

IMAGING of ACUTE MYELOPATHY

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MYELOPATHY = broad term referring any lesion of the spinal cord

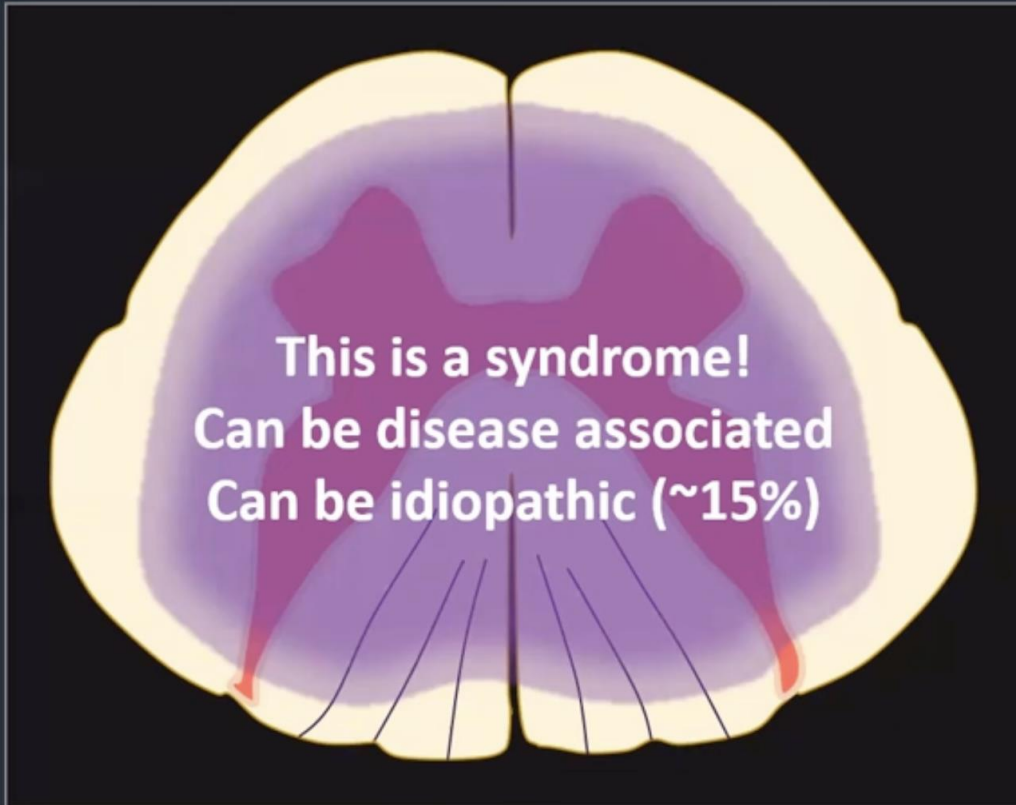
Time to reach maximum deficit

- **Acute myelopathy:** Symptoms progress to their worst within 21 days
- **Subacute myelopathy:** Symptoms progress over weeks to months
- **Chronic myelopathy:** Symptoms progress over months to years

Cause :

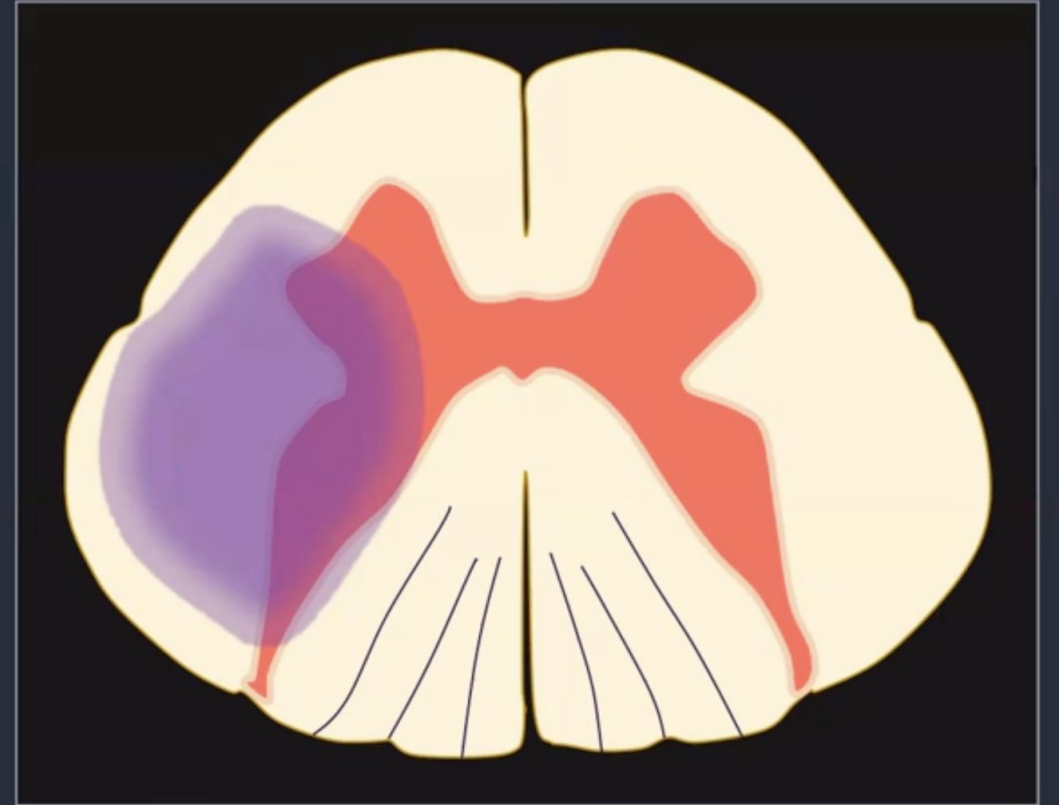
- **Traumatic myelopathy**
- **Non-traumatic myelopathy:** Caused by ischaemic, infectious, inflammatory, nutritional, or metabolic causes
- **Systemic disease:** Such as Sjögren's syndrome or systemic lupus erythematosus
- **Delayed radiation myelopathy (DRM)**
- **Spinal cord infarct (SCI)**

Acute transverse myelitis



Clinical presentation of acute myelitis in which **both halves of the spinal cord** are involved, resulting in bilateral sensory or motor changes

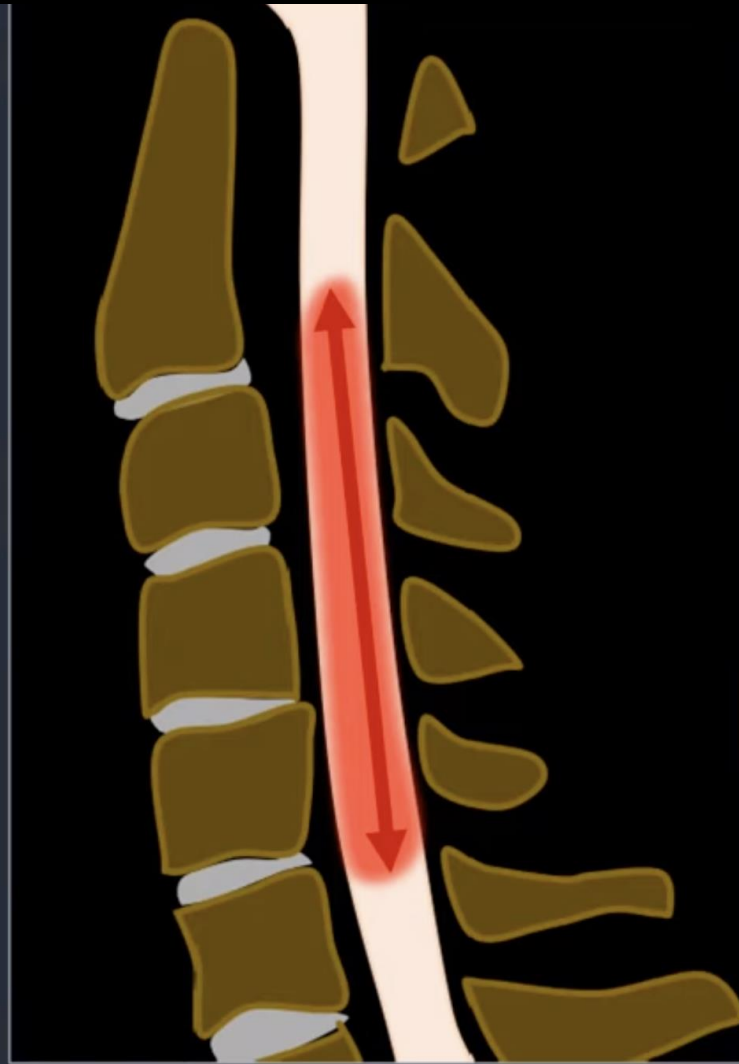
Acute partial myelitis



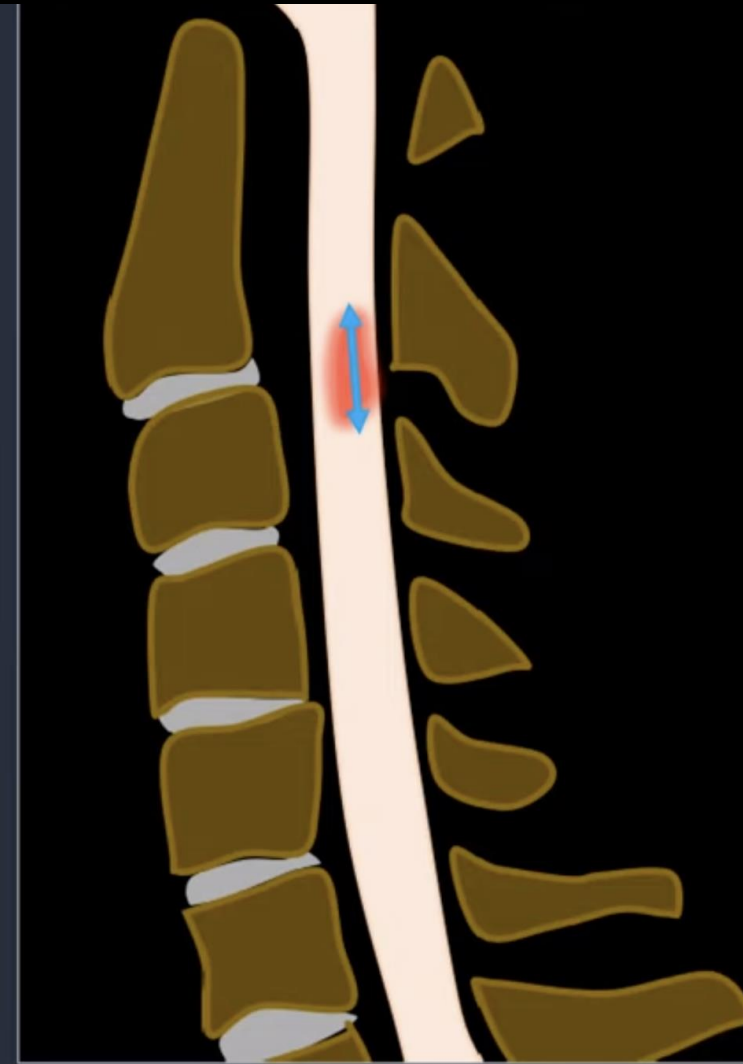
Myelitis limited to **one side of the spinal cord or one particular tract**; more commonly seen in specific diseases such as multiple sclerosis

* MYELITIS = spinal cord inflammation

≥ 3 vertebral
segments



Longitudinally
extensive myelitis



< 3 vertebral
segments

Short segment
(extensive) myelitis

Cause :

COMPRESSIVE

VS

NON COMPRESSIVE

INFLAMMATORY

VS

NON

INFLAMMATORY

Just the Facts: ED approach to Myelopathy

Drs. Zachary Macdonald, Emma Ferguson, Hans Rosenberg

What is a myelopathy?

A condition characterized by damage or dysfunction of the spinal cord, disrupting signal transmission, and leading to a broad range of clinical symptoms.

Etiologies

Compressive

- degenerative changes
- trauma
- tumors
- vascular malformations
- abscess

Non-Compressive

myelitis from:

- viral/bacterial/fungal
- toxin-induced
- radiation-induced
- metabolic (e.g. vitamin B12 defi)
- paraneoplastic

Signs and Symptoms

Autonomic Dysfunction

AND/OR

Sensory

- pain/temperature
- proprioception/vibration

AND/OR

Upper Motor Neuron

- inc tone, clonus, reflexes
- dec power
- Babinski/Hoffman Sign

Investigations

MRI is necessary to distinguish between compressive and non-compressive etiologies. Clinical signs and symptoms are NOT sufficient.

Vascular

**Spinal cord
infarction**

*Nadir within 4 hours
(Hyper-)acute*

Inflammatory

**Partial or
transverse
myelitis**

*Nadir 4h-21d
Acute*

Acute Myelopathy

Demyelination

MS

ADEM

NMOSD

MOGAD

(Para-)infectious

Direct infection

Postinfectious

**Systemic
inflammatory**

Lupus

Sjögren

Behçet

Sarcoid

Idiopathic

1. T2-hyperintensity: real or artifact ?

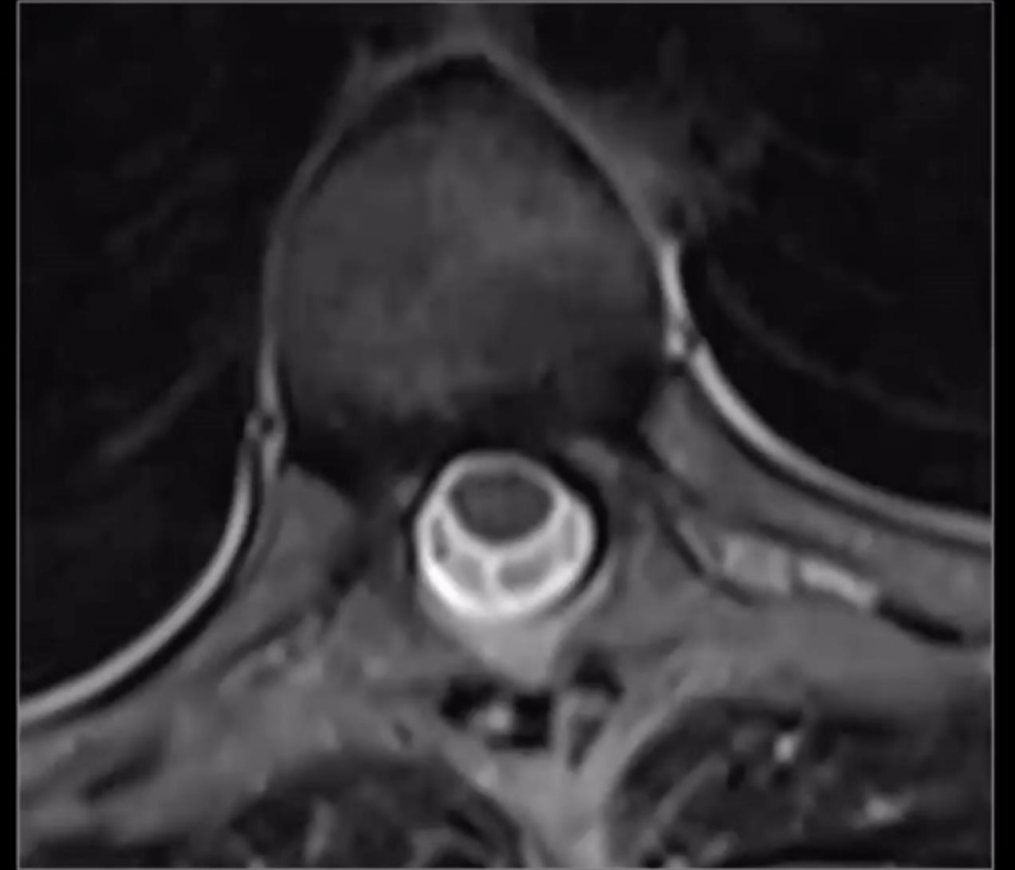
2. Is there any spinal cord compression ?

