Educational session 20 – MAGNIMS teaching course

MRI and MS misdiagnosis & differential diagnosis

Frederik Barkhof

UCL

Magnims
Magnetic Resonance Imaging in Multiple Sclerosis

VU university medical center

Disclosures

- Steering Committee – Bayer, Merck, Biogen (aducanumab), TEVA
- Consultant – Novartis, Merck, Roche, Jansen, IXICO
- DSMB member – Roche-Genentech (crenezumab)
- Research agreements – Philips, TEVA, GE, Novartis
- Sponsor – NIHR-UCLH-BRC, Dutch Foundation MS Research, TEVA, Novartis, EU-H2020, EU-JU (IMI), NMSS
- Editorial board member – Brain, Neuroradiology, MSJ, Neurology, Radiology

Agenda

- Reasons for misdiagnosis of MS
  - a priori chance of MS and OND
  - spectrum bias: representative populations?
- McDonald 2017 - focus on specificity
  - exclude vascular disease (very common)
  - identify other inflammatory disease (rare)
- Red flags on imaging
  - imaging MIMICS criteria

Reasons for MS misdiagnosis

Table 3 Contributors to MS misdiagnosis

	Yes	No	Unknown
	n (%)	n (%)	n (%)
Inappropriate application to MS diagnostic criteria of neurologic symptoms atypical for a demyelinating attack	72 (65)	24 (22)	14 (13)
Inappropriate application to diagnostic criteria of a historical episode of neurologic dysfunction without corroborating objective evidence of a lesion (on neurologic examination, evoked potentials, or imaging)	53 (48)	38 (35)	19 (17)
Overreliance on the presence of MRI abnormalities meeting DIS to confirm a diagnosis of MS in a patient with "nonspecific neurologic symptoms"	66 (60)	28 (25)	16 (15)
Erroneous determination of juxtacortical or periventricular lesion location to fulfill DIS	36 (33)	43 (39)	31 (28)
Erroneous determination of DIT because of variability of MRI slice orientation (i.e., MRIs performed on different scanners leading to the appearance of new lesions)	13 (12)	64 (58)	33 (30)

Solomon AJ. Neurology 2016

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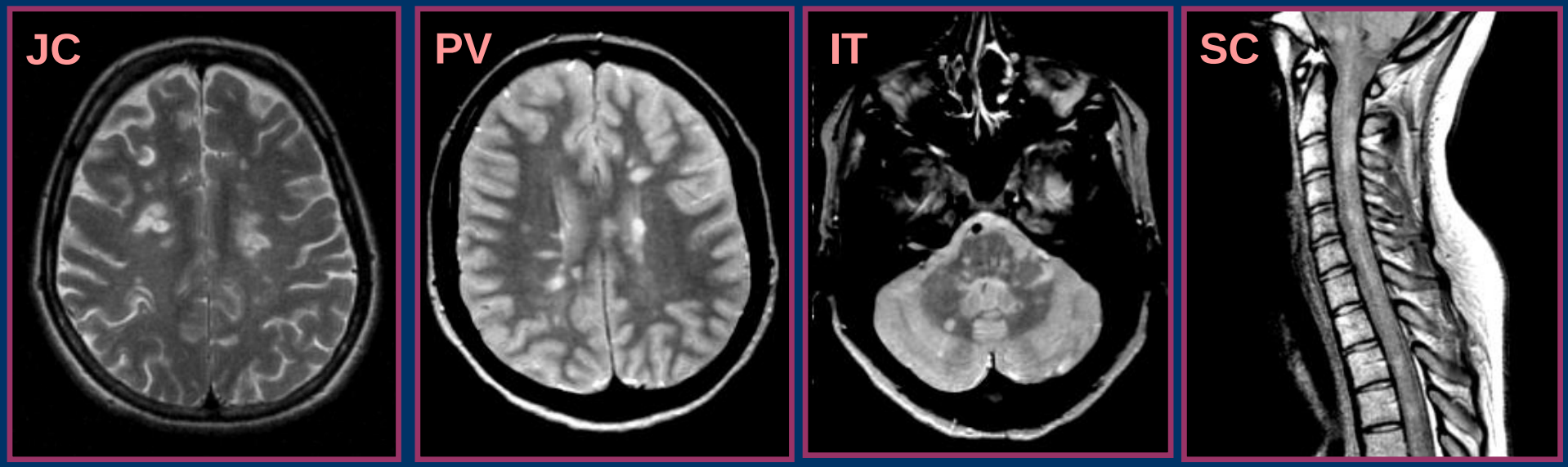
Solomon AJ. Neurology 2016

McDonald criteria - DIS



lesions in 2/4 locations required

Development setting representative?

