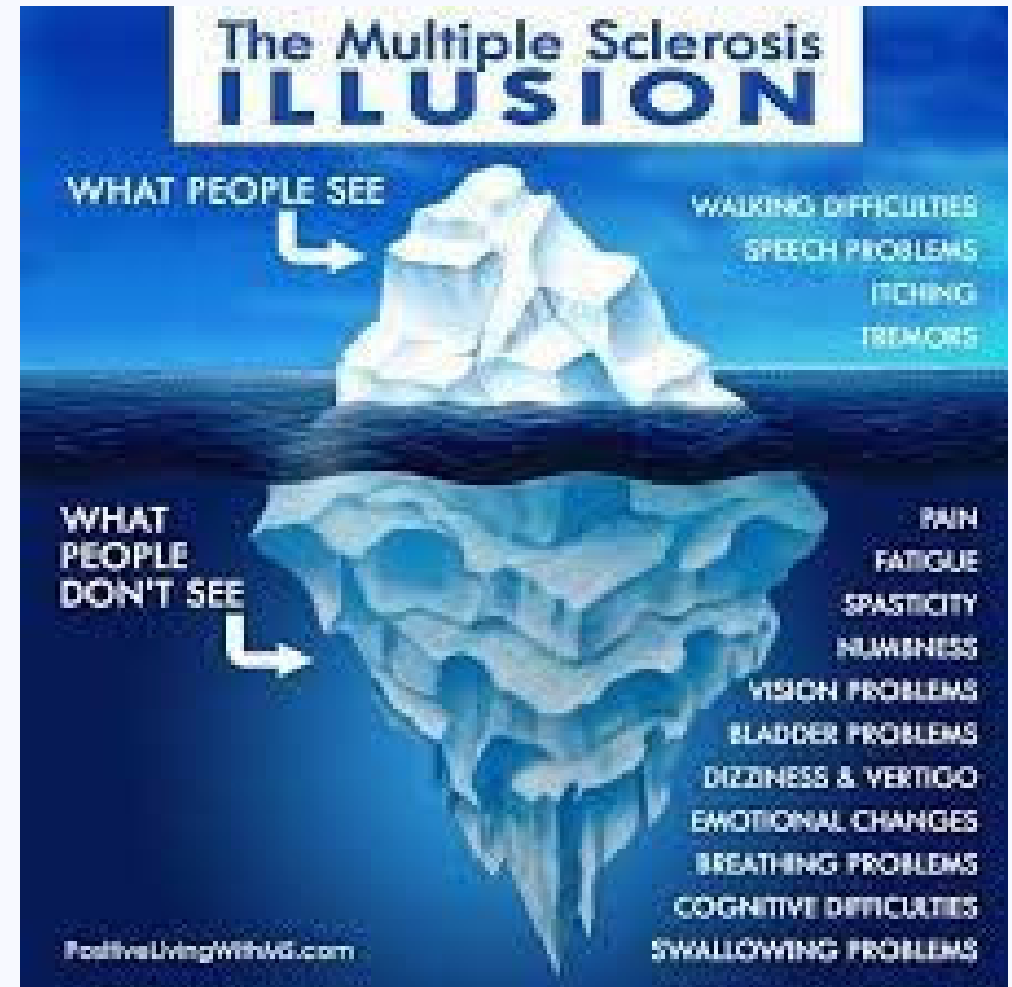
- Acute unilateral optic neuritis
- Facial sensory loss or trigeminal neuralgia in a young adult (< 40 yrs of age)
- Cerebellar ataxia and nystagmus
- Partial myelopathy
- Sensory symptoms in a CNS pattern
- Double vision due to internuclear ophthalmoplegia or sixth nerve palsy
- Lhermitte's sign
- Asymmetric limb weakness
- Urge incontinence or erectile dysfunction

Atypical Presentations

- Bilateral optic neuritis or unilateral optic neuritis with poor visual recovery
- Complete gaze palsy or fluctuating ophthalmoparesis
- Intractable nausea, vomiting, or hiccups
- Complete transverse myelopathy with bilateral motor and sensory involvement
- Encephalopathy or subacute cognitive decline
- Headache or meningismus
- Isolated fatigue or asthenia
- Constitutional symptoms

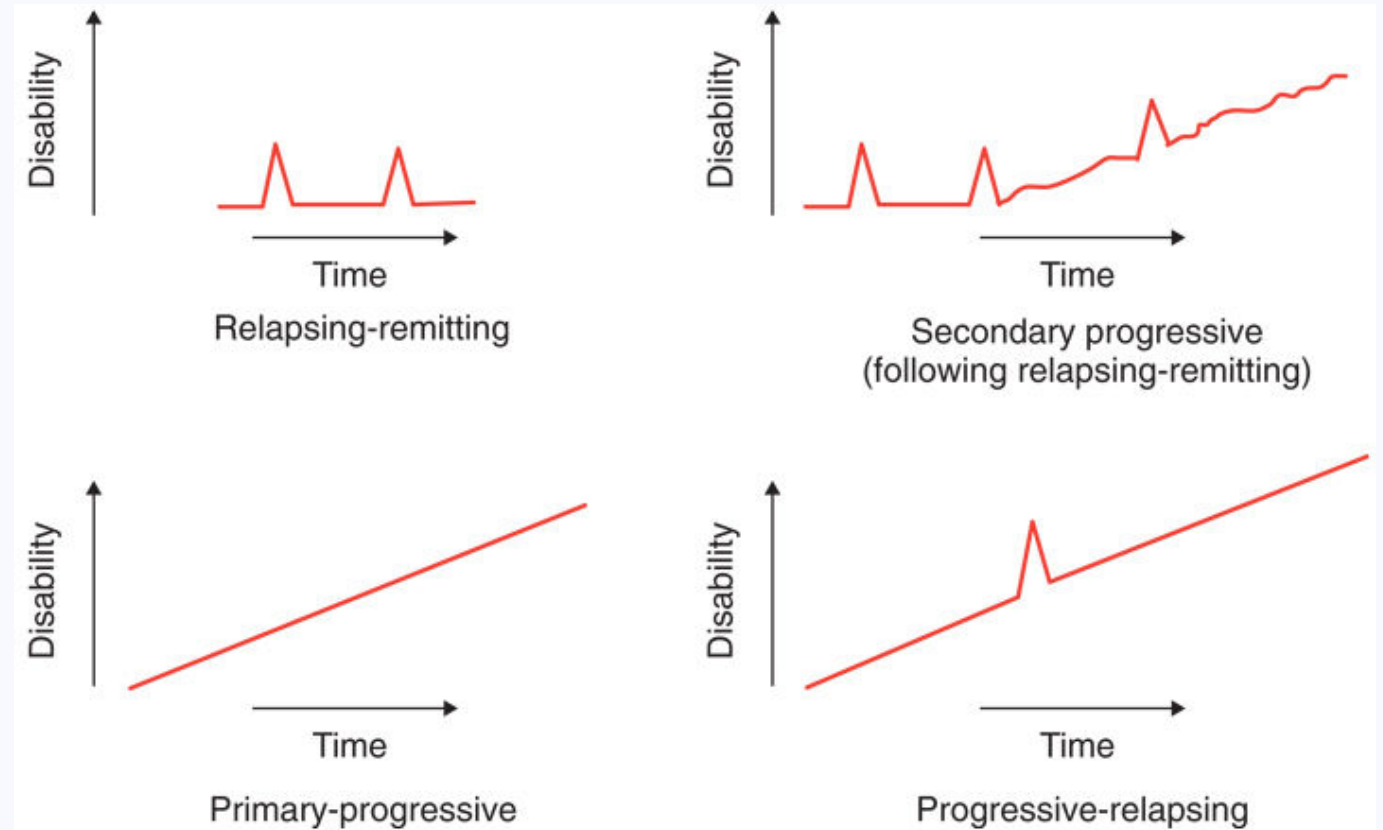
Invisible Symptoms

- Fatigue 65%-97%
- Bladder dysfunction 52%-97%
- Sexual dysfunction 40%-90%
- Pain 29%-86%
- Spasticity 40%-85%
- Cognitive impairment 40%-70%
- Bowel dysfunction 35%-68%



Disease Courses

- Relapsing forms of MS
- Secondary progressive MS
- Primary progressive
- Progressive relapsing
- Active vs. non-active



Over time, MS diagnostic criteria have changed to allow for earlier diagnosis of MS

SPECIAL ARTICLE

New Diagnostic Criteria for Multiple Sclerosis: Guidelines for Research Protocols

Charles M. Poser, MD,¹ Donald W. Paty, MD,² Labe Scheinberg, MD,³ W. Ian McDonald, FRCP,⁴ Floyd A. Davis, MD,⁵ George C. Ebers, MD,⁶ Kenneth P. Johnson, MD,⁷ William A. Sibley, MD,⁸ Donald H. Silberberg, MD,⁹ and Wallace W. Tourtellotte, MD¹⁰

Diagnostic Criteria for Multiple Sclerosis: 2005 Revisions to the “McDonald Criteria”

Chris H. Polman, MD, PhD,¹ Stephen C. Reingold, PhD,² Gilles Edan, MD,³ Massimo Filippi, MD,⁴ Hans-Peter Hartung, MD,⁵ Ludwig Kappos, MD,⁶ Fred D. Lublin, MD,⁷ Lianne M. Metz, MD,⁸ Henry F. McFarland, MD,⁹ Paul W. O'Connor, MD,¹⁰ Magnhild Sandberg-Wollheim, MD,¹¹ Alan J. Thompson, MD,¹² Brian G. Weinstenker, MD,¹³ and Jerry S. Wolinsky, MD¹⁴

New diagnostic criteria for multiple sclerosis integrating magnetic resonance image assessment with clinical and other paraclinical methods were introduced in 2001. The “McDonald Criteria” have been extensively assessed and used since 2001. New evidence and consensus now strengthen the role of these criteria in the multiple sclerosis diagnostic workup to demonstrate dissemination of lesions in time, to clarify the use of spinal cord lesions, and to simplify diagnosis of primary progressive disease. The 2005 Revisions to the McDonald Diagnostic Criteria for MS should simplify and speed diagnosis, whereas maintaining adequate sensitivity and specificity.