3. Acute or subacute/ chronic onset ?

T2-hyperintensity: real or
artifact ?



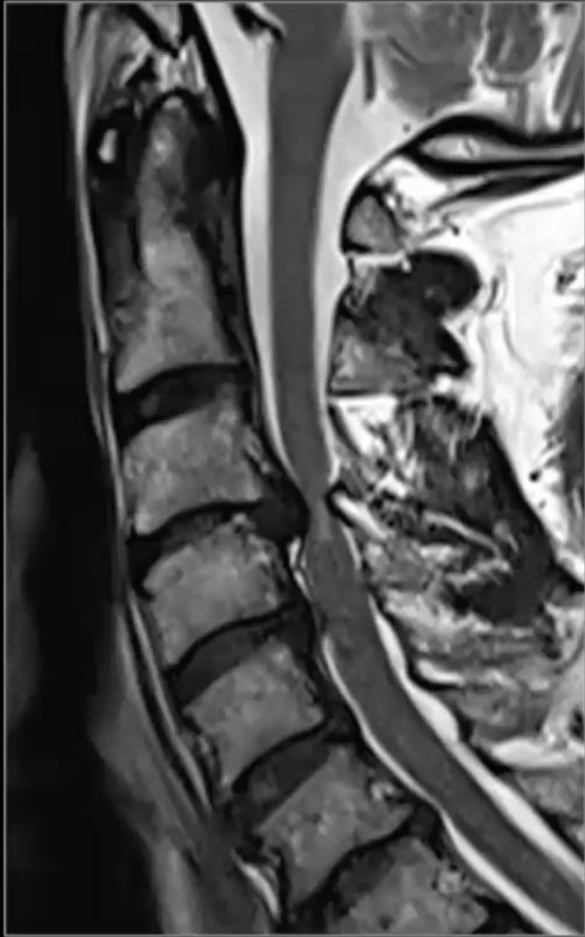
Spinal cord imaging prone to artifact – due to truncation/ Gibbs artifact (difference interface between CSF & spinal cord).



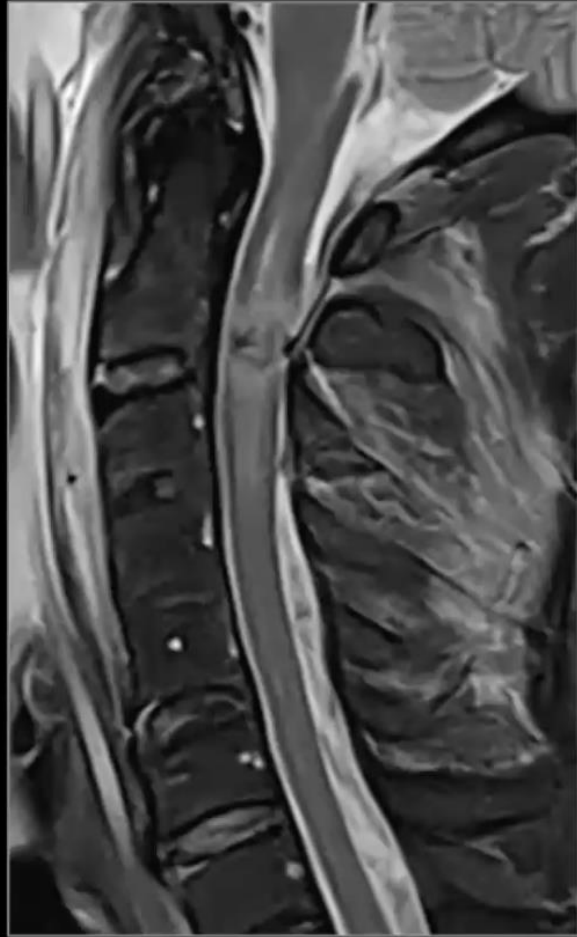
**CSF-flow related
artefacts**

Is there any spinal cord
compression ?

Compressive myelopathy



Spondylosis



Trauma

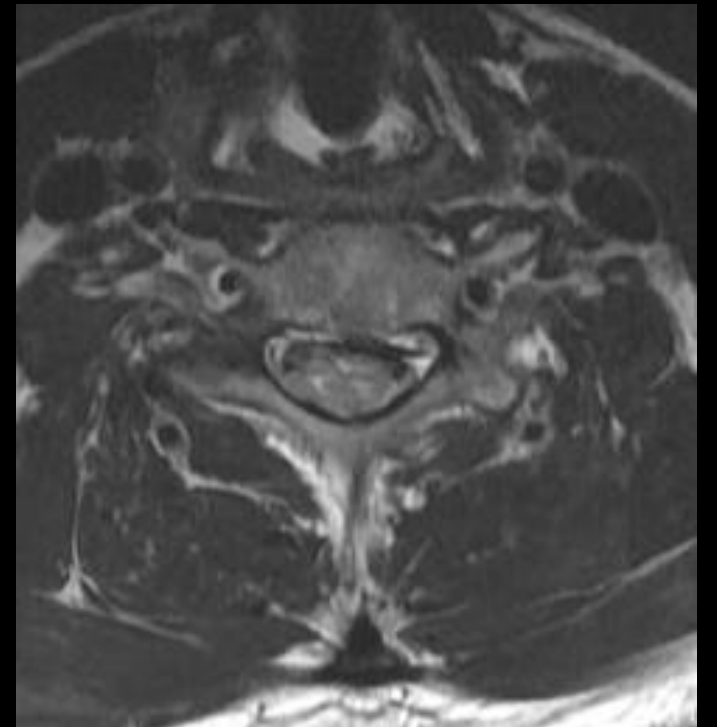
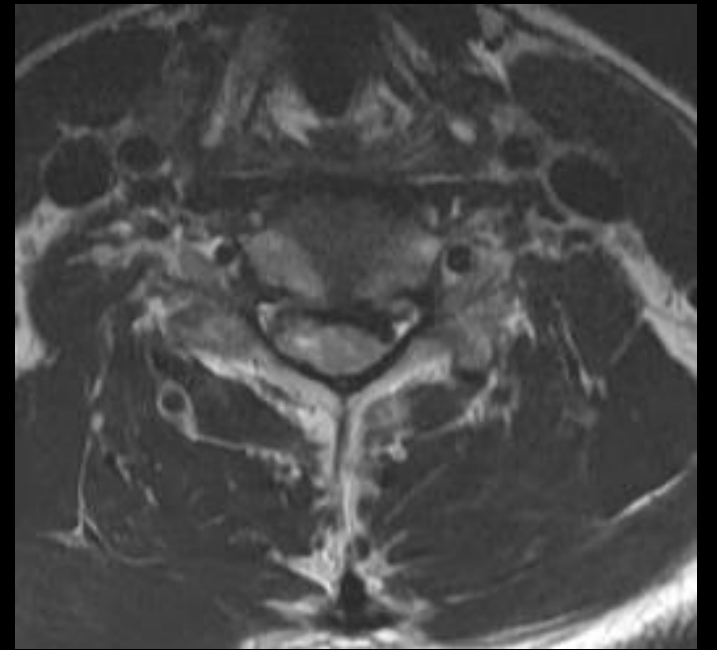


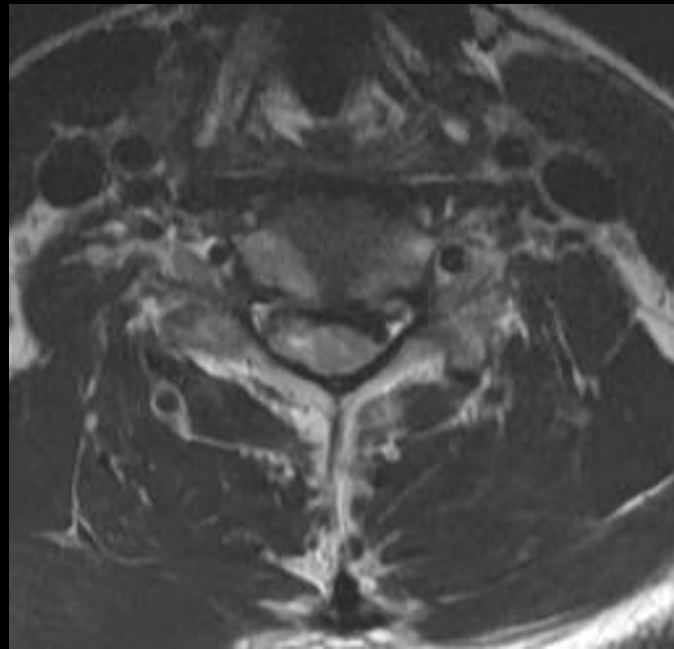
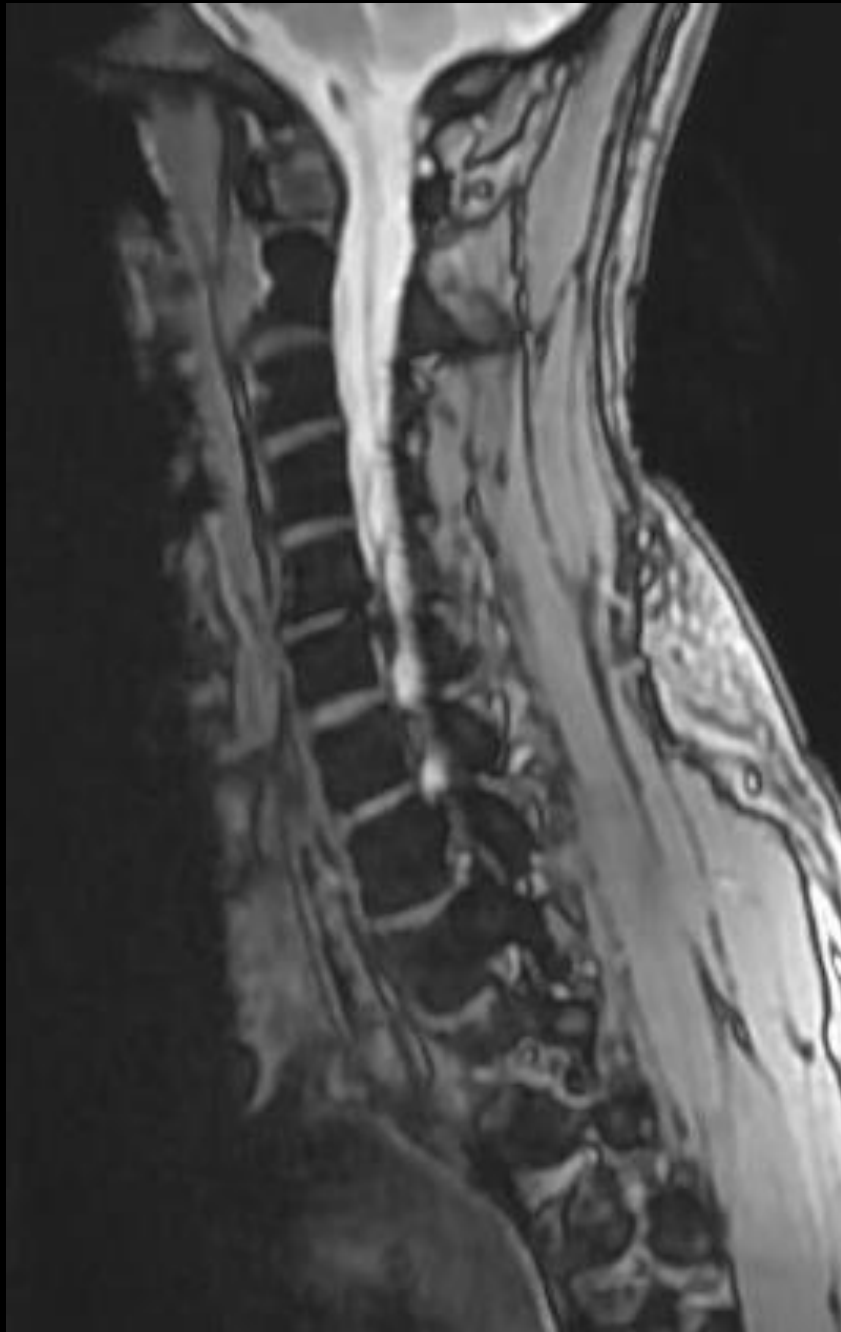
Tumor



Other...

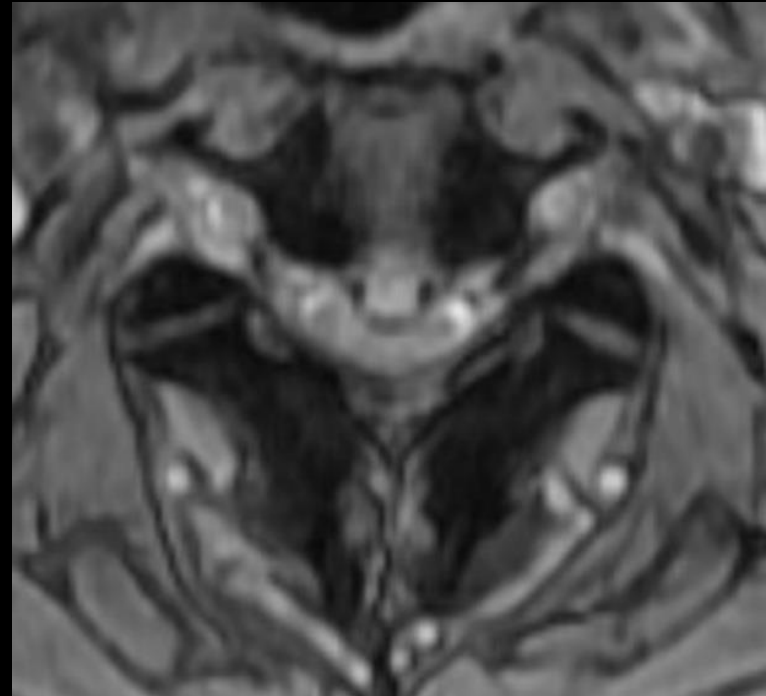
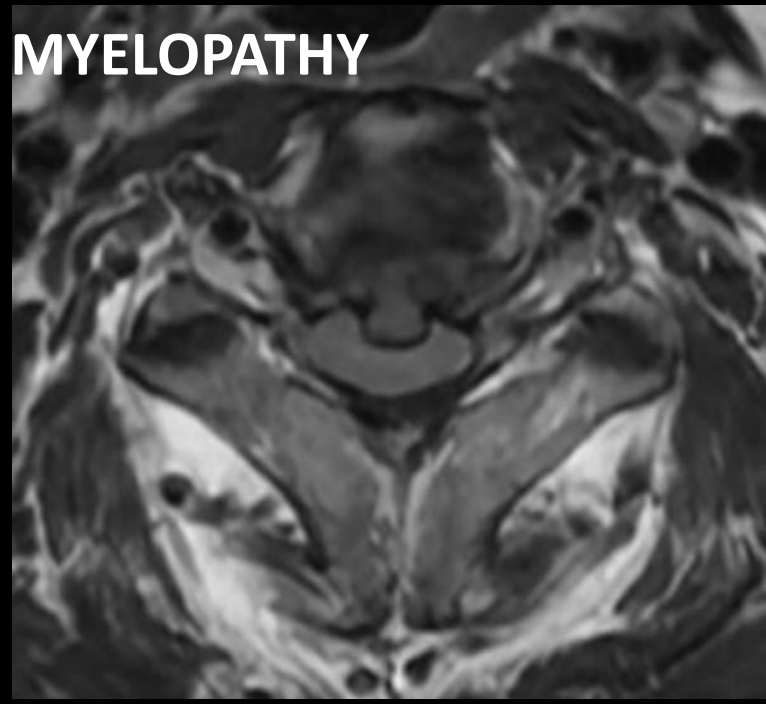
F, 50 y SPONDYLOSIS, WITH MYELOPATHY