- Tertiary referral centres
  - strict indications, dedicated training
  - prospective case finding, few incidental (MRI) findings
- Other diagnosis ruled out
  - using variety of tests
  - during FU – partly retrospective
- Issues to generalize to standard practice
  - lower clinical threshold, application to historical symptoms
  - incidental MRI findings
  - less experience with alternative Dx

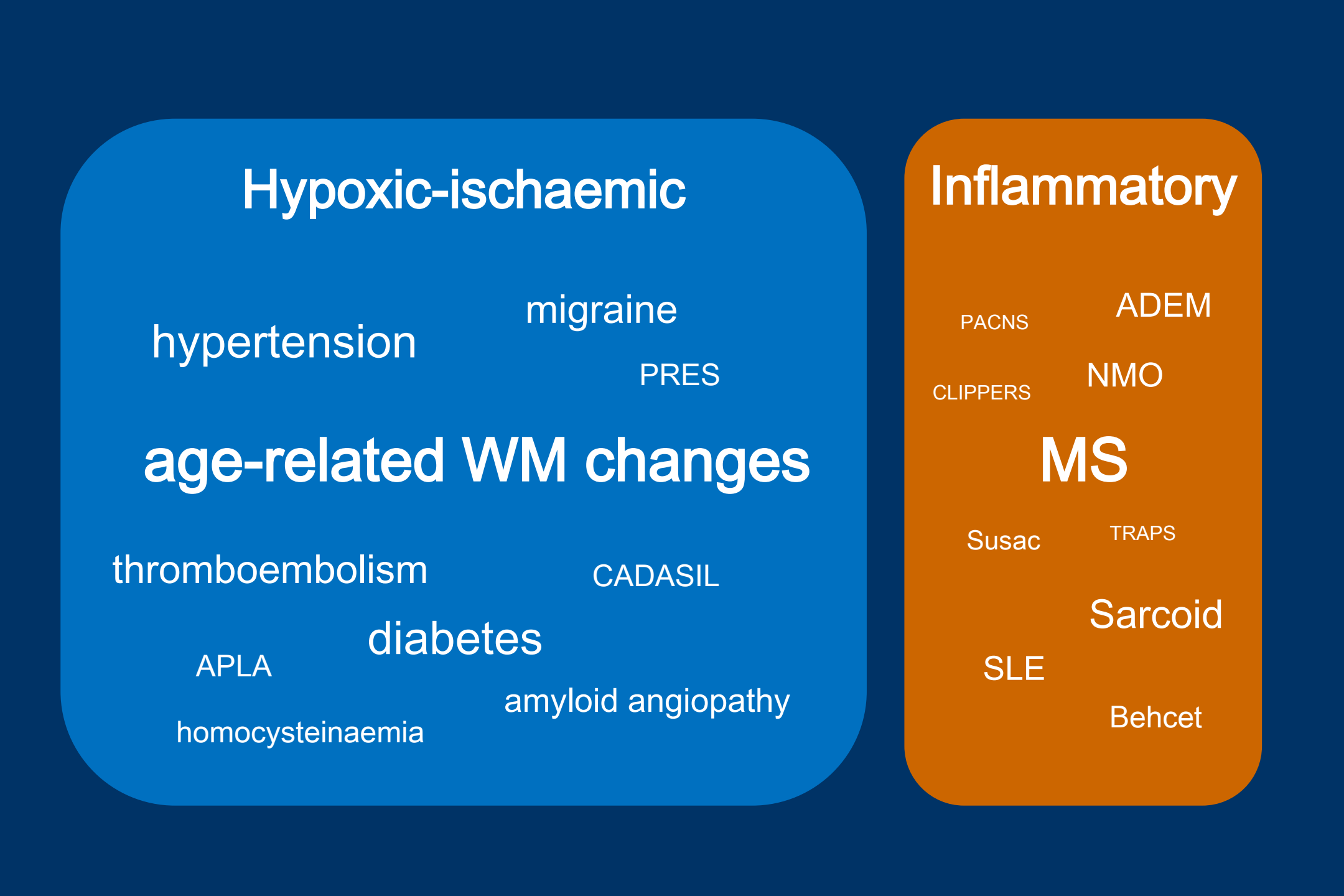

Hypoxic-ischaemic
migraine
hypertension
PRES
age-related WM changes
thromboembolism
CADASIL
diabetes
APLA
amyloid angiopathy
homocysteinaemia
Inflammatory
PACNS
ADEM
NMO
CLIPPERS
MS
Susac
TRAPS
Sarcoid
SLE
Behcet

Table 3. Alternative Diagnoses for Patients Without Multiple Sclerosis or Possible Multiple Sclerosis*

Alternative Diagnoses	No. of Patients
Other neurological disease	88
Migraine	25
Stroke	7
Neuropathy	6
Transverse myelitis	4
Cervical stenosis	4
Nonspecific headache	4
ADEM	4
Radiculopathy	3
CTS	3
BET	3
PAPS	2
Parkinson disease	2
Atypical facial pain	2
Arteriovenous malformation	2
Optic neuritis	2
Metabolic abnormality	2
Meningitis	2
Lumbar stenosis	1
Temporal arteritis	1
Fragile X	1
MSA	1
Sciatica	1
Hydrocephalus	1
Human immunodeficiency virus	1
Fistula	1
Perry-Romberg	1
Ulnar neuropathy	1
Encephalitis	1
Systemic lupus	1
Sjögren syndrome	1

Carmosino MJ. Arch Neurol 2005

Table 1 Diagnoses and syndromes mistaken for multiple sclerosis

	No. (%)
Migraine alone or in combination with other diagnoses	24 (22)