Ann Neurol 2005;58:840–846



Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria

Alan J. Thompson, Brenda L. Banwell, Fredrik Barkhof, William M. Carroll, Timothy Coetzee, Giancarlo Comi, Jorge Correia, Franz Fazekas, Massimo Filippi, Mark S. Freedman, Kazuo Fujihara, Steven I. Galka, Hans-Peter Hartung, Ludwig Kappos, Fred D. Lublin, Ruth Ann Marrie, Aaron E. Miller, David H. Miller, Xavier Montalban, Ellen M. Mowry, Per Soelberg Sorensen, Mar Tintoré, Anthony L. Traboulsee, Maria Trojano, Bernard M. J. Uitendaele, Sandra Vukusic, Emmanuelle Waubant, Brian G. Weinstenker, Stephen C. Reingold, Jeffrey A. Cohen

The 2010 McDonald criteria for the diagnosis of multiple sclerosis are widely used in research and clinical practice. Scientific advances in the past 7 years suggest that they might no longer provide the most up-to-date guidance for clinicians and researchers. The International Panel on Diagnosis of Multiple Sclerosis reviewed the 2010 McDonald criteria and recommended revisions. The 2017 McDonald criteria continue to apply primarily to patients experiencing a typical clinically isolated syndrome, define what is needed to fulfil dissemination in time and space of lesions in the CNS, and stress the need for no better explanation for the presentation. The following changes were made in patients with a typical clinically isolated syndrome and clinical or MRI demonstration of dissemination in space, the presence of CSF-specific oligoclonal bands allows a diagnosis of multiple sclerosis; symptomatic lesions can be used to demonstrate dissemination in space or time in patients with supratentorial, infratentorial, or spinal cord syndrome; and cortical lesions can be used to demonstrate dissemination in space. Research to further refine the criteria should focus on optic nerve involvement, validation in diverse populations, and incorporation of advanced imaging, neurophysiological, and body fluid markers.

Recommended Diagnostic Criteria for Multiple Sclerosis: Guidelines from the International Panel on the Diagnosis of Multiple Sclerosis

W. Ian McDonald, FRCP,¹ Alistair Compston, FRCP,² Gilles Edan, MD,³ Donald Goodkin,⁴ Hans-Peter Hartung, MD,⁵ Fred D. Lublin, MD,⁶ Henry F. McFarland, MD,⁷ Donald W. Paty, MD,⁸ Chris H. Polman, MD,⁹ Stephen C. Reingold, PhD,¹⁰ Magnhild Sandberg-Wollheim, MD,¹¹ William Sibley, MD,¹² Alan Thompson, MD,¹³ Stanley van den Noort, MD,¹⁴ Brian Y. Weinstenker, MD,¹⁵ and Jerry S. Wolinsky, MD¹⁶

The International Panel on MS Diagnosis presents revised diagnostic criteria for multiple sclerosis (MS). The focus remains on the objective demonstration of dissemination of lesions in both time and space. Magnetic resonance imaging is integrated with clinical and other paraclinical diagnostic methods. The revised criteria facilitate the diagnosis of MS in patients with a variety of presentations, including “monosymptomatic” disease suggestive of MS, disease with a typical relapsing-remitting course, and disease with insidious progression, without clear attacks and remissions. Previously used terms such as “clinically definite” and “probable MS” are no longer recommended. The outcome of a diagnostic evaluation is either MS, “possible MS” (for those at risk for MS, but for whom diagnostic evaluation is equivocal), or “not MS.”

Ann Neurol 2001;50:121–127

Diagnostic Criteria for Multiple Sclerosis: 2010 Revisions to the McDonald Criteria

Chris H. Polman, MD, PhD,¹ Stephen C. Reingold, PhD,² Brenda Banwell, MD,³ Michel Clanet, MD,⁴ Jeffrey A. Cohen, MD,⁵ Massimo Filippi, MD,⁶ Kazuo Fujihara, MD,⁷ Eva Havrdova, MD, PhD,⁸ Michael Hutchinson, MD,⁹ Ludwig Kappos, MD,¹⁰ Fred D. Lublin, MD,¹¹ Xavier Montalban, MD,¹² Paul O'Connor, MD,¹³ Magnhild Sandberg-Wollheim, MD, PhD,¹⁴ Alan J. Thompson, MD,¹⁵ Emmanuelle Waubant, MD, PhD,¹⁶ Brian Weinstenker, MD,¹⁷ and Jerry S. Wolinsky, MD¹⁸

New evidence and consensus has led to further revision of the McDonald Criteria for diagnosis of multiple sclerosis. The use of imaging for demonstration of dissemination of central nervous system lesions in space and time has been simplified, and in some circumstances dissemination in space and time can be established by a single scan. These revisions simplify the Criteria, preserve their diagnostic sensitivity and specificity, address their applicability across populations, and may allow earlier diagnosis and more uniform and widespread use.

ANN NEUROL 2011;69:292–302

2024 vs 2017: Revisions Diagnostic Criteria

2017 McDonald Criteria

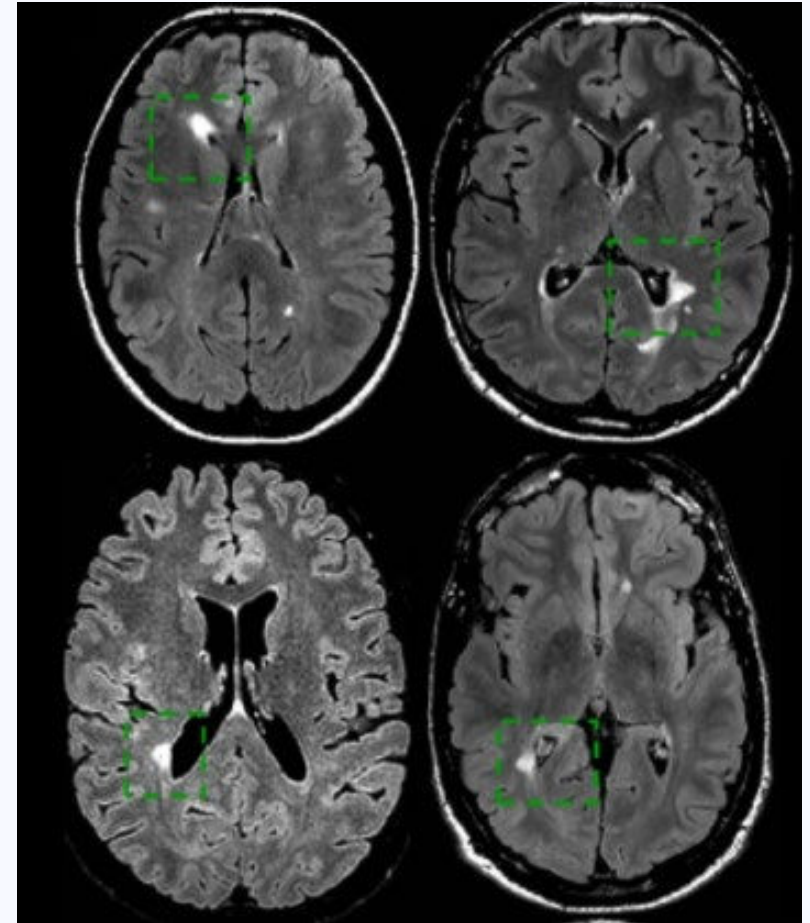
Clinical Presentation	Additional Data Needed for Diagnosis
≥ 2 clinical attacks and objective evidence of ≥ 2 lesions	None
≥ 2 clinical attacks and objective evidence of 1 lesion	DIS: an additional attack implicating a different CNS site OR by MRI ^a
1 clinical attack and objective clinical evidence of ≥ 2 lesions	DIT: an additional clinical attack OR by MRI ^b OR CSF-specific oligoclonal bands
1 clinical attack and objective evidence of 1 lesion	DIS: an additional clinical attack implicating a different CNS site OR by MRI ^a OR DIT: an additional clinical attack OR by MRI ^b OR CSF-specific oligoclonal bands

2024 Proposed revisions

- **Typical symptoms are no longer required to make the diagnosis of MS**
- **Inclusion of optic nerve as a separate topographic site**
- **Increased emphasis on spinal cord lesions**
- **Expanded use of biomarkers**
 - Kappa free light chains (kFLC) in the CSF
- **Use of novel MRI findings**
 - Central vein sign
 - Paramagnetic rim lesions

In 2024, typical symptoms are no longer required to make a diagnosis of MS

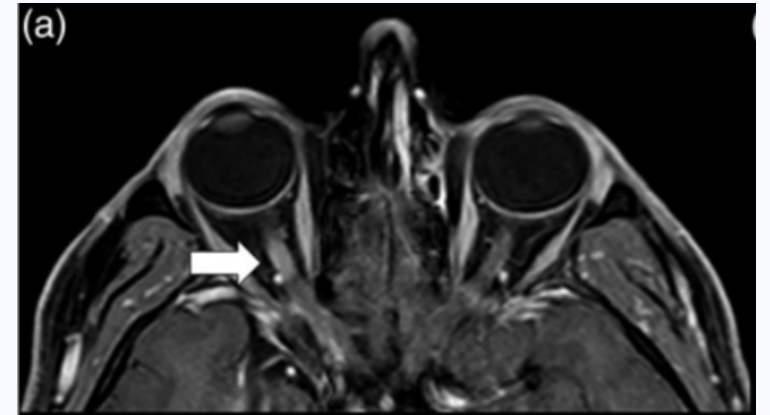
- The recent proposed revisions to the McDonald criteria emphasize the importance of imaging the diagnosis of MS.
- This means that **an abnormal MRI showing typical lesions is required to make a diagnosis of MS.**
- People with no clinical symptoms and with incidental MRI findings suggestive of MS (previously **radiologically isolated syndromes**) can now be diagnosed with MS under certain conditions. In these situations, it is critical to rule out other causes and ensure the specificity of MRI findings.