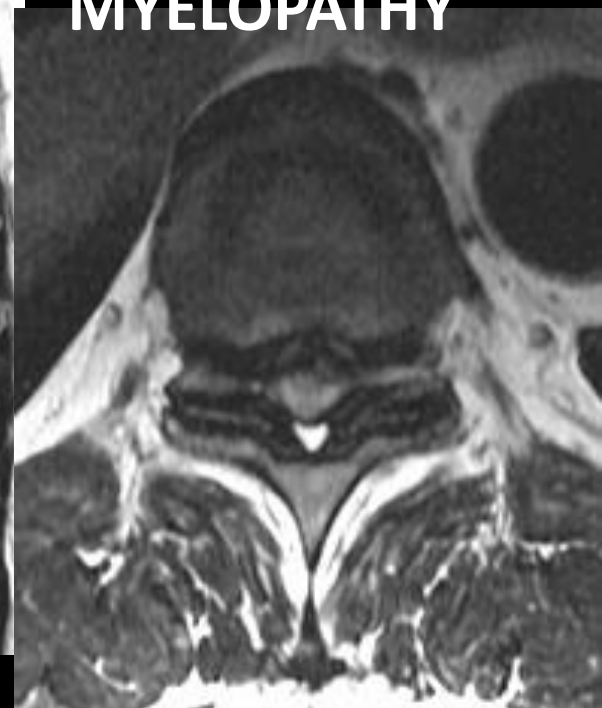




F, 50 y
OPLL WITH CORD
COMPRESSION &
MYELOPATHY

M, 54 y CENTRAL EXTRUSION DISC, with CORD COMPRESSION & MYELOPATHY

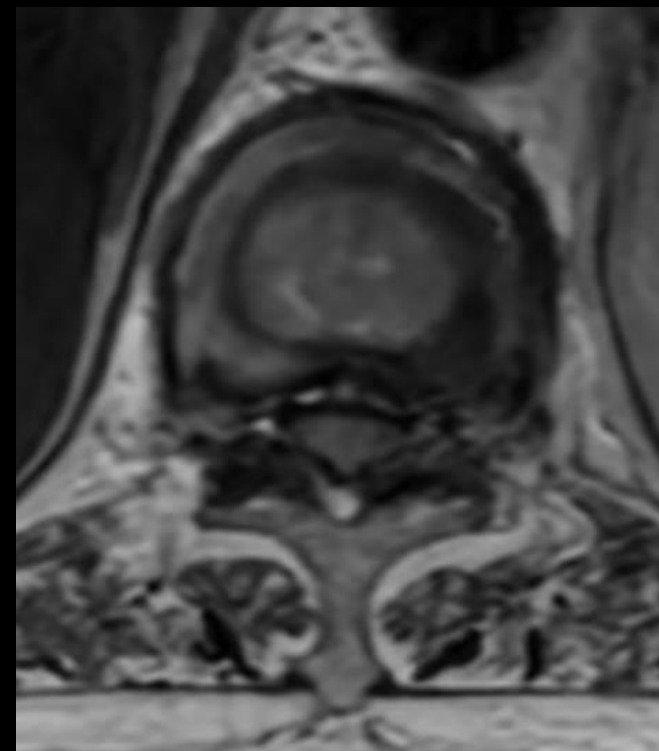




**M, 60 y
DISH + HNP +
OSSIFIED
FLAVUM
LIGAMENT, WITH
CORD
COMPRESSION &
MYELOPATHY**

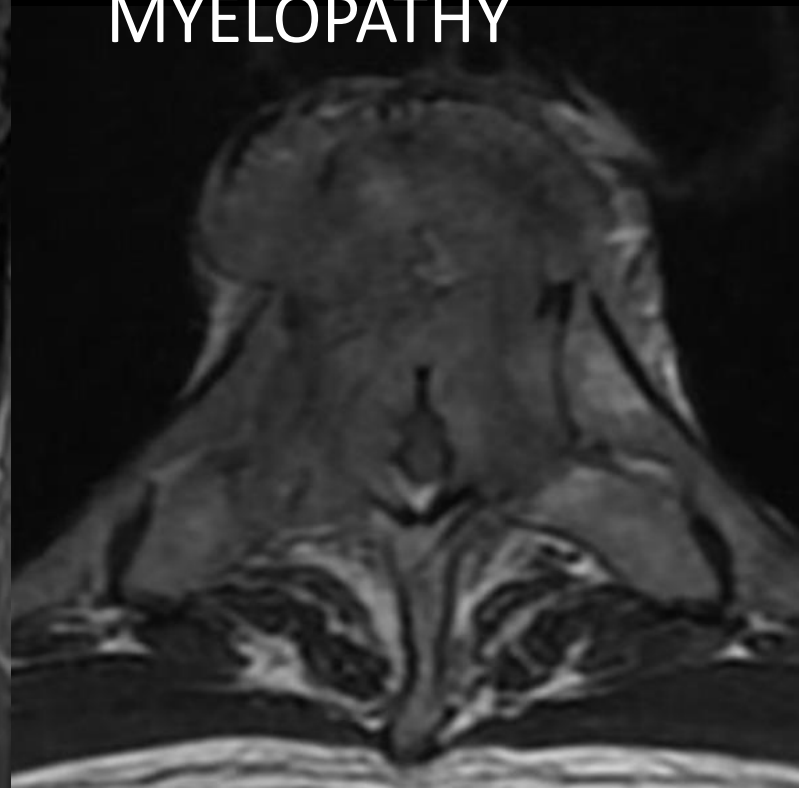


M, 40 y
RECENT BURST
FRACTURE WITH
CORD
COMPRESSION &
CORD EDEMA





F, 67 y
METASTATIC BONE
DISEASE with
SEVERE CORD
COMPRESSION &
MYELOPATHY



CASES

Acute Non Compressive Myelopathy



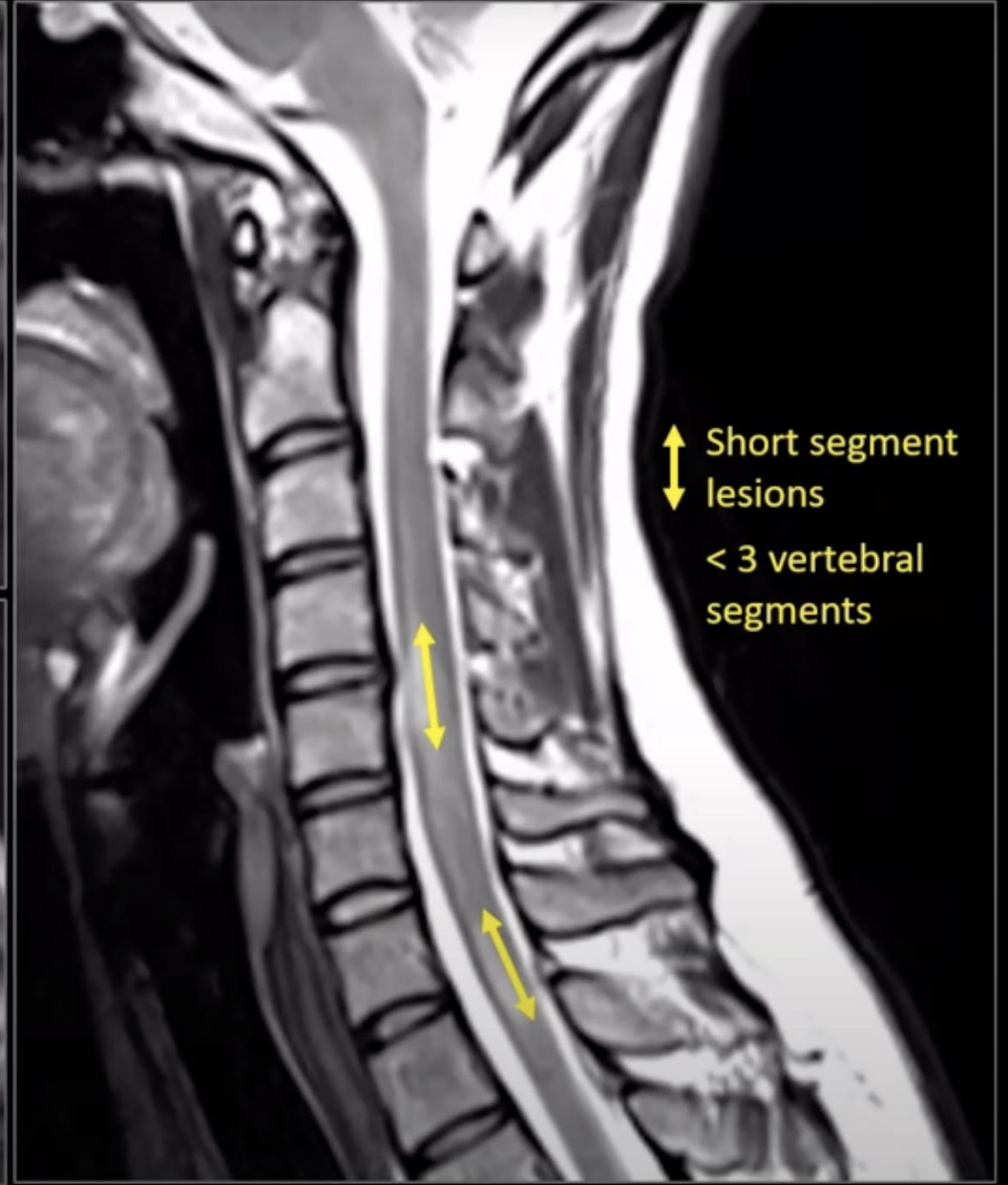
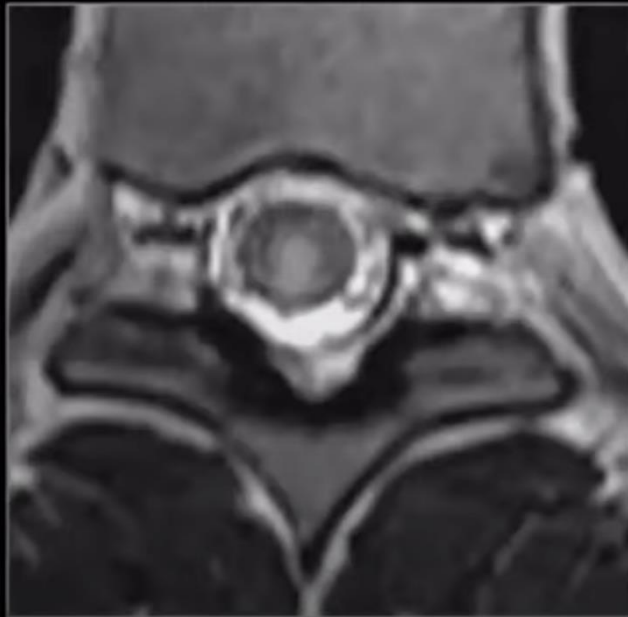
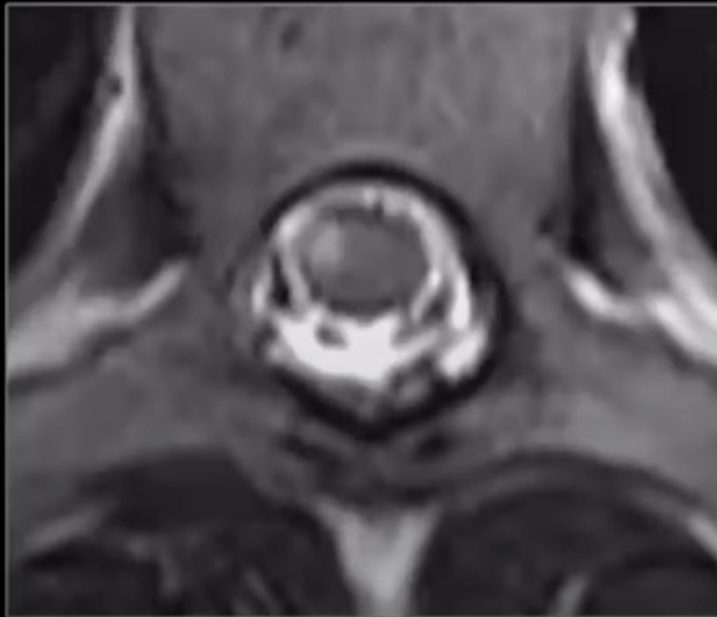
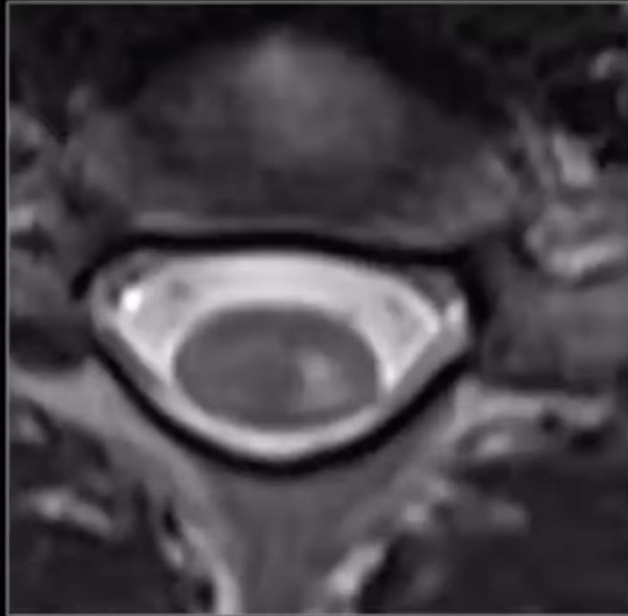
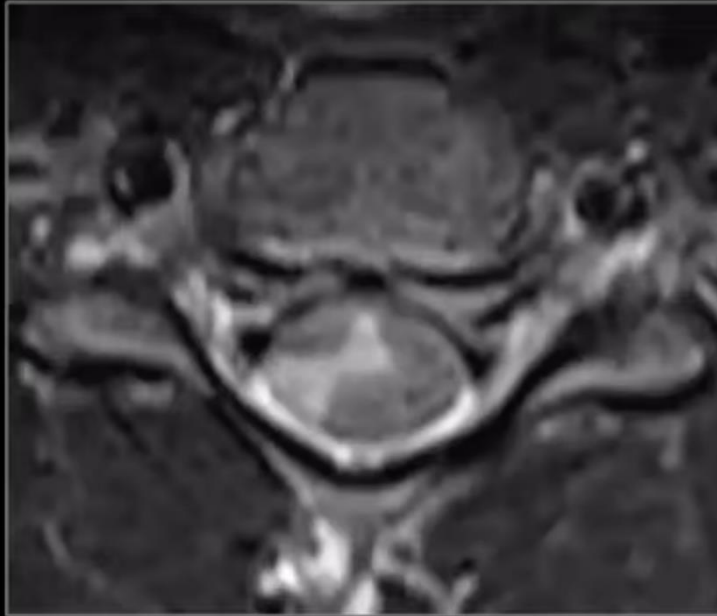
T2