Fibromyalgia	16 (15)
Nonspecific or nonlocalizing neurologic symptoms with abnormal MRI	13 (12)
Conversion or psychogenic disorder	12 (11)
Neuromyelitis optica spectrum disorder	7 (6)
Clinically isolated syndrome	3 (3)
Neurodegenerative cerebellar syndrome	2 (2)
MRI changes caused by vascular disease	2 (2)
Parkinsonism with nonspecific white matter abnormalities	2 (2)
"Radiologically isolated syndrome"	2 (2)
Cervical spondylosis with myelopathy	2 (2)
Genetic leukodystrophy	2 (2)
Idiopathic transverse myelitis	2 (2)
Noninflammatory myelopathy	2 (2)
Nonspecific symptoms with positive CSF OCBs	2 (2)
Stroke, nonembolic	2 (2)
Anti-Ma2 paraneoplastic syndrome	1 (1)
Acute disseminated encephalomyelitis	1 (1)
Astrocytoma	1 (1)
Mitochondrial disorder	1 (1)
Neurosarcoidosis	1 (1)
Moyamoya disease	1 (1)
Hypertension and alcohol abuse	1 (1)
Neuropathy	1 (1)
Unclear diagnosis; complaints of paresthesias	1 (1)
Nonspecific or nonlocalizing neurologic symptoms with normal MRI	1 (1)
Viral meningoencephalitis with subsequent abnormal MRI and acute labyrinthitis	1 (1)
White matter lesions due to TNF-α inhibitor use for psoriasis	1 (1)
Behçet syndrome	1 (1)
CADASIL	1 (1)
Degenerative joint disease of lumbar spine	1 (1)

Solomon AJ. Neurology 2016

Reason for FP radiological diagnosis

Table 4. Etiology of T2 or Fluid-Attenuated Inversion Recovery (FLAIR) Lesions Other Than Multiple Sclerosis or Possible Multiple