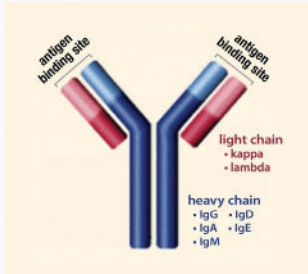
Brain MRI showing typical MS periventricular lesions

Inclusion of the Optic Nerve as a New Topography

- Fifth anatomical location to demonstrate dissemination in space (DIS).
- Optic nerve lesions with no better explanation identified by **MRI** may serve as evidence of optic nerve involvement to demonstrate DIS.
- **Visual evoked potentials** (significant interocular asymmetry or p100 peak time above upper limit of normal with no better explanation) may serve as evidence of optic nerve involvement.
- **OCT** (Optical Coherence Tomography) may serve as evidence of optic nerve involvement.



MRI of the orbits showing typical optic nerve involvement



Novel CSF Biomarker: Kappa Free Light Chains

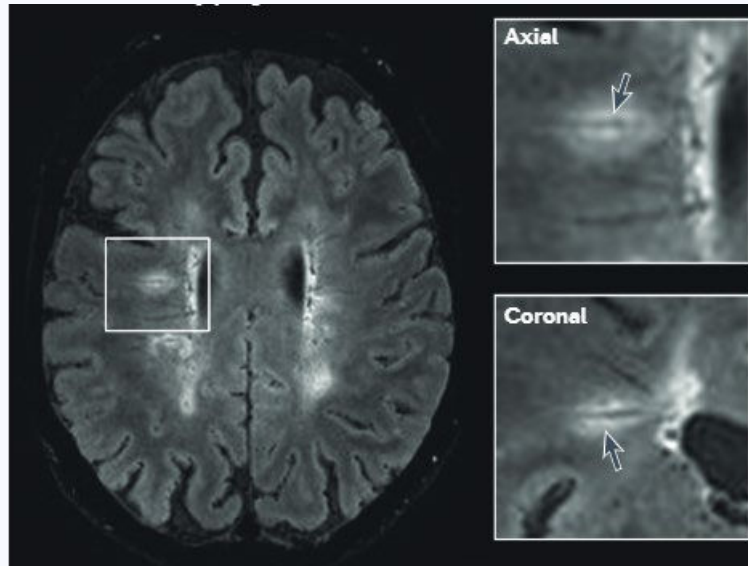
Table 3. Sensitivity, specificity, positive and negative predictive value for elevated KFLC, MRI parameters and OCB regarding conversion of clinically isolate syndrome to definite multiple sclerosis.

	N	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Q KFLC	77	86.8 (71.9–95.6)	38.5 (23.4–55.4)	57.9 (44.1–70.9)	75.0 (50.9–91.3)
OCB	77	92.1 (78.6–98.3)	33.3 (19.1–50.2)	57.4 (44.1–70.0)	81.3 (54.4–96.0)
Intrathecal IgG-Synthesis	76	43.2 (27.1–60.5)	64.1 (47.2–78.8)	53.3 (34.3–71.7)	54.3 (39.0–69.1)
IgG-Index >0.70	76	43.2 (27.1–60.5)	64.1 (47.2–78.8)	53.3 (34.3–71.7)	54.3 (39.0–69.1)
Barkhof	66	12.5 (3.5–29.0)	88.2 (72.5–96.7)	50.0 (15.7–84.3)	51.7 (38.2–65.0)

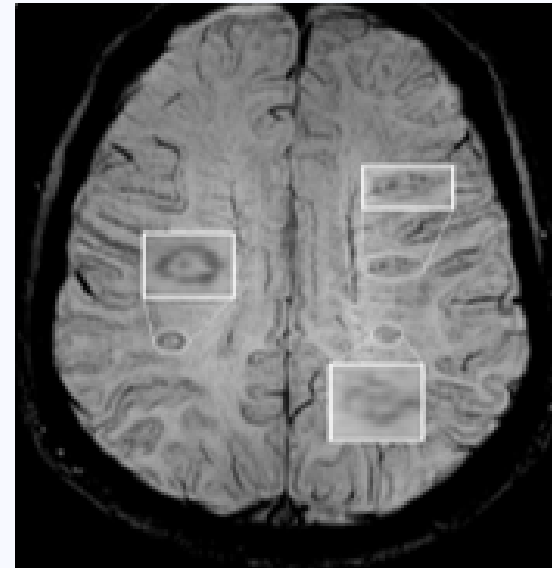
- KFLC are produced during chronic intrathecal inflammation and differentiate CIS/MS from other neurological diseases/controls
- KFLC represent a valid, easier and rater-independent alternative to oligoclonal band (OCB) detection
- The presence of Kappa free light chains in the CSF is now considered interchangeable with OCBs and can substitute OCBs for the diagnosis of MS.

Novel MRI Biomarkers

The inclusion of novel imaging features has been proposed to **increase the specificity of the new MS** diagnostic criteria and help differentiate MS lesions from other etiologies.



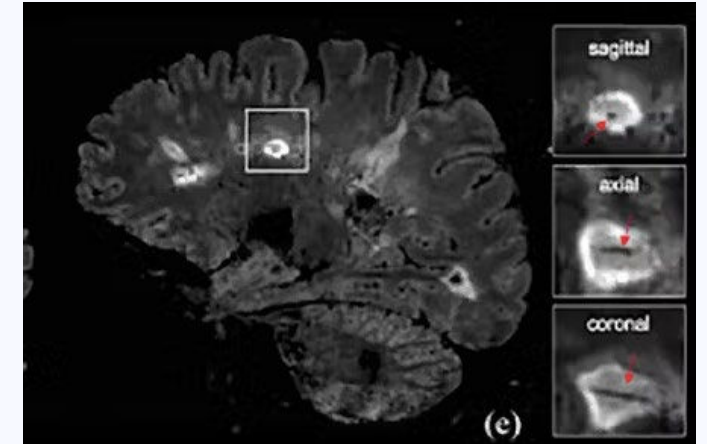
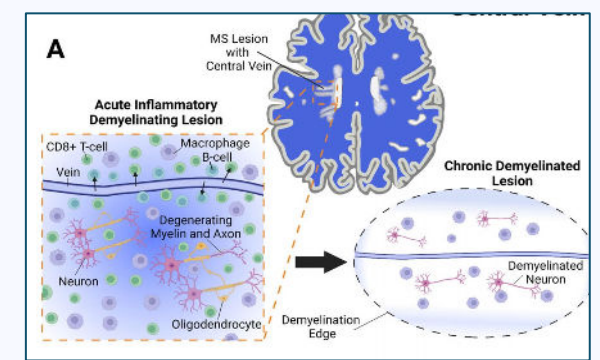
Central Vein Sign (CVS)



Paramagnetic Rim Lesions (PRLs)

Central Vein Sign (CVS)

- The presence of central veins inside MS lesions is a well-established finding in histopathological studies
- The venocentric distribution of lesions exists in all MS clinical phenotypes (RRMS, SPMS and PPMS) and is not frequently seen in other conditions (NMOSD, chronic small vessel disease, migraines, Susac)
- **Best visualized using T2*-based sequences**
 - Susceptibility-weighted imaging (SWI): currently most available
 - FLAIR* imaging combines T2* and FLAIR and provides better detection (see image), but is not routinely available
- **The presence of 6 CVS-positive lesions can be used to support the diagnosis of MS but is not required if enough criteria are met.**



- Thin hypointense line or small dot
- Visualized in at least two perpendicular planes and appears as a thin line in at least one plane
- Small apparent vein diameter (<2mm)
- Runs partially/entirely through the lesion
- Positioned centrally in the lesion

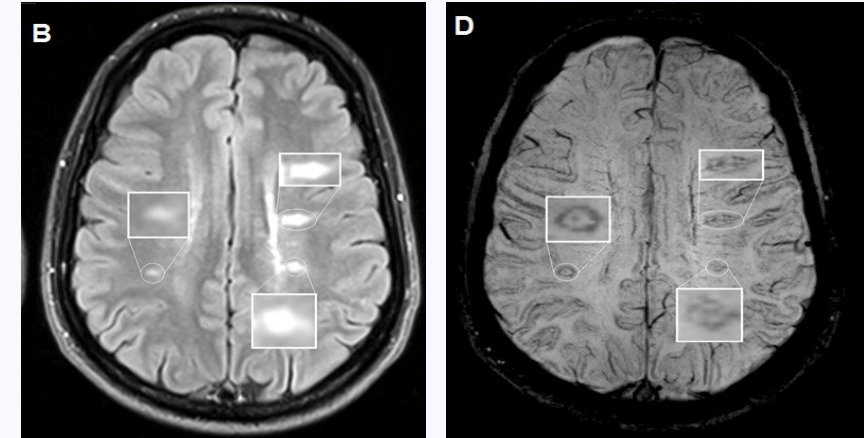
Paramagnetic Rim Lesions (PRLs)