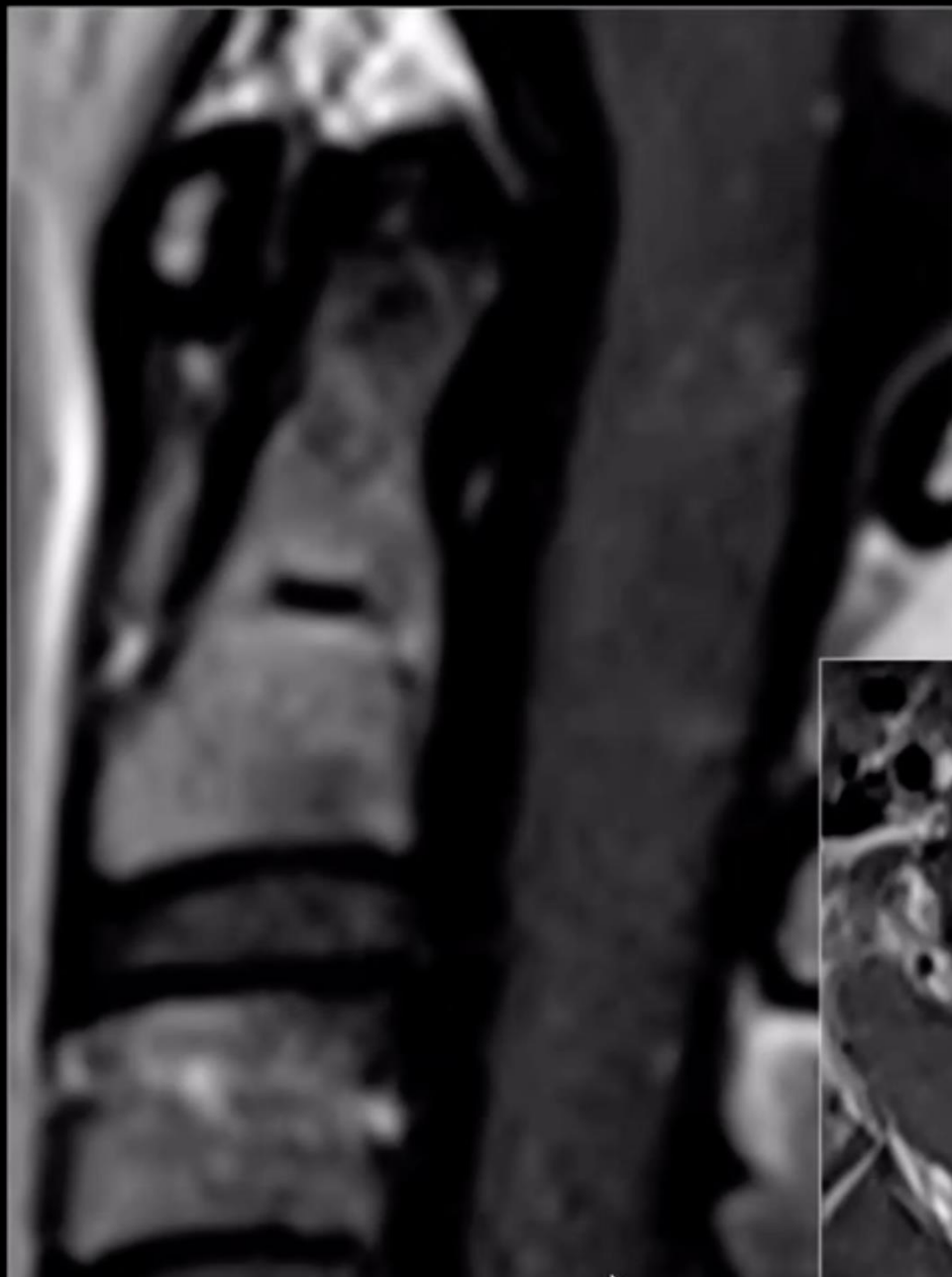


T2-STIR

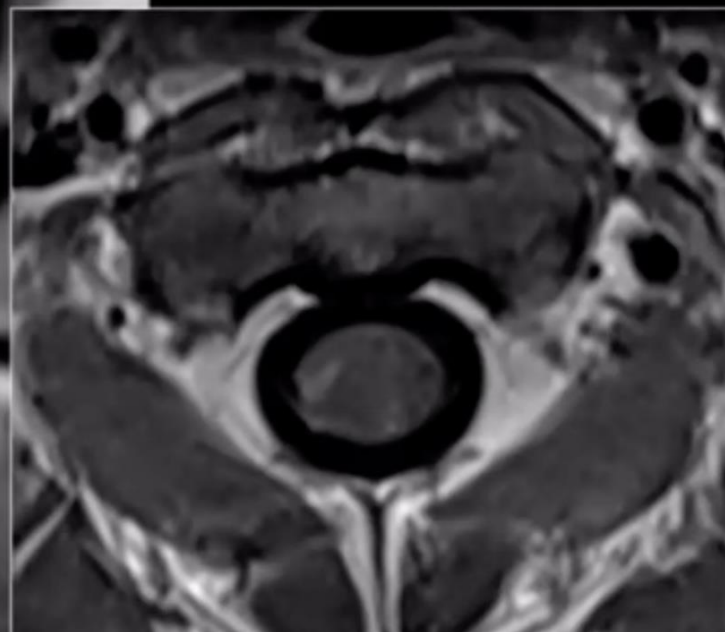


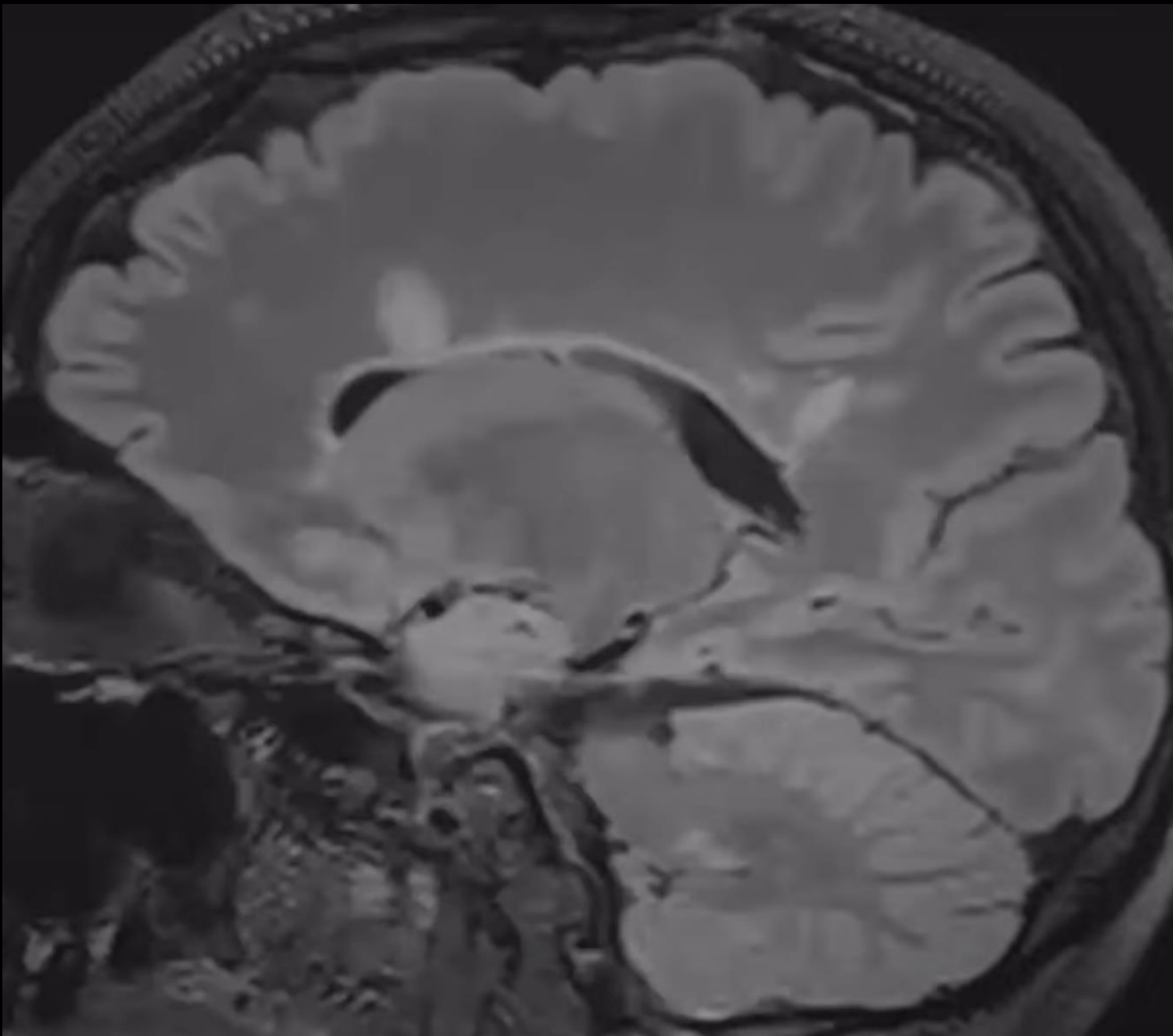
F, 30 years





**Open Ring
Enhancement**





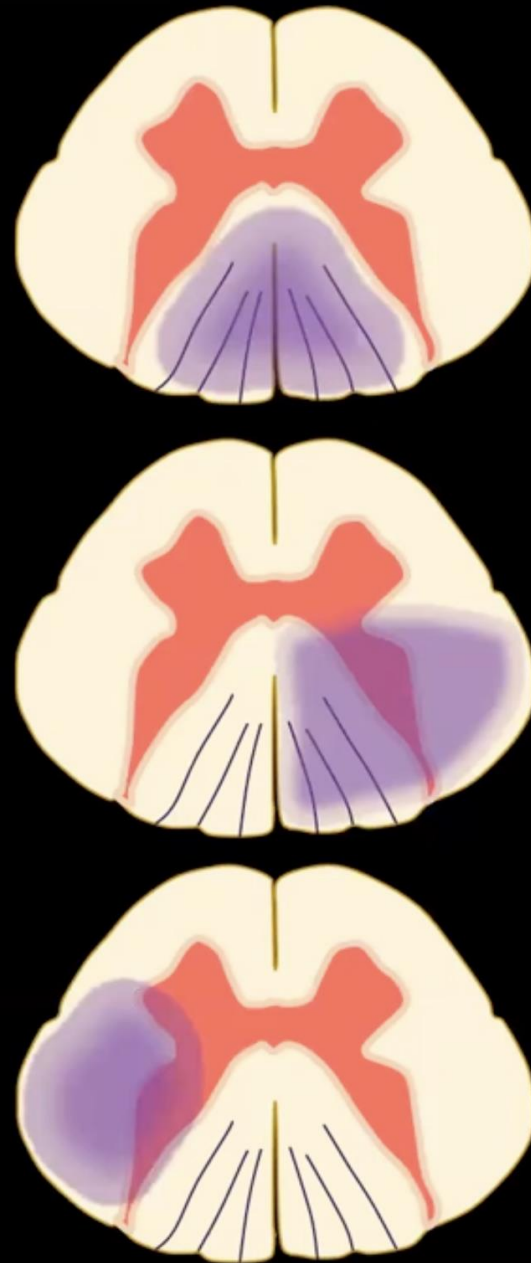
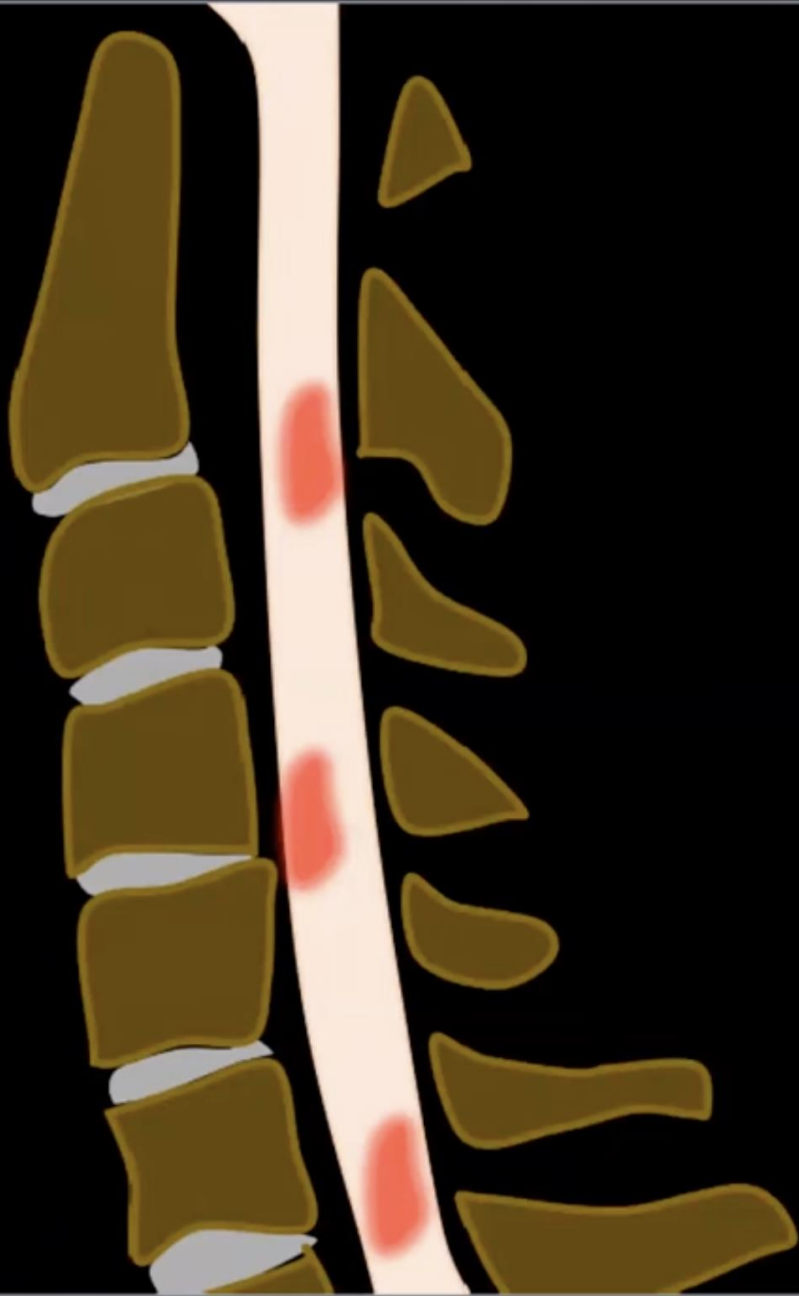
Multiple short
segment spinal
cord lesions --- do
Brain MRI



Ovoid lesions
at Dawson's
finger

MULTIPLE SCLEROSIS

- 80-90% spinal cord affected
- 20 % only spinal cord lesion.
- Short segment lesions.
- Peripheral/ eccentric (lateral/ dorsal), can be wedge shaped
- Usually partial, but can be complete myelitis.
- Acute : enhancement (usually open ring) & spinal cord swelling.
- Chronic : atrophy



Spinal cord lesions MS

- Short segment
- Peripheral
 - (lateral)
 - (dorsal)
- Often triangular
- GD(+): open ring

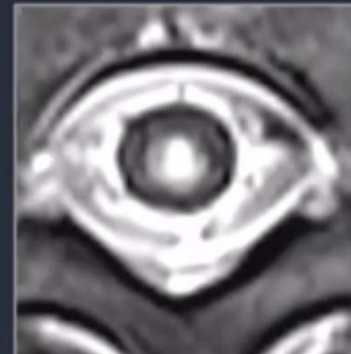
Neuromyelitis optica spectrum disorder (NMOSD)

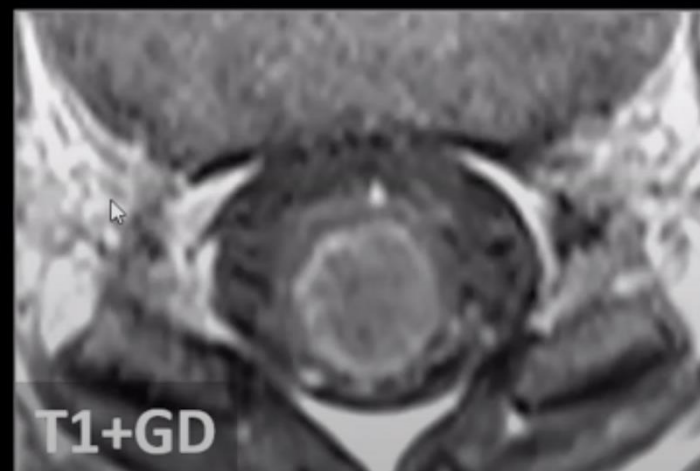
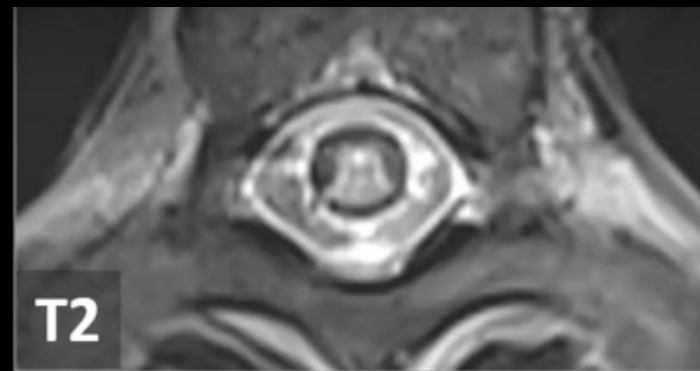
- Auto-immune inflammatory demyelinating CNS disorders
- Ab against AQP4-water channels
- **Optic nerve:** *Long segment, more posterior than in MS*
- **Myelum lesions:** *Long segment, central location*
- **Brain lesions:** *Periventricular (most AQP4 channels)*