Iron

Microglia, Macrophages

- PRLs reflect **perilesional chronic inflammation**, in particular residual and detrimental iron-laden microglia/macrophage accumulation at the lesion edge after acute inflammation subsides.
- **Best visualized using susceptibility-sensitive sequences: SWI, T2*, etc...**
- PRLs are frequent in MS (50% of patients have at least 1 PRL) and rare in other conditions (~7% in non-MS cases)
- ≥ 1 PRL in SWI has high specificity (99.7%) and low sensitivity (24%)
- **The presence of ≥ 1 PRL can be used to support the diagnosis of MS but is not required if enough criteria are met.**



- T2-hyperintense core that does not enhance with GBCA and is surrounded by a paramagnetic rim along at least two-thirds of the lesion perimeter
- The rim co-localizes with the edge of all or part of a lesion core that is hyperintense on T2
- The rim is discernible on at least two consecutive slices (2D acquisition) or in two orthogonal planes (3D acquisition)

Signs or Symptoms suggestive of MS

Initial workup suggestive of MS?

- Medical history and clinical examination
- Imaging and laboratory tests
- Differential diagnosis

No

MS diagnosis should only be considered after ruling out better explanations

Yes

Lesions present in ≥ 2 CNS topographies
or
Patients with ≥ 12 months progression and ≥ 2 spinal cord lesions¹

Multiple Sclerosis may be diagnosed if one or more of the following are also demonstrated:

- CSF+²
- CVS+³
- Dissemination in Time⁴
- Lesions are present in 4 or 5 CNS topographies

Lesions present in 1 CNS topography
(including patients with ≥ 12 months progression)

Multiple Sclerosis may be diagnosed if one or more of the following are also demonstrated:

- CSF+ and CVS+
- CSF+ and PRL+⁵
- Dissemination in Time and CVS+
- Dissemination in Time and PRL+

Key Takeaways



1

The new MS diagnosis criteria emphasize the **central role of MRI** and paraclinical tests in diagnosing MS even in the absence of clinical symptoms.

2

It is essential to recognize typical vs atypical clinical and radiologic presentations. MS remains a **diagnosis of elimination**, and other causes need to be ruled out, particularly in atypical cases.

3

Lesions in **5 topographies**, including the optic nerve, are now considered for the diagnosis of MS. Additional MRI markers, such as the **central vein sign** or the presence of **paramagnetic rim lesions**, can be used to increase the specificity of the new criteria. These are strongly recommended in individuals over age 50.

Differential Diagnosis of MS



Autoimmune

- NMOSD
- MOGAD
- Sjogrens
- SLE
- Sarcoidosis
- CNS Vasculitis

Infectious/ Malignancy

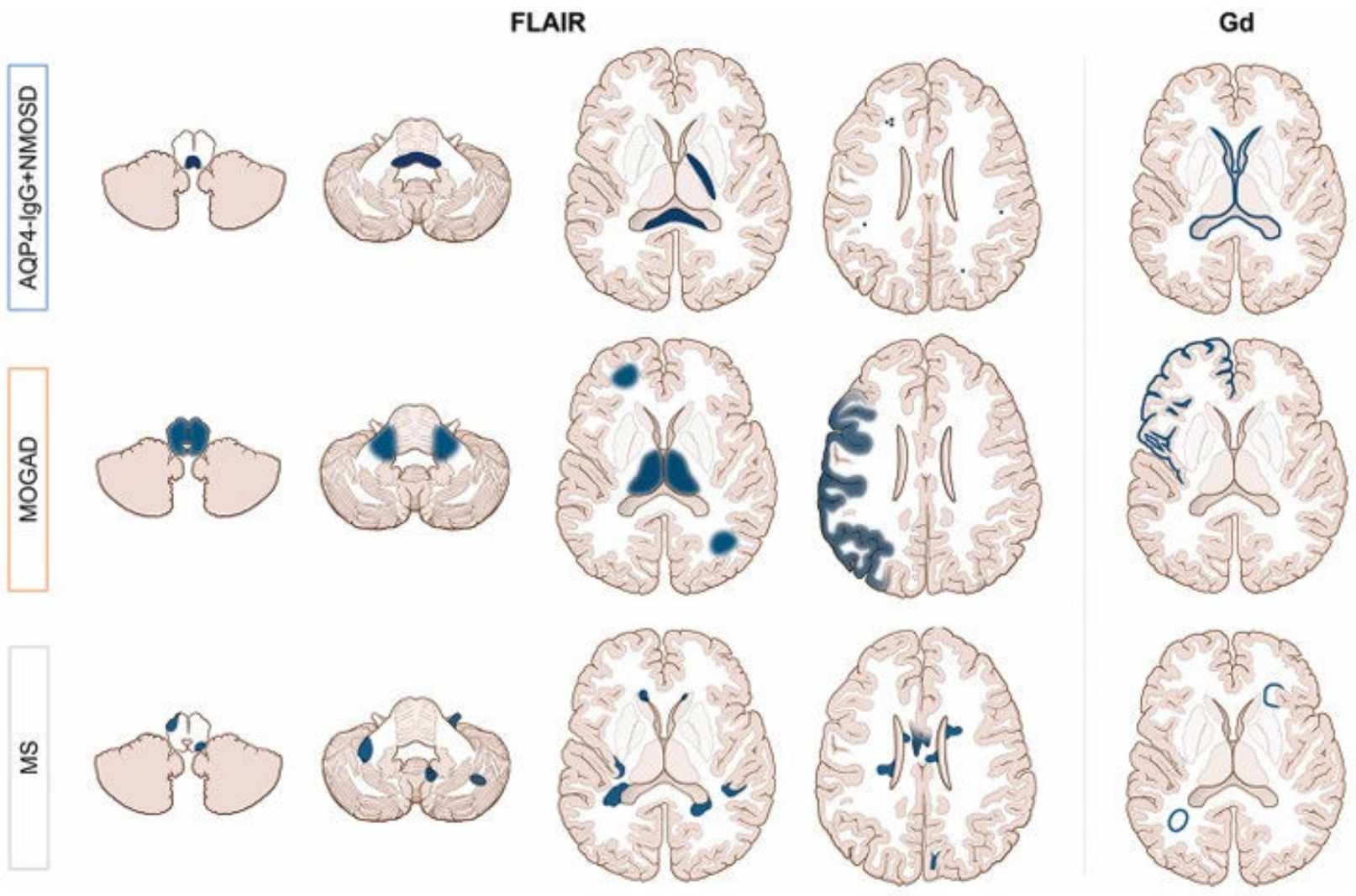
- Lyme
- Tuberculosis
- Neurosyphilis
- CNS Lymphoma