Neuromyelitis optica spectrum disorder (NMOSD)

- **Long segment** (spanning ≥ 3 contiguous vertebral bodies)
- **Central cord** / gray matter predominance







T2: Bright spotty lesions

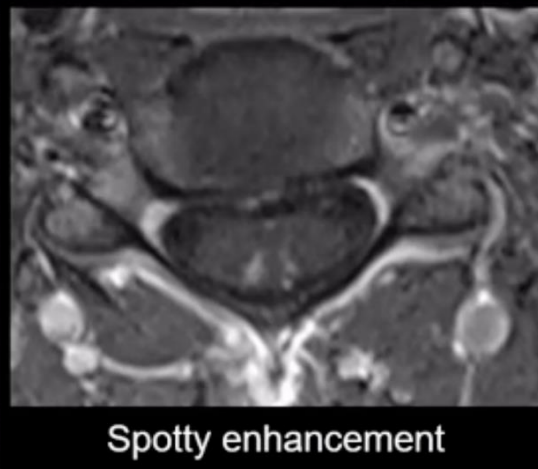
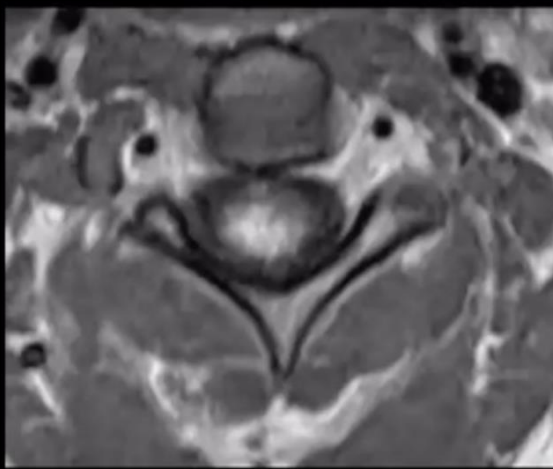
Patchy cloud-like enhancement



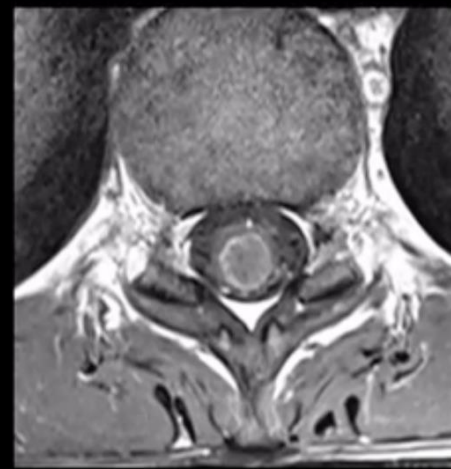
Ring- or lens-shaped enhancement

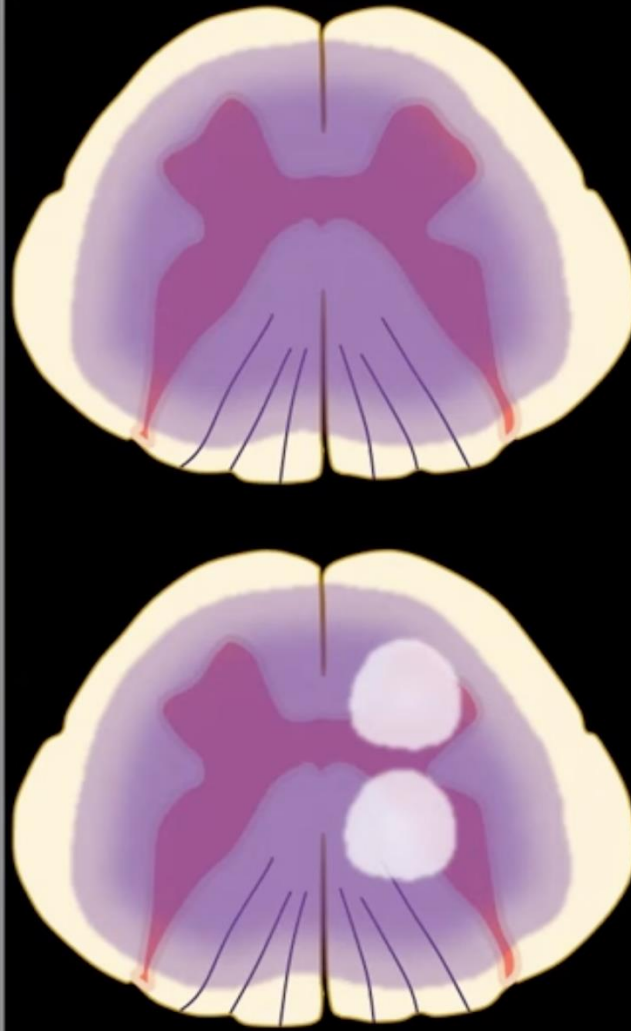
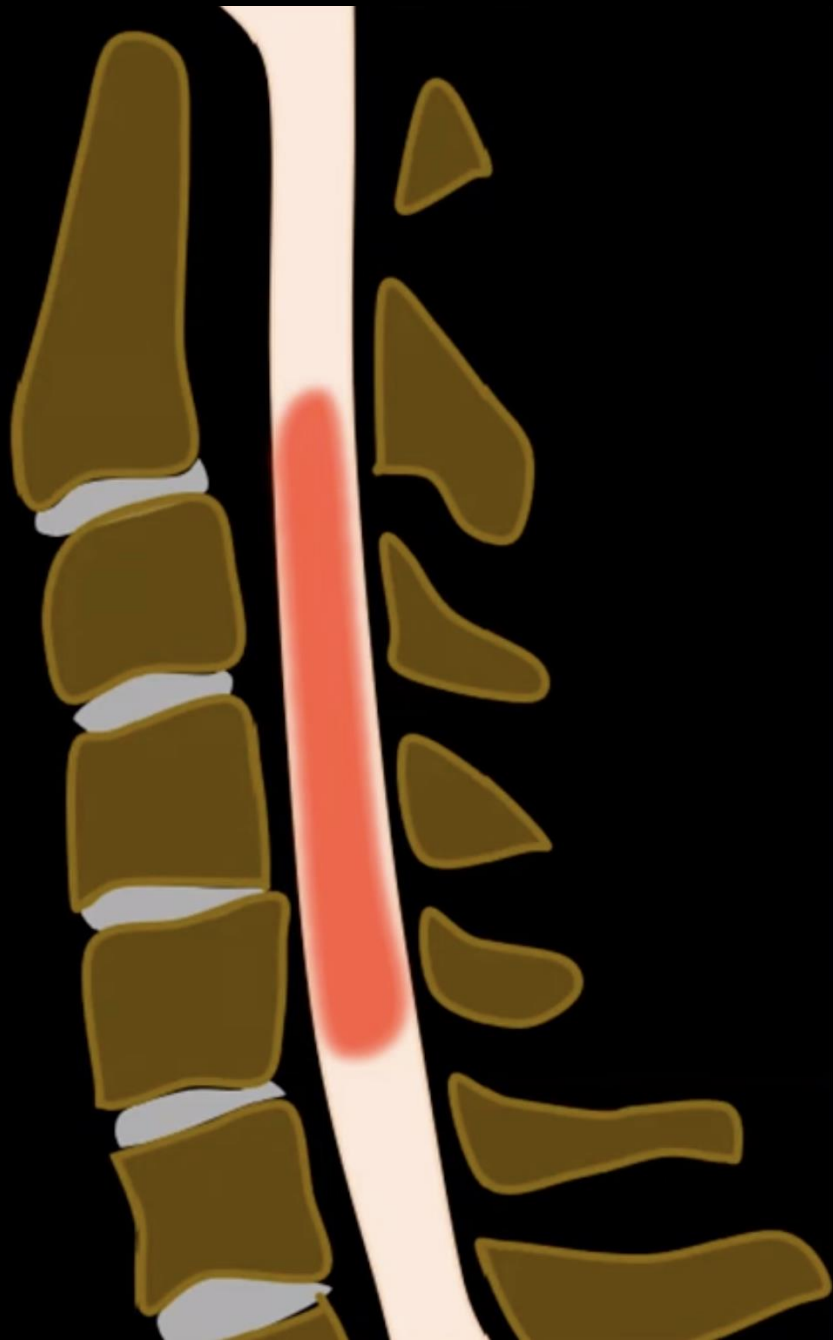


GD: Variable enhancement patterns



Spotty enhancement



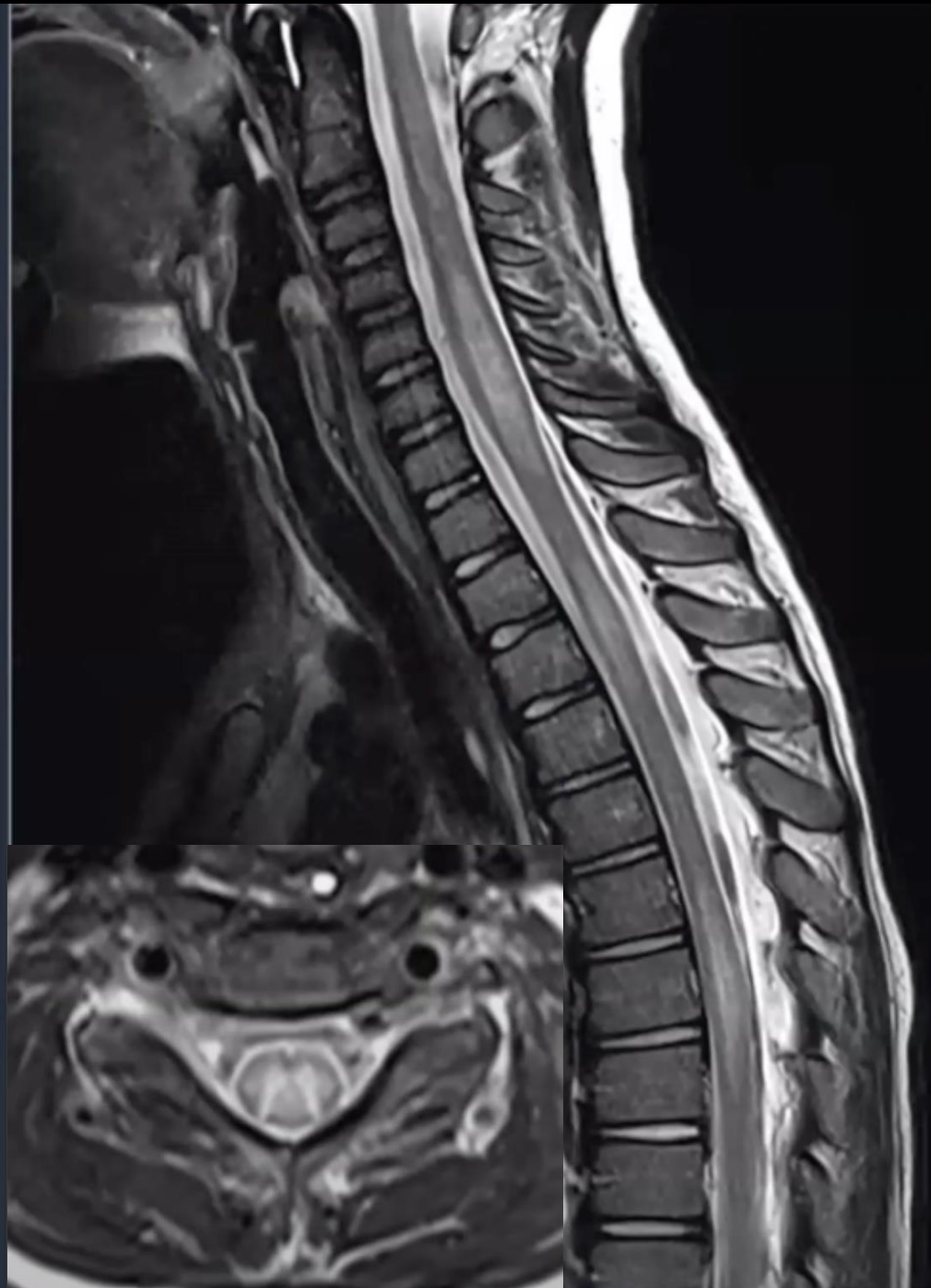
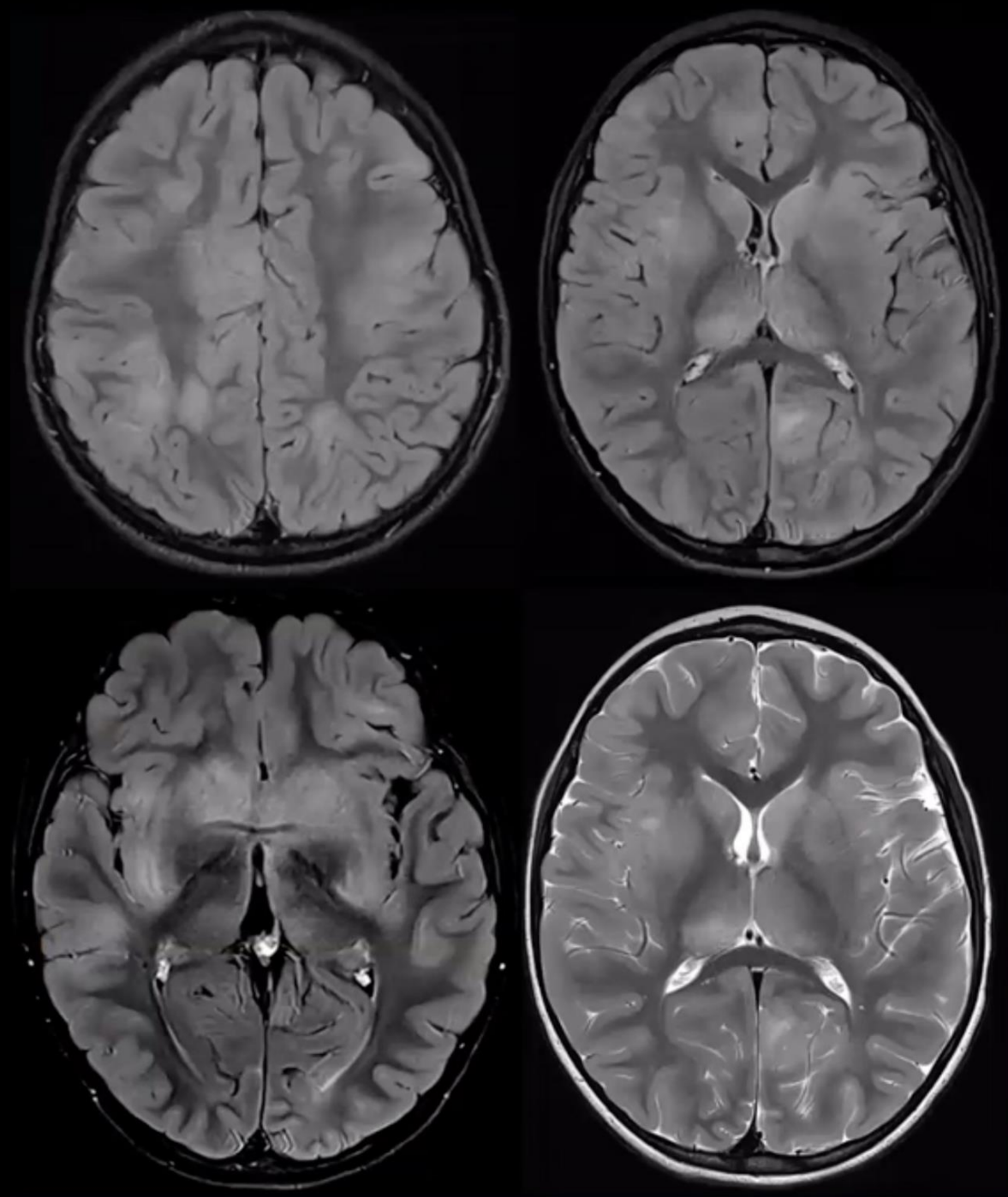