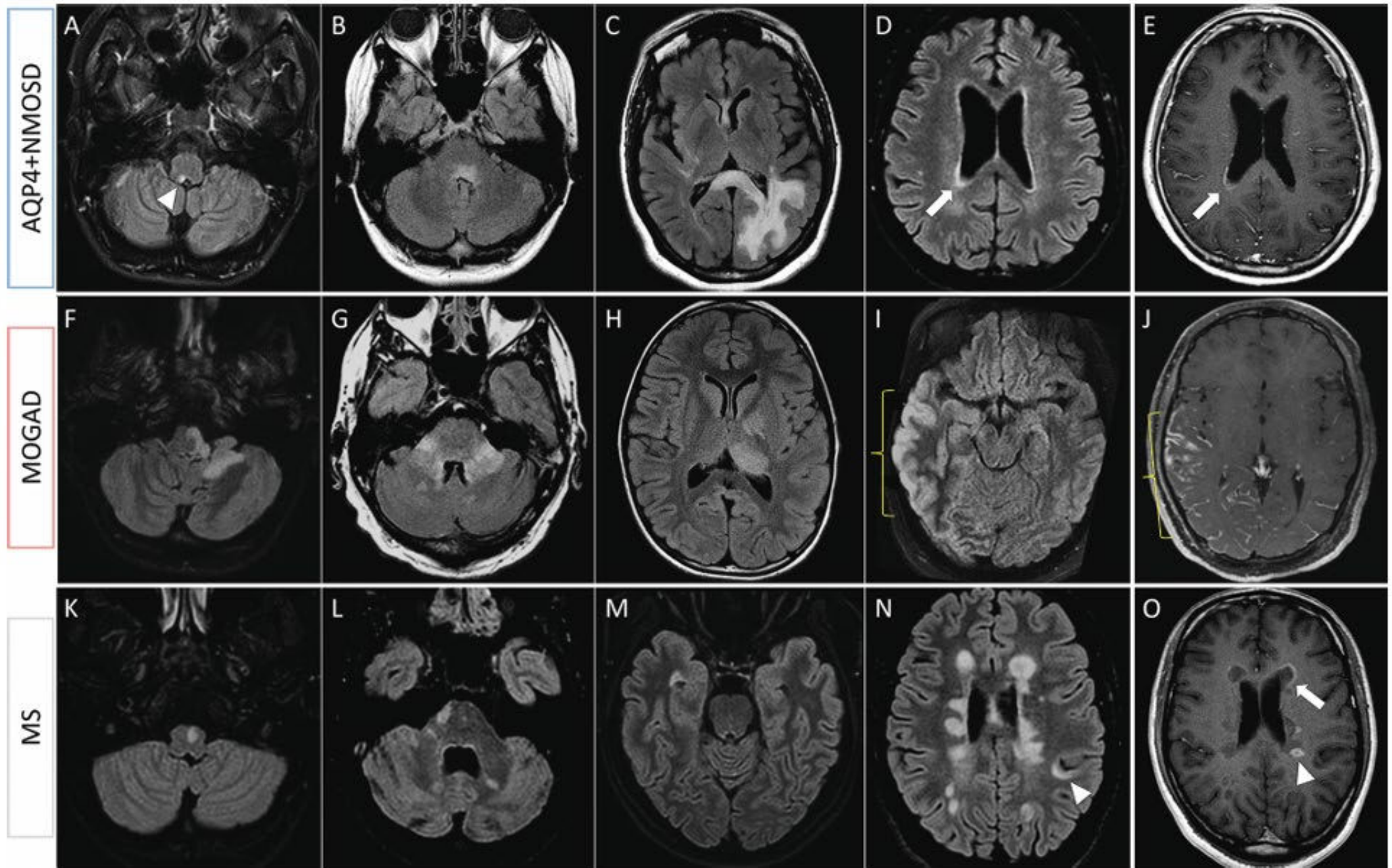
Metabolic

- Vitamin B12 Deficiency
- Vitamin E deficiency
- Copper deficiency

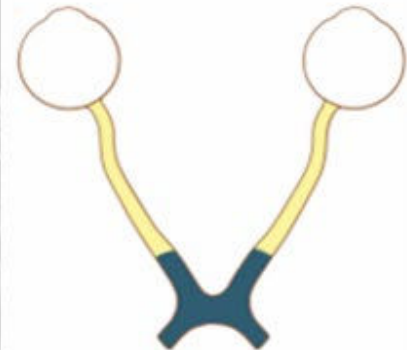
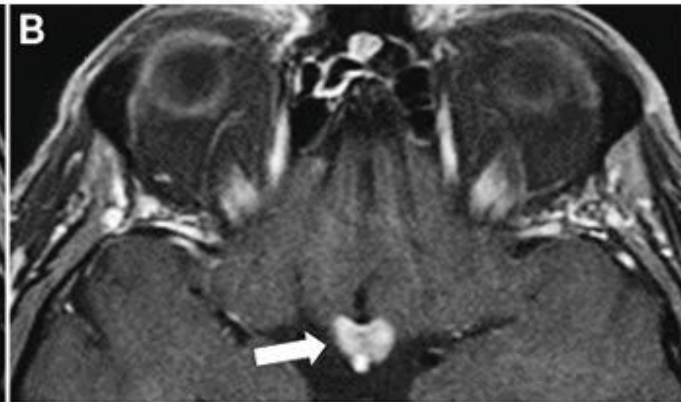
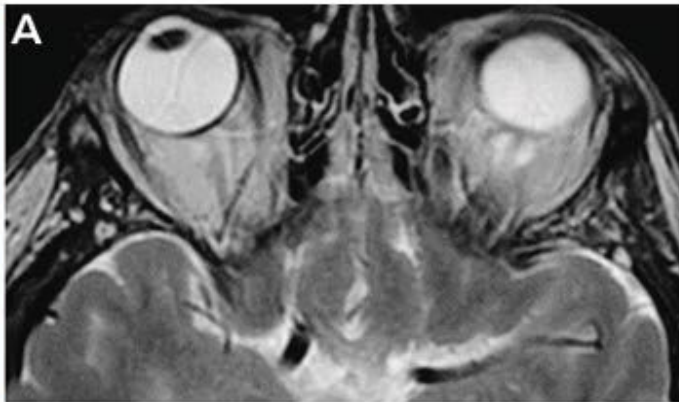
MS vs NMO vs MOGAD: Differences in Presentation

	NMOSD	MOGAD	MS
Demographics	30-50yo, Females 9:1	0-40yo, M=F	20-40, Females 3:1
Antecedent Infection	Infrequent	Common	Infrequent
Disease Course	Relapsing	Relapsing 50%, Monophasic 50%	Relapsing, progressive
Optic Neuritis	Bilateral, Chiasm or Posterior	Bilateral, Anterior (likely to have papilledema)	Unilateral, Short segment
Transverse Myelitis	LETM	LETM (frequent conus medullaris involvement)	Multiple short lesions, periphery of cord
Other	Area Postrema Syndrome Narcolepsy	ADEM Cerebral Cortical Encephalitis	INO, Trigeminal neuralgia, 6 th nerve palsy, asymmetric weakness
Clinical Recovery	Risk for poor recovery	Generally good (can have residual Bowel/Bladder)	Generally good
MRI Recovery	Rare	50-80%	Rare

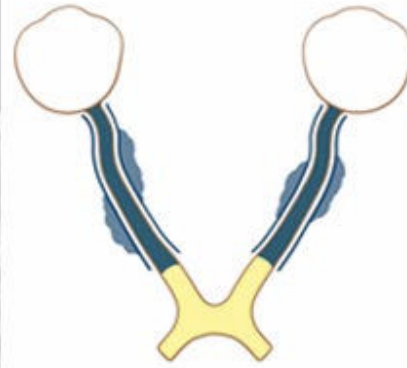
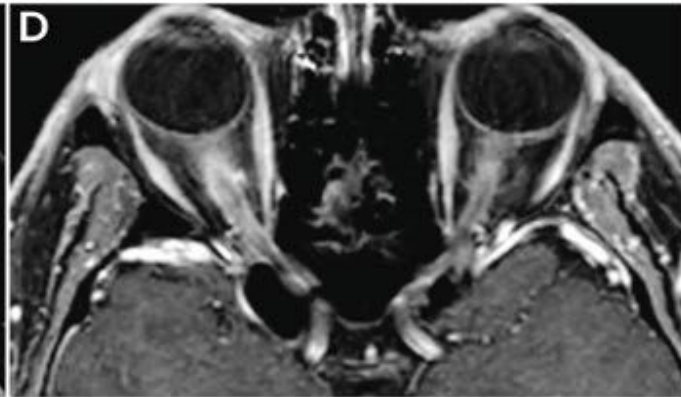
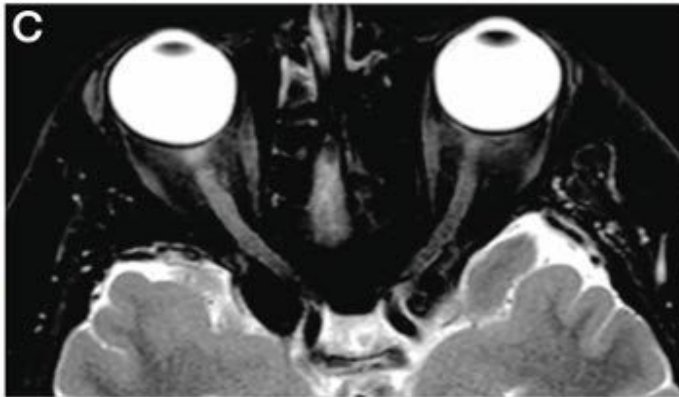




AQP4+NMOSD



MOGAD



MS

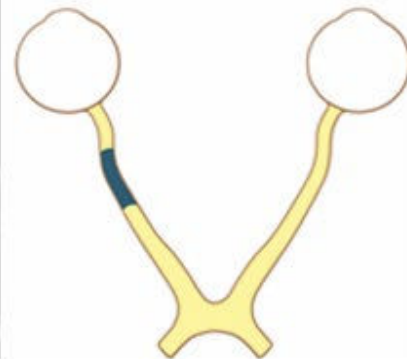


FIGURE 4.1

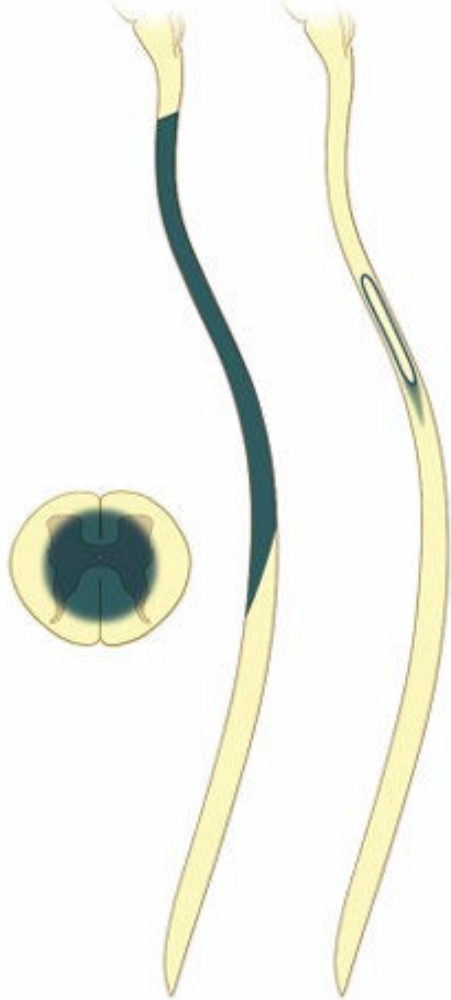
AQP4+NMOSD

MOGAD

MS

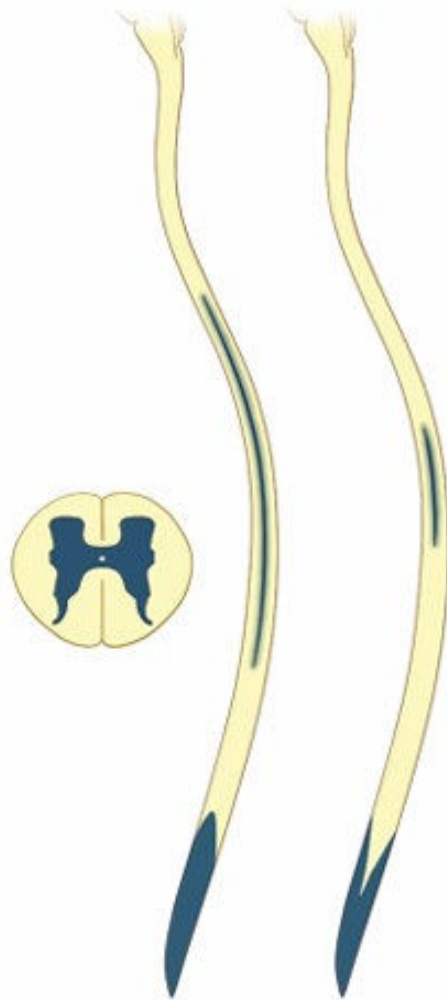
T2

Gd



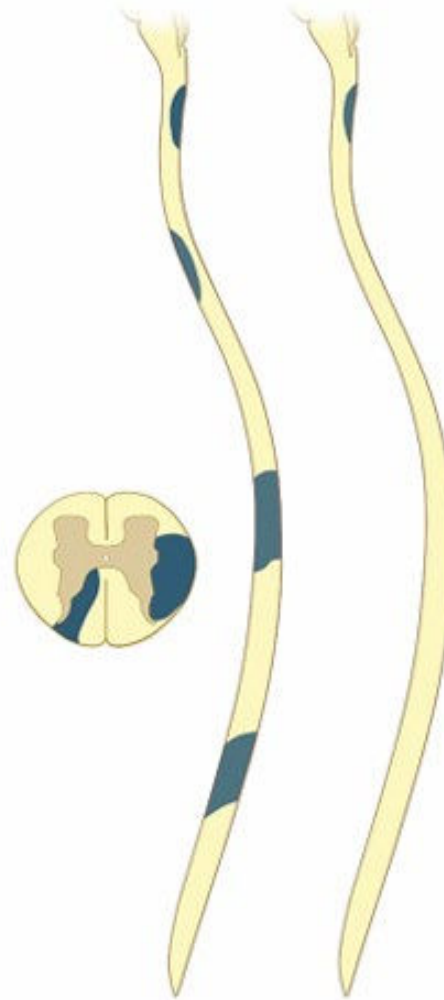
T2

Gd



T2

Gd



NMOSD: LETM

- Bright, spotty lesions
- Elongated ring or patchy enhancement

MOGAD: LETM +/-

- Short lesions, Conus, H-sign (axial), 50% enhancement acutely

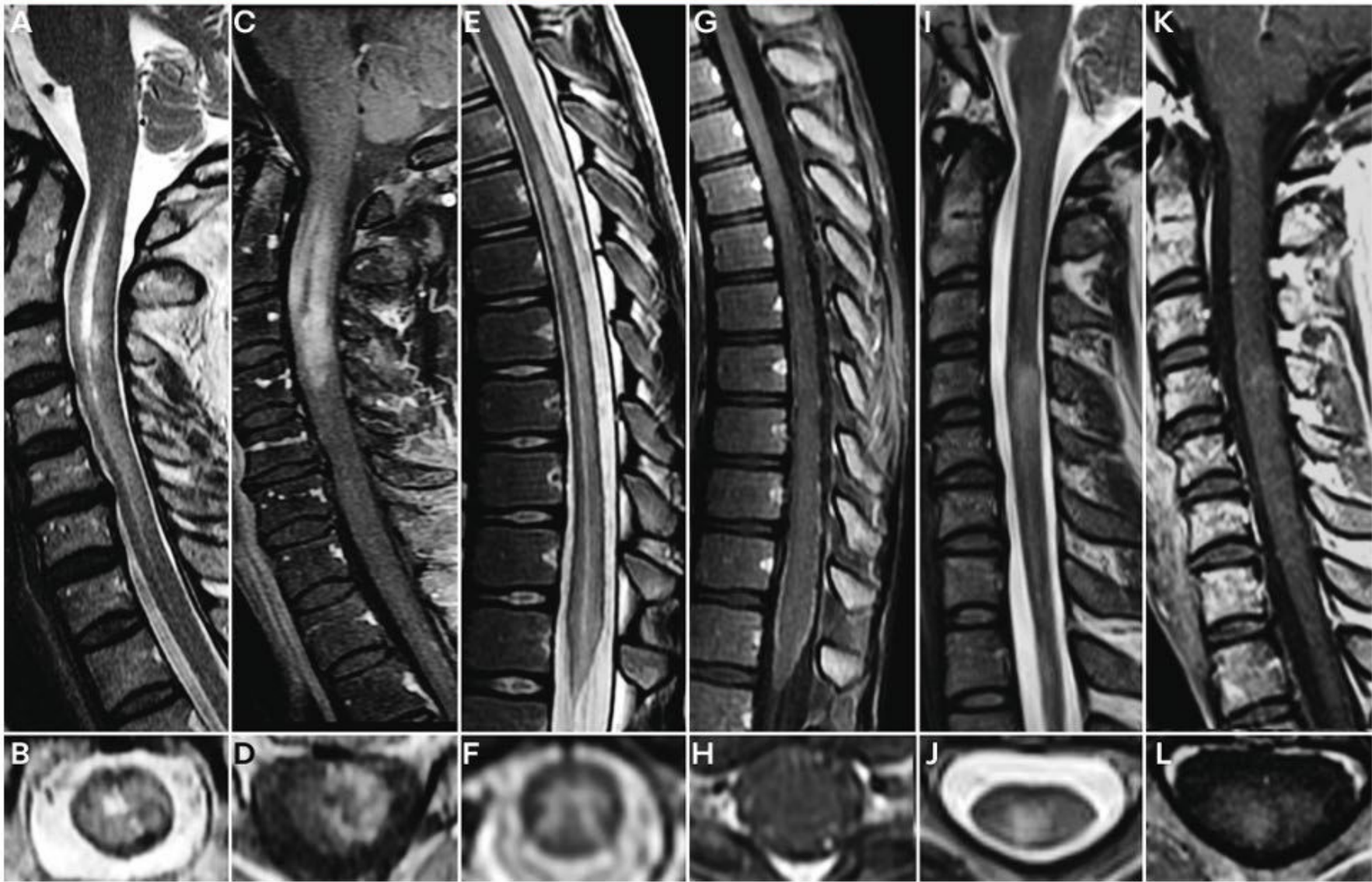
MS:

- Multiple short lesions, periphery of cord, ring or nodular enhancement

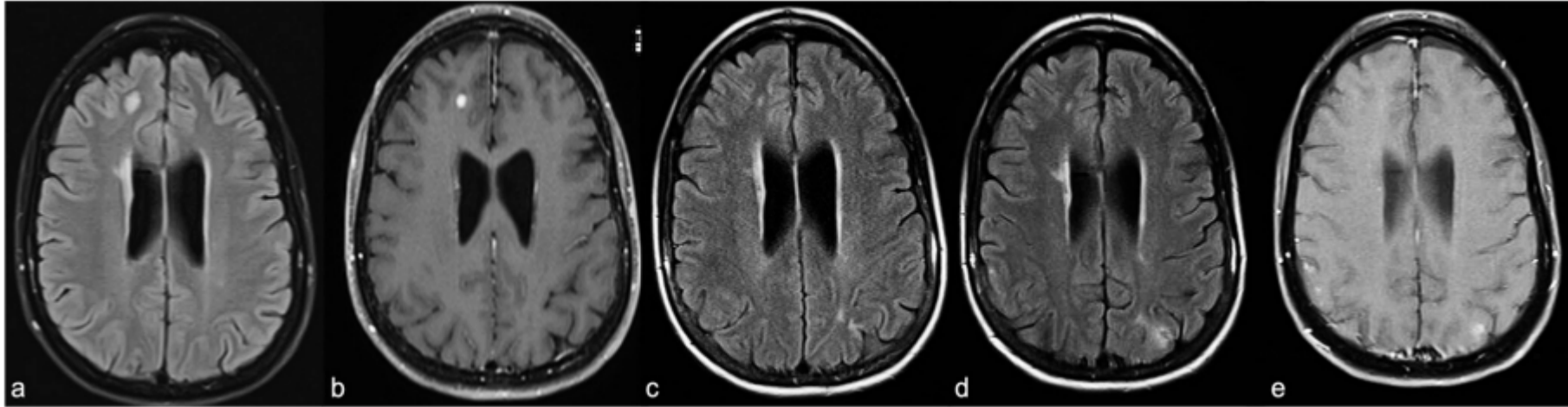
AQP4+NMOSD

MOGAD

MS



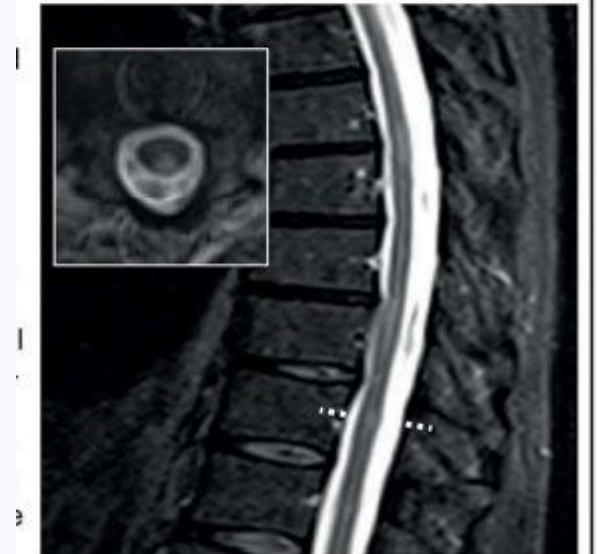
Sjogrens



- **Presentation:** Myelitis, optic neuritis, cognitive
- Sicca symptoms not always present
- **Brain lesions:** can be similar to MS
- **Cord:** central > peripheral, can have diffuse dorsal column involvement, short or long