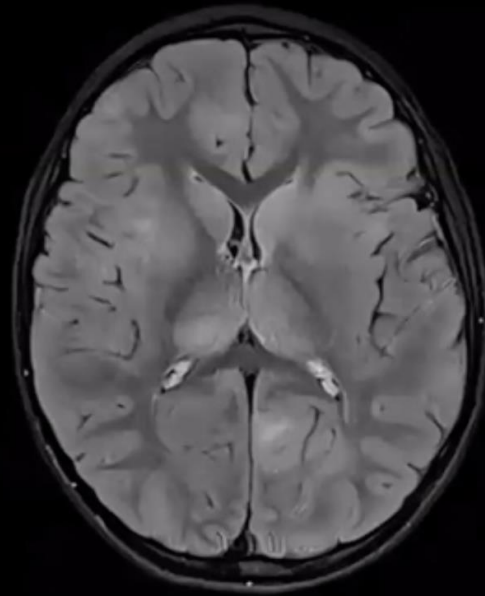
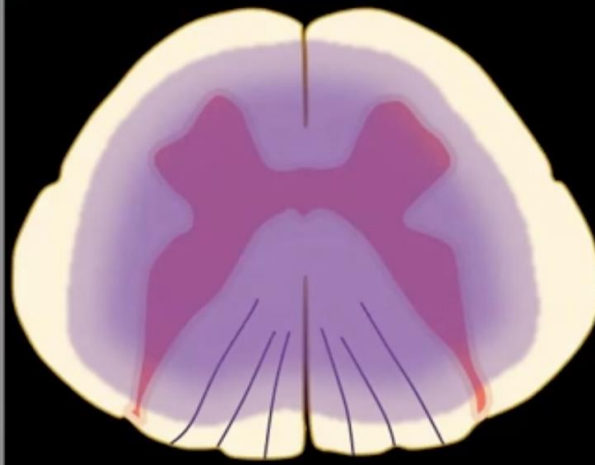
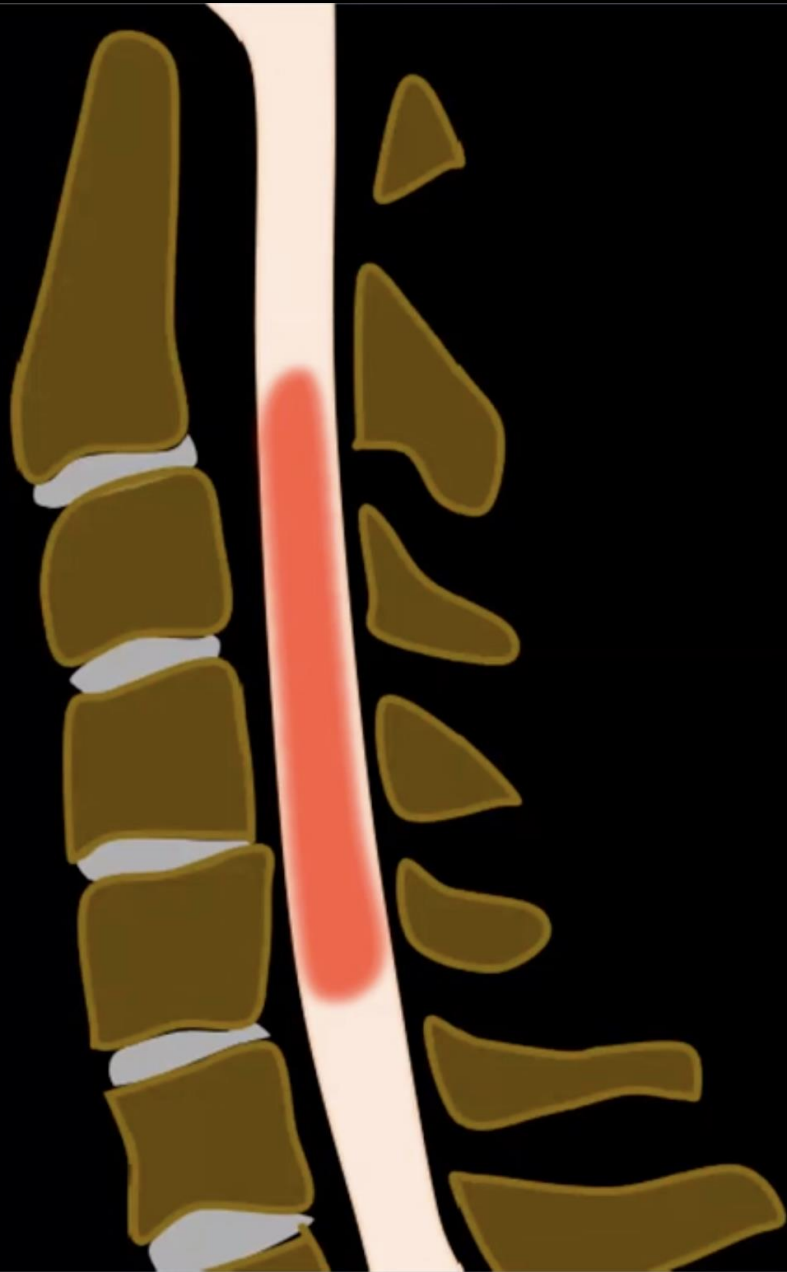
NMOSD

- Longitudinally extensive
- Transverse involvement
- Cervical > thoracic SC
- T2 bright spotty lesions
- GD(+): diverse

ADEM / ACUTE DISSEMINATED ENCEPALOMYELITIS

- Acute, monophasic demyelinating disease of the brain & the spinal cord.
- Typically following a prior (1-2 weeks) viral infection/ vaccination.
- Primary manifestation : encephalopathy.
- Spinal involvement : 25%
- Any age, majority in child





ADEM

- Longitudinally extensive
- Transverse involvement
- No defining characteristics to differentiate from other LETMs
- GD(+): diverse
- Brain MRI: +++

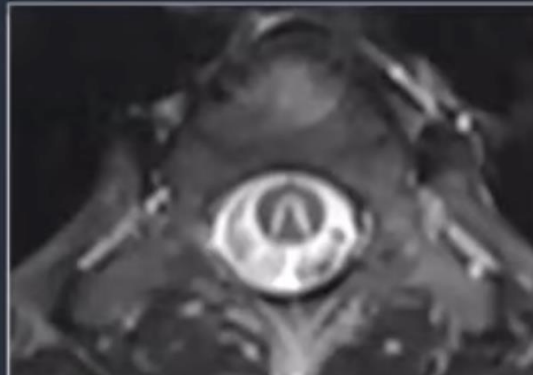
MOGAD

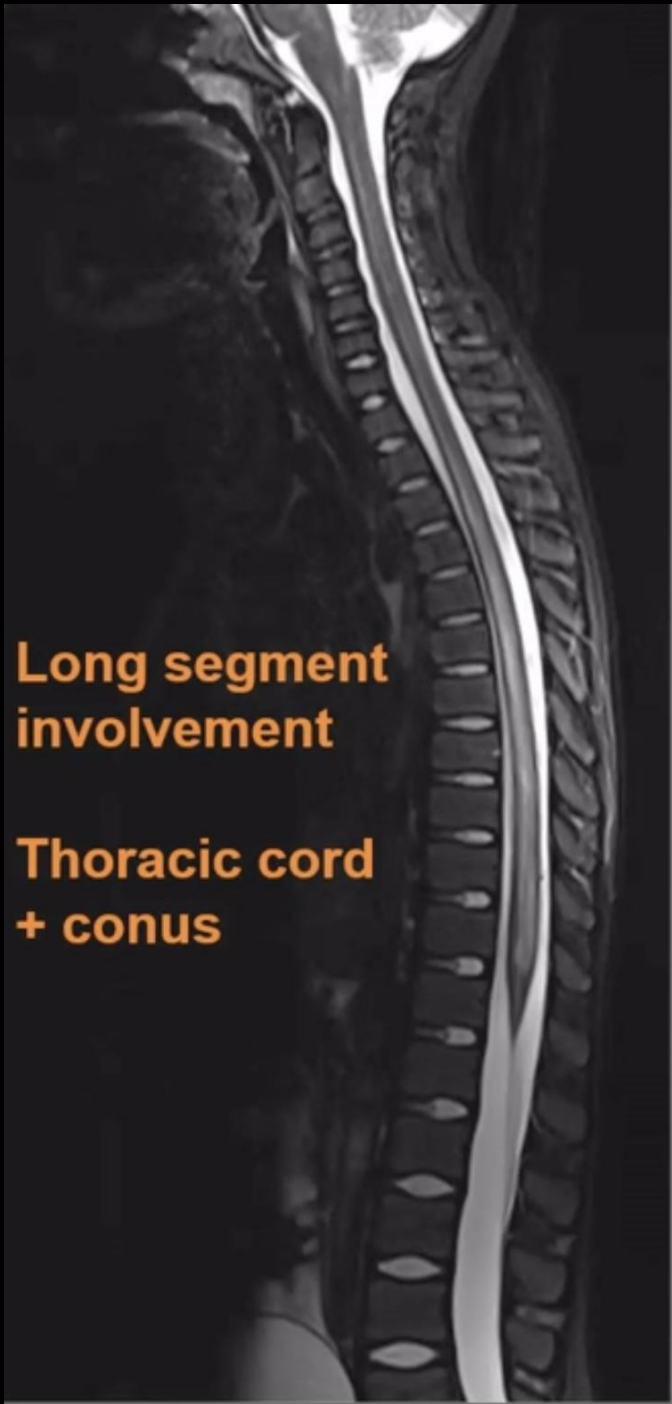
- **M**yelin **O**ligendrocyte **G**lycoprotein
Antibody-associated **D**isease
- Group of inflammatory demyelinating disorders characterized by IgG antibodies to MOG

MOG-antibody associated disease (MOGAD)

- **Long segment** (spanning ≥ 3 contiguous vertebral bodies)
- **Central cord** / gray matter predominance

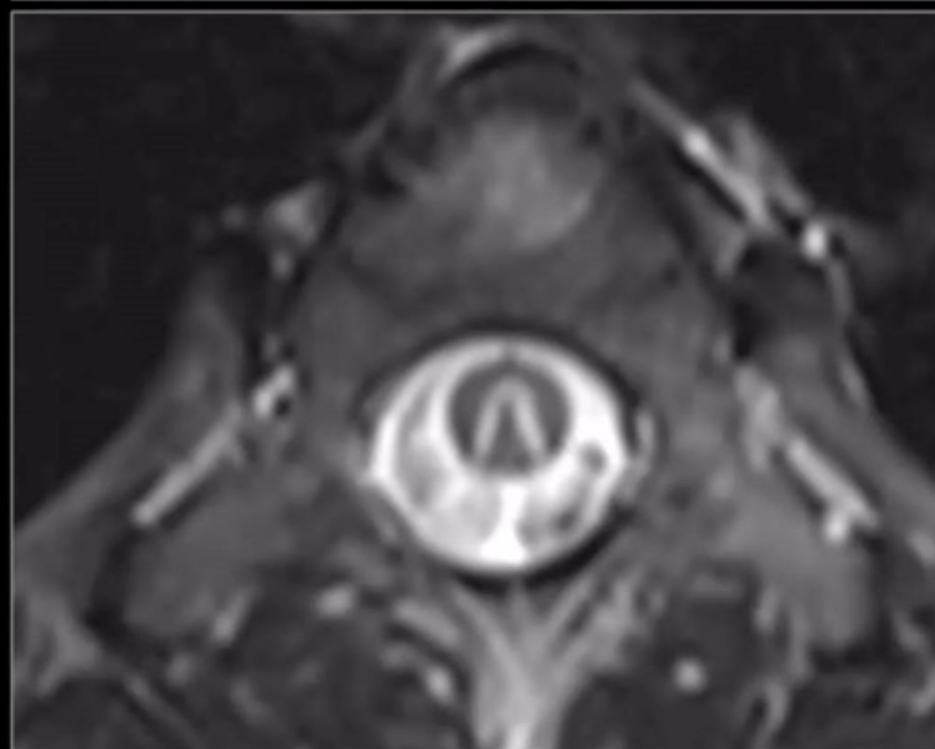
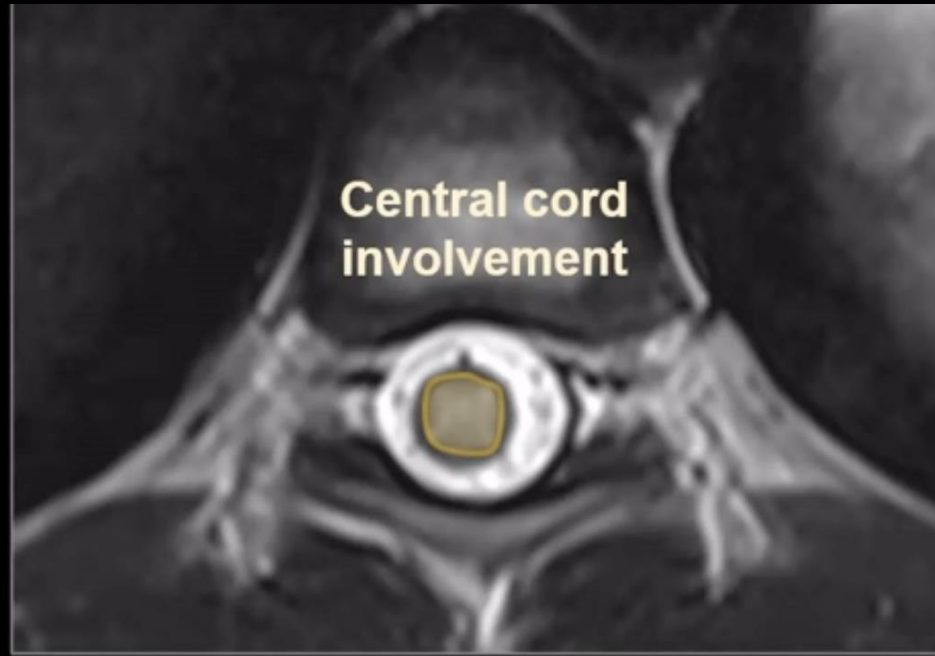
H-sign





**Long segment
involvement**

**Thoracic cord
+ conus**





MOGAD

- Longitudinally extensive
- Transverse involvement
- Thoracic SC > cervical SC^{*}
- Conus
- H-sign



VZV myelitis

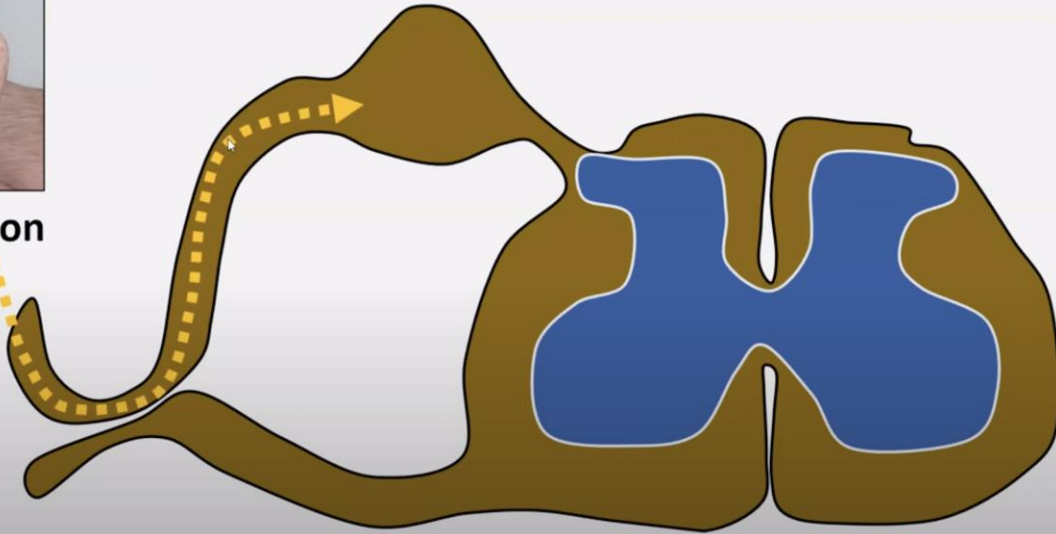
(Para-)infectious myelitis

- Direct infection of the spinal cord is uncommon
- Bacterial, viral, fungal, parasitic → mostly viral
- Herpesviruses (HSV, VZV) → remain dormant after primo-infection, can reactivate and extend into the spinal cord causing myelitis
- Post- or para-infectious → auto-immune mediated following a (viral) infection elsewhere in the body (*~ADEM, but no brain lesions*)

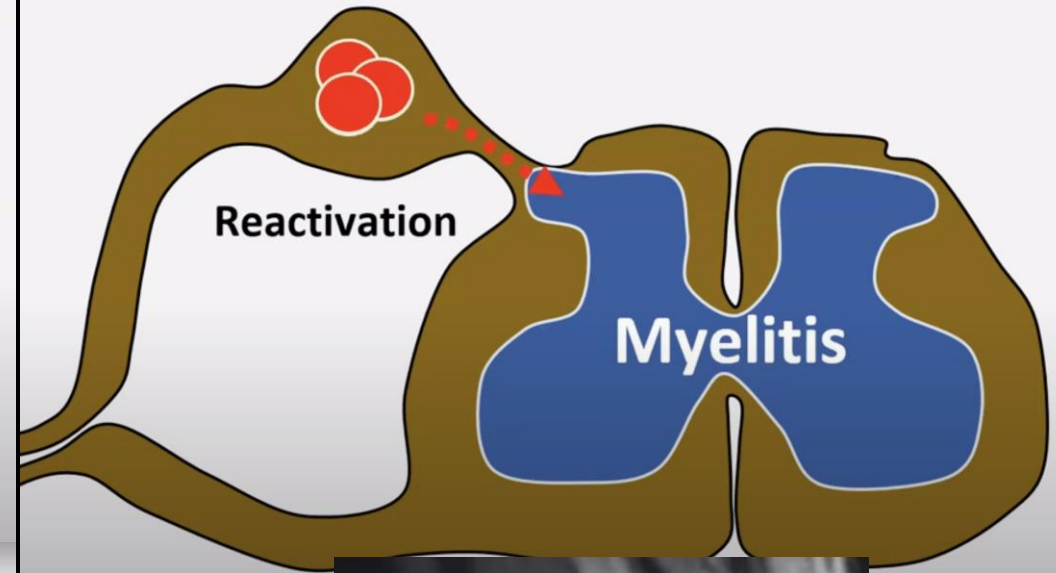


Chickenpox is generally a benign, self-limiting disease in children

Primo infection

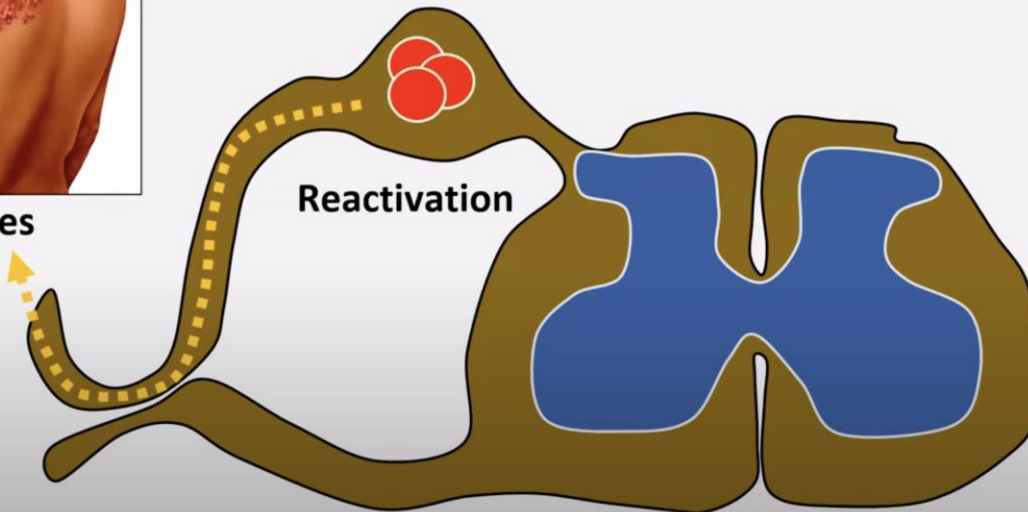


In cases of decreased immunity the virus reactivates → myelitis!

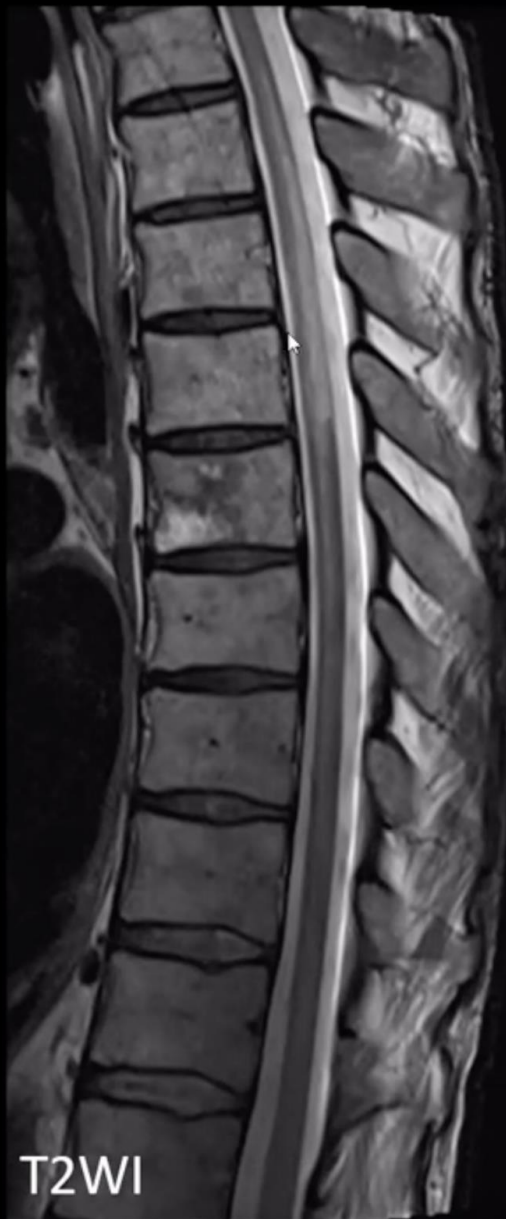


In cases of decreased immunity the virus reactivates → shingles!

Shingles



Spinal Cord Ischemic Infarction



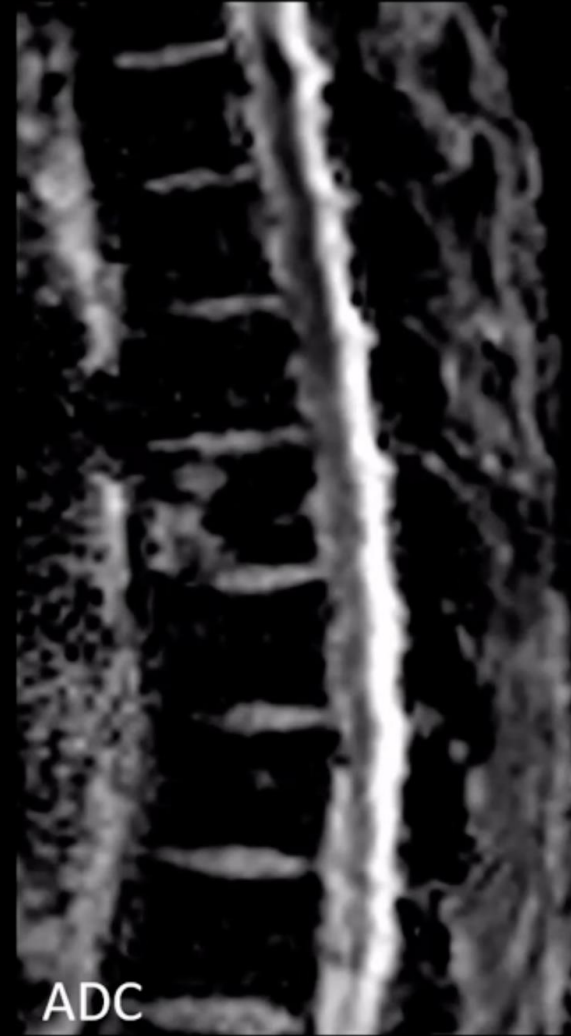
T2WI



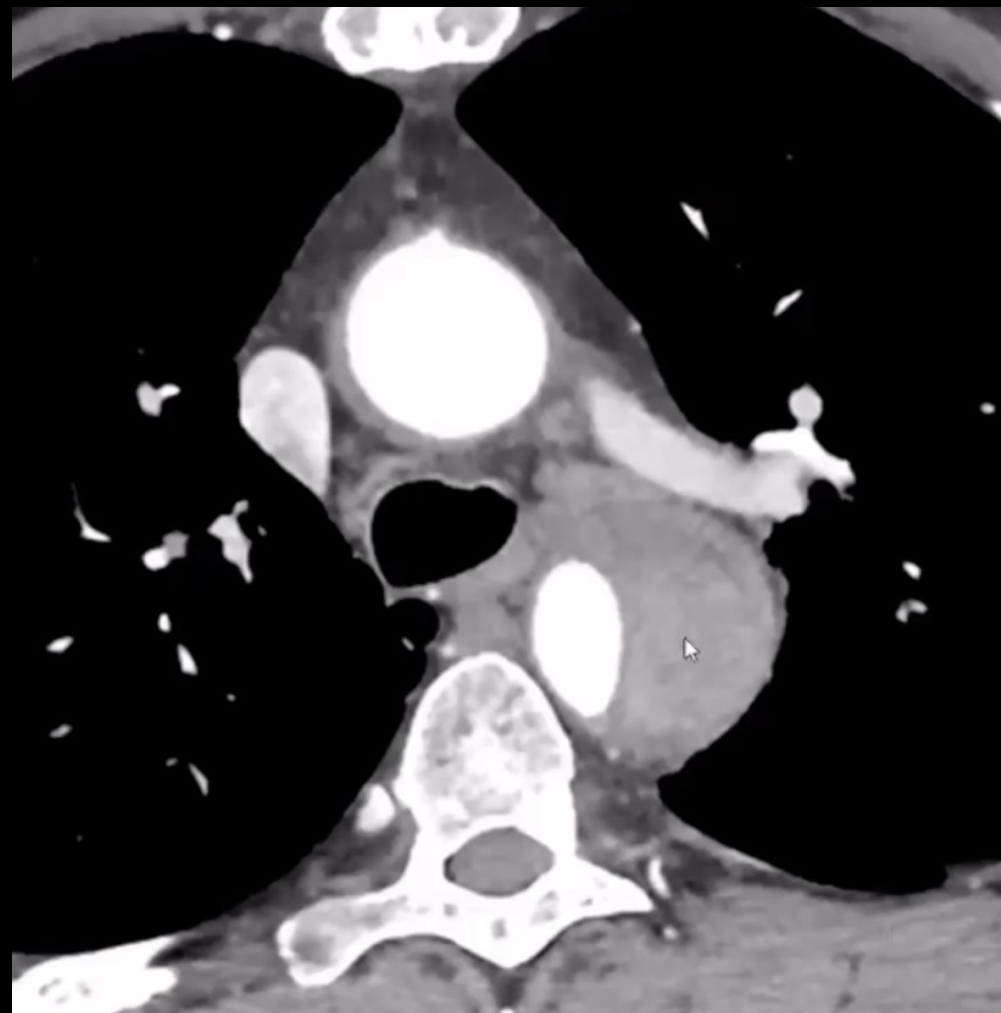
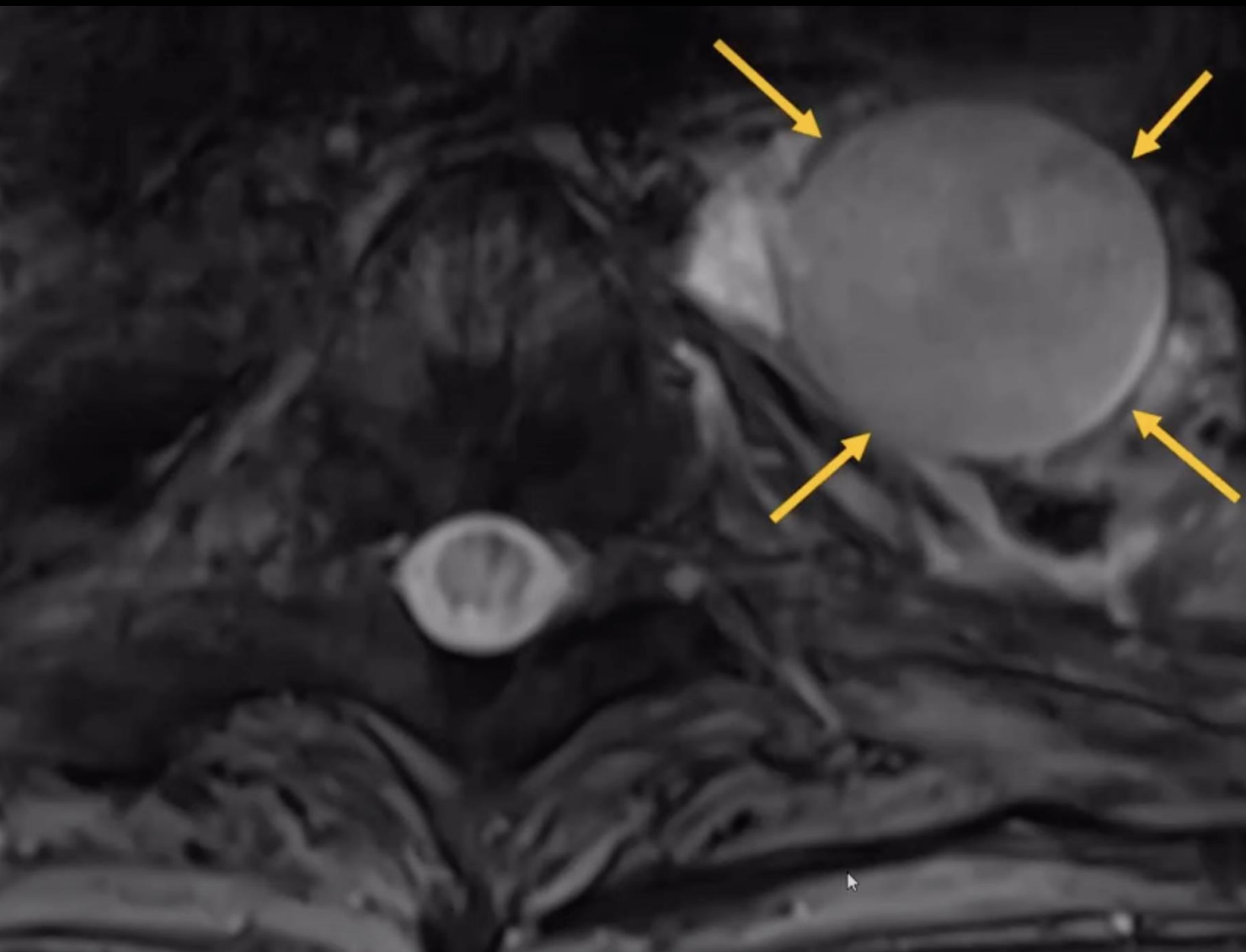
T2-STIR