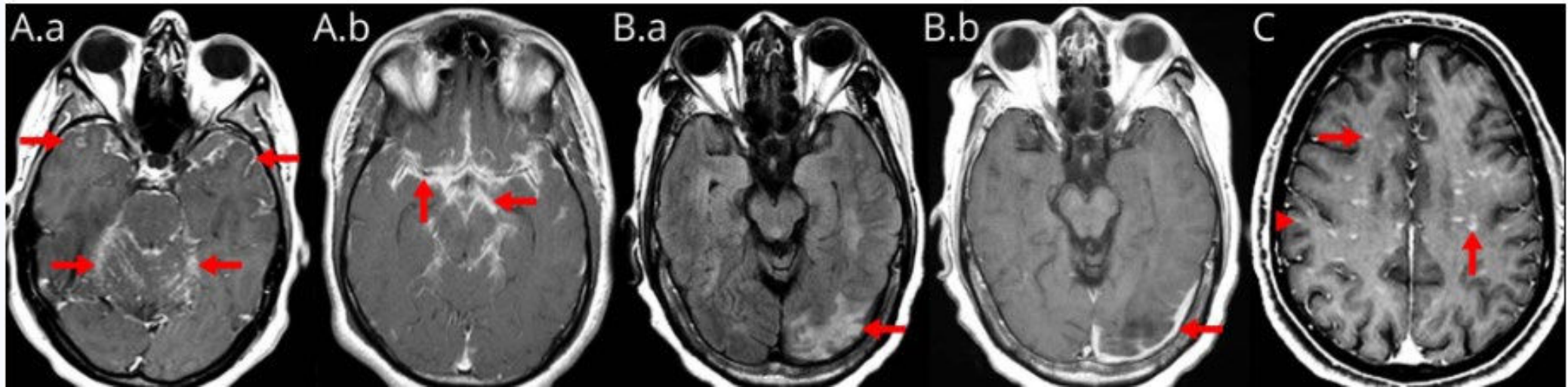


Representative sagittal and axial T2 weighted cervical MR image



Representative sagittal and axial T2 weighted spinal MR image

Neurosarcoidosis



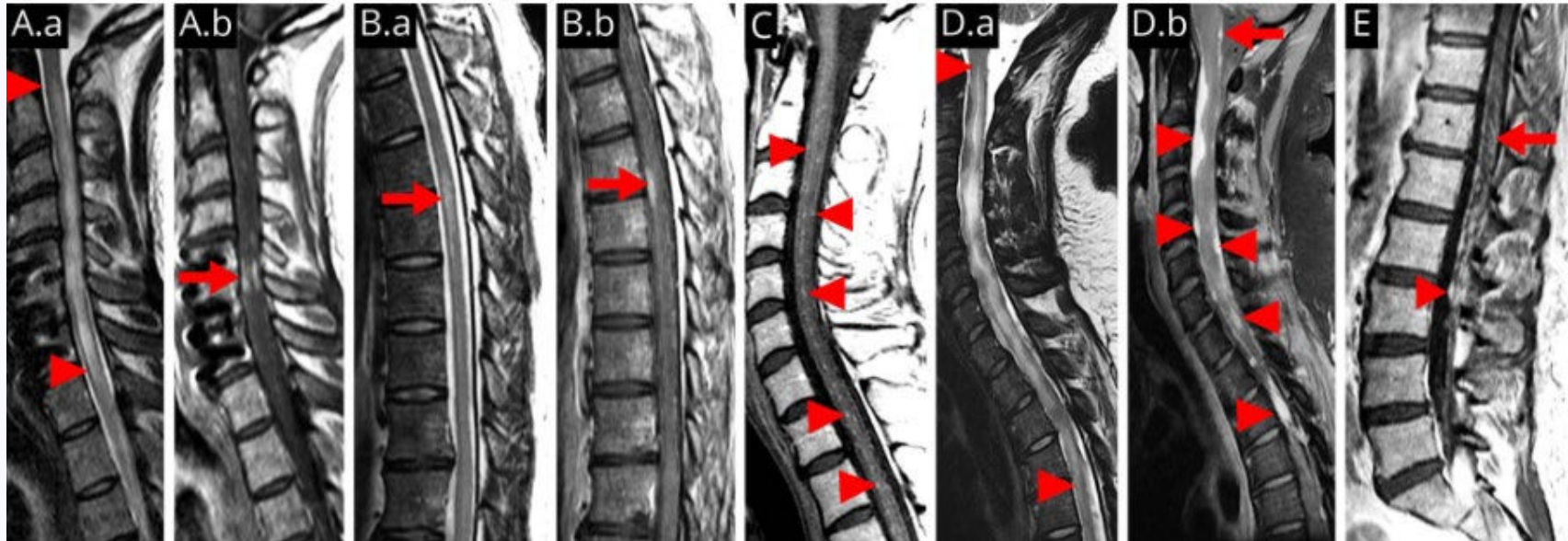
Leptomeningeal enhancement of the base of the brain

Mass-like lesion with dural thickening and enhancement

Perivascular enhancement

- **Brain manifestations:** headaches, cranial neuropathy, focal deficits
- **MRI:** Pachy- or leptomeningeal enhancement (often diffuse), cranial nerve infiltration, mass-like lesions, perivascular meningeal enhancement
- **CSF:** mild to moderate pleocytosis (usually <100 cells/ μ L), with lymphocyte predominance and elevated protein, is typical; OCBs can be seen in 20–40% of cases, non-specific.
- **CT chest:** look for mediastinal lymphadenopathy

Neurosarcoidosis



LETM with contrast enhancement

Short segment myelitis with contrast enhancement

Perivascular enhancement

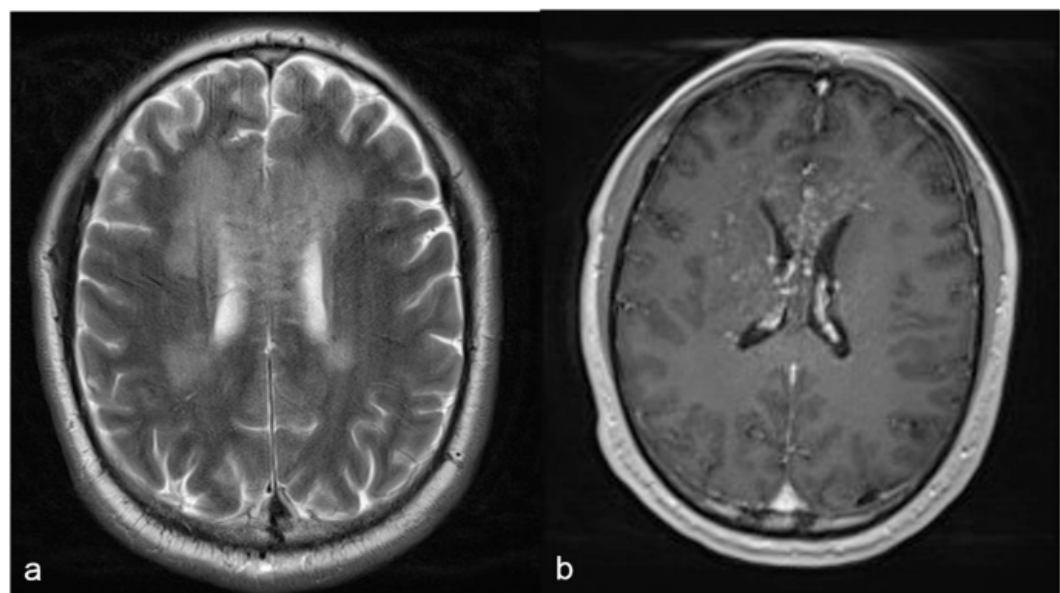
Extra-axial masses compressing the cord

Lumbo-sacral radiculomyelitis

Trident sign

- Spinal cord presentations are common
- **MRI:** diverse patterns involving spinal cord, meningeal and perivascular spaces
- **Trident sign:** key diagnostic feature