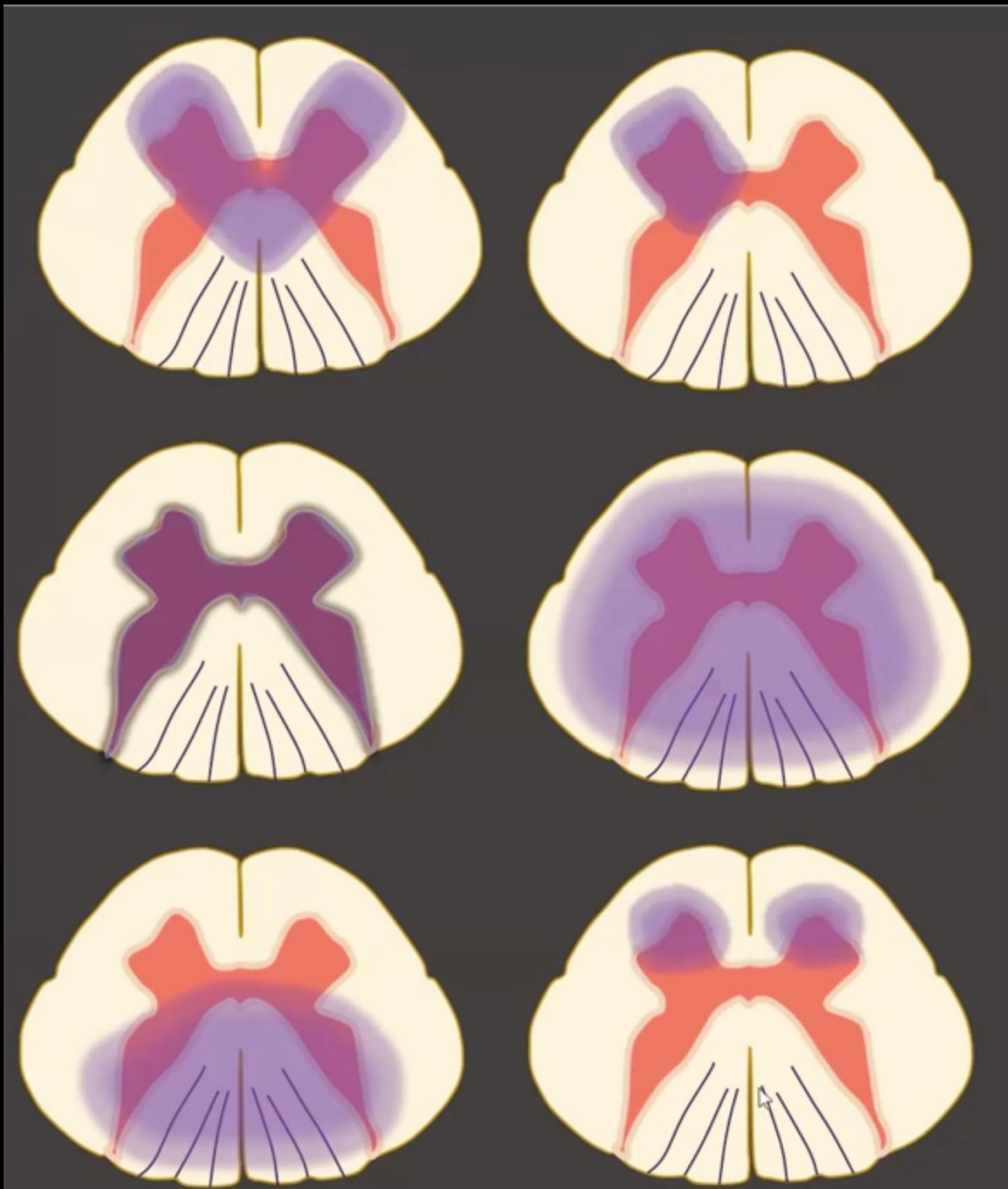


B1000 DWI



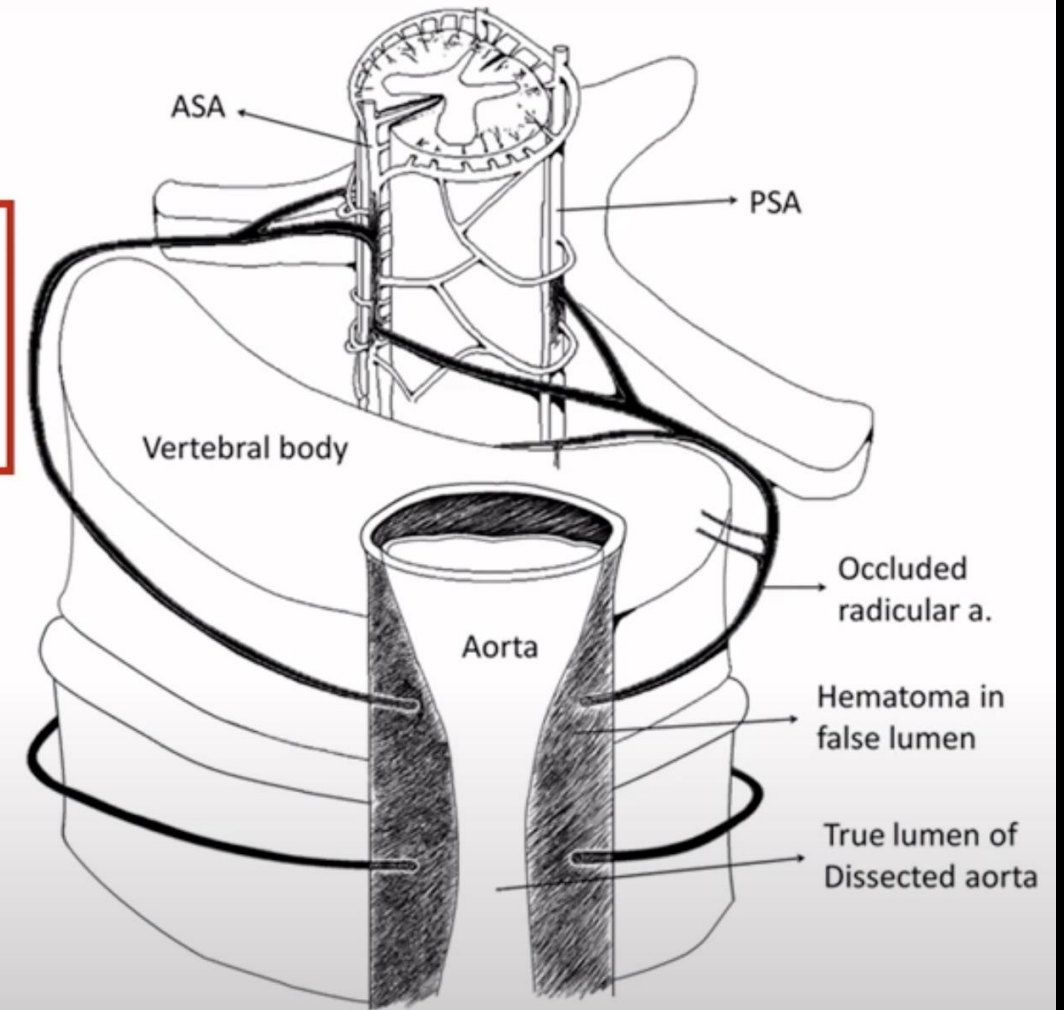
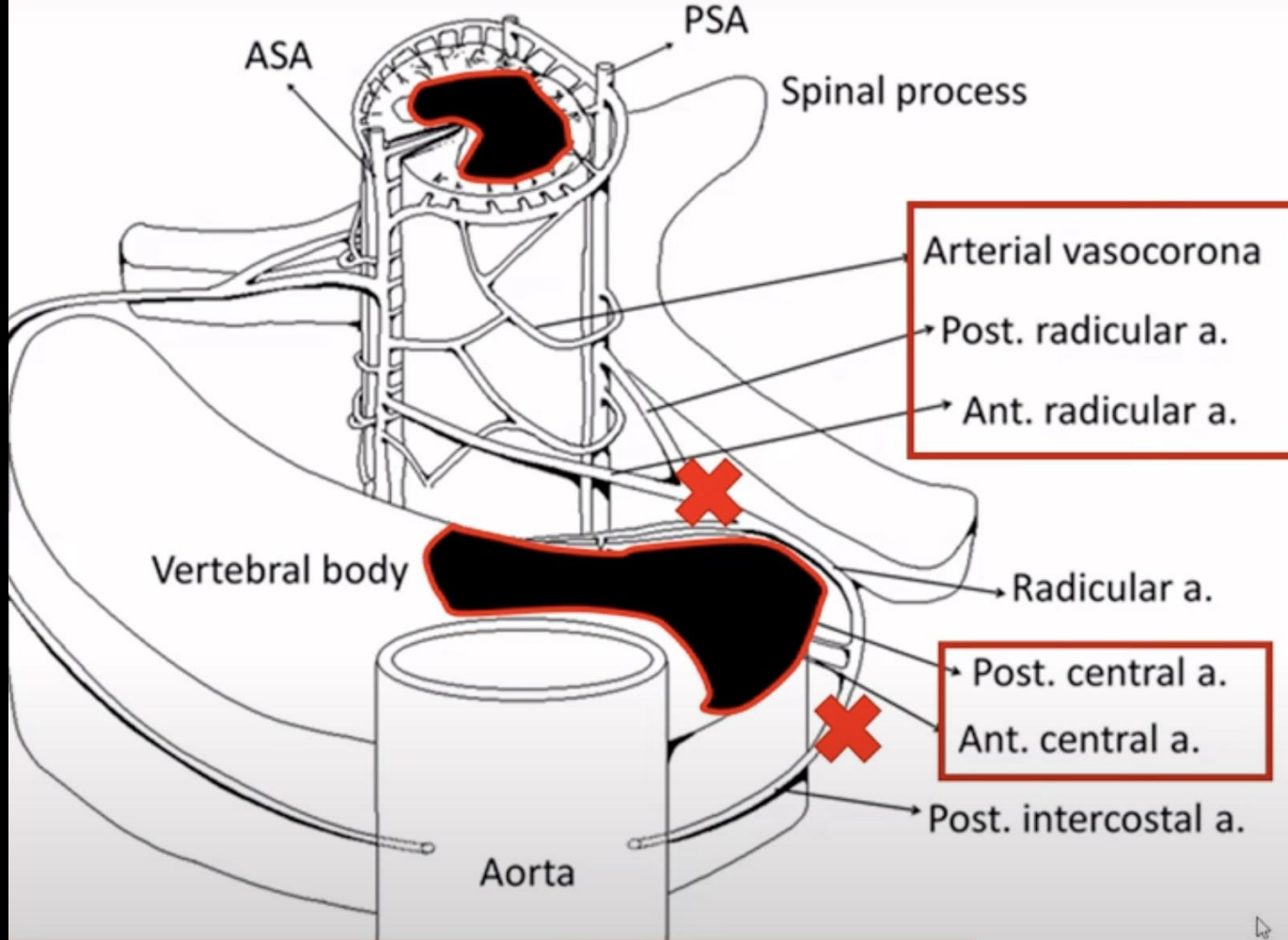
ADC





Spinal cord ischemic infarction

- **Rare: < 1% of all strokes**
 - Complex vascular supply
 - Numerous anastomoses & connections
- **Very heterogeneous etiology**
 - In the elderly: aortic disease
 - In younger patients: VA dissection
 - But also: drugs, cardiac embolism, hypotension...
- **Most common in cervical & thoracic cord**
 - Cervical spinal cord (~40%)
 - Thoracic spinal cord (~40%)
- **Clinical:** rapid progression to nadir < 4h



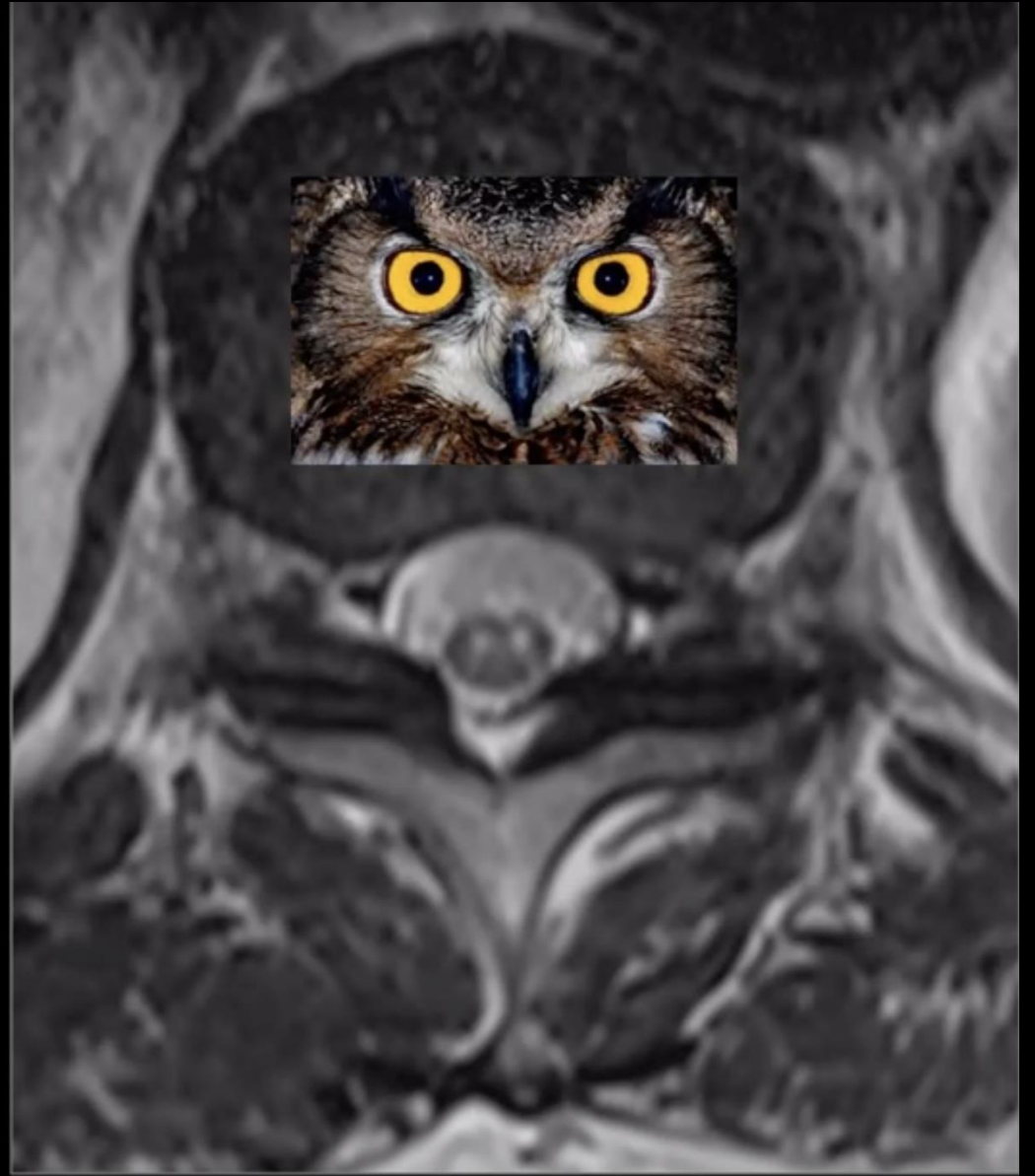
Vertebral body infarctions + spinal cord infarctions → proximal problem, more frequent in aortic disease



Diffusely swollen
T2-hyperintense
spinal cord

Foci of bone
marrow edema
posteriorly in
the thoracic
vertebra

Vertebral bony infarction
is a confirmatory sign of
suspected spinal ischemic
infarction



TAKE HOME POINTS

- Make sure that intramedullar T2-hyperintensity is real.
- Compressive vs Non Compressive ?
- Acute vs subacute-chronic onset ?
- Acute non compressive myelopathy :
 - vascular : spinal cord infarction (hyperacute-within 4 hours)
 - Inflammatory : partial/ transverse myelitis (4 hours-21 days) -- demyelination (MS, NMOSD, ADEM, MOGAD); para-infectious (direct infection, post infectious); systemic inflammatory (Lupus, Sjorgen, Bechet, Sarcoid).

THANK YOU