CNS Vasculitis

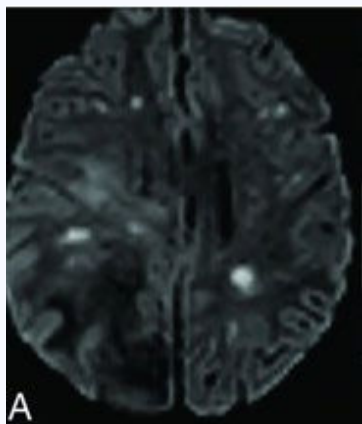
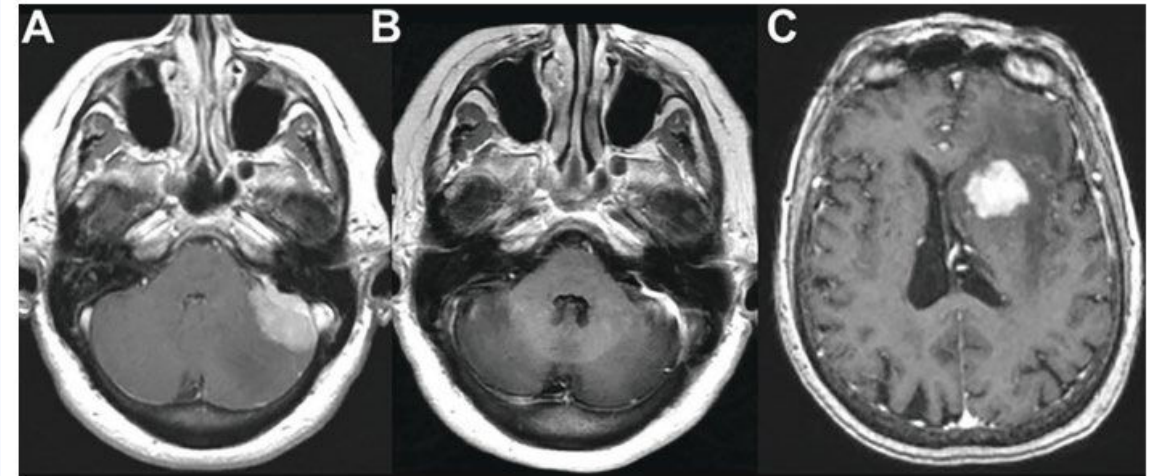


- **Presentation:** subacute onset headaches, cognitive changes, focal neurologic symptoms, seizures
- **MRI:**
 - Diffuse T2 hyperintensity
 - Punctate pattern of enhancement (perivascular)
 - Meningeal enhancement
 - Multifocal areas of infarction
- **Angiography:** frequently negative particularly when only small vessels are involved
- **CSF:** lymphomonocytic pleiocytosis, elevated protein, rare OCBs
- **Parenchymal and meningeal biopsy is the gold standard for diagnosis**

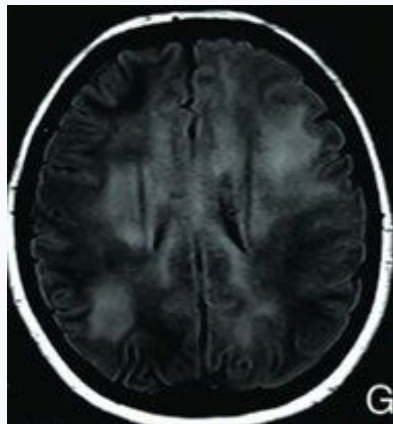
Primary and Intravascular CNS Lymphoma

Primary CNS Lymphoma

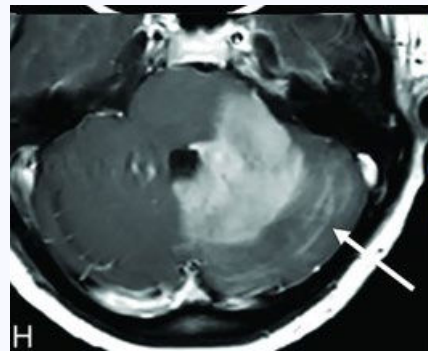
- Symptom development over weeks (headaches, seizures, neurologic symptoms)
- Typically, single lesion, supratentorial (87%) with homogeneous enhancement



Acute patched ischemia-like lesions on DWI



Diffuse WM Hyperintensities



Cerebellar enhancement with associated leptomeningeal disease

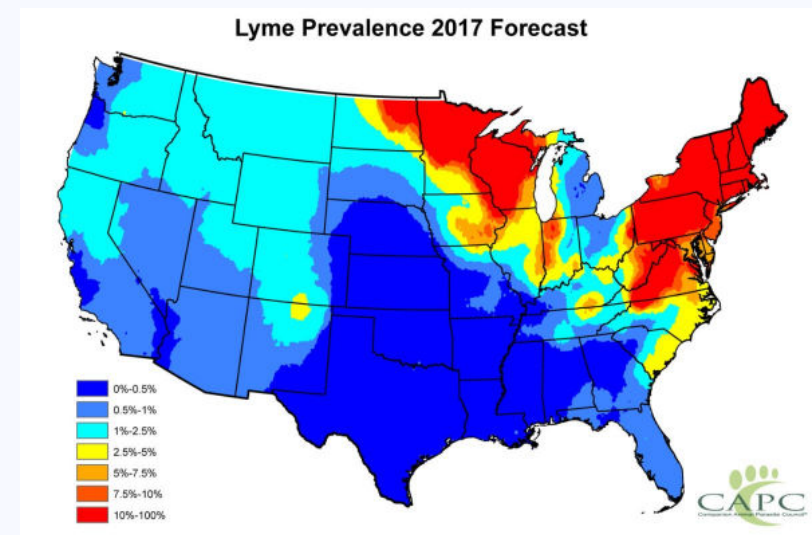
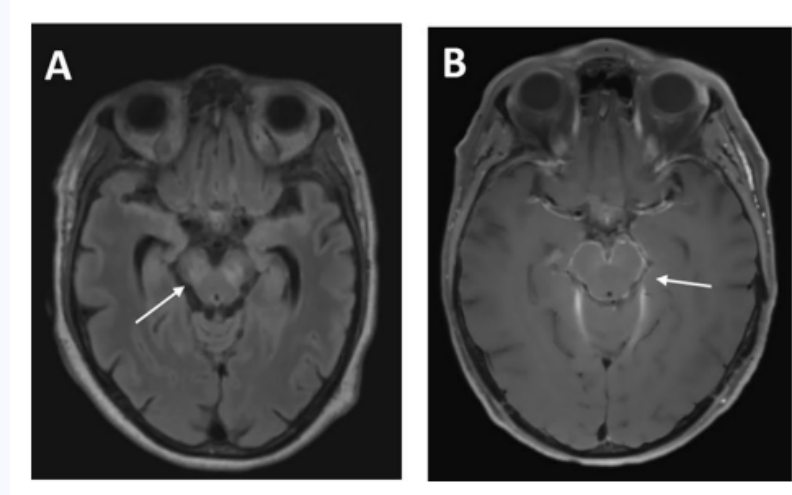
Intravascular Lymphoma

- Selective proliferation of malignant B large-tumor cells within the brain vessels
- Subacute headaches, cognitive changes, stroke-like episodes
- MRI can show diffuse WM changes

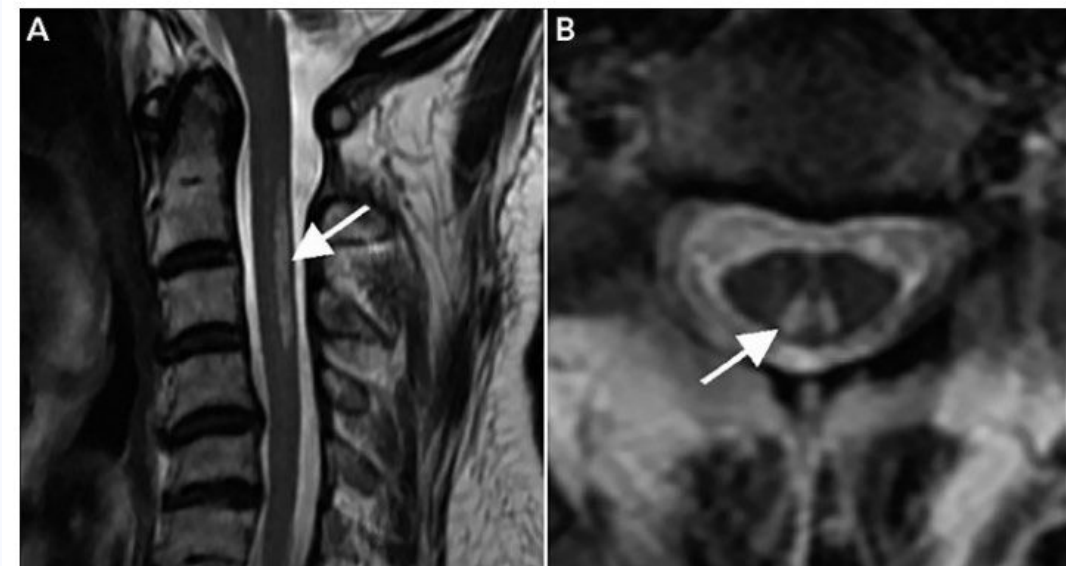
Infectious Causes

Lyme Disease:

- Bilateral perimesencephalic hyperintensities
- Leptomeningeal enhancement



Metabolic



Myelopathy with dorsal column involvement:

- B12 deficiency
- Vitamin E deficiency
- Copper deficiency

Red Flags



- Age > 50 – Be careful and always rule out other causes
- Bilateral Optic Neuritis, poor visual recovery
- Leptomeningeal Enhancement
- Encephalopathy
- Intractable nausea, vomiting, hiccups
- Systemic symptoms (fever, weight loss, joint pain, narcolepsy, headache/meningismus)
- Persistent enhancement > 6mo or interval resolution on MRI
- LETM
- Atypical CSF findings

Q+A



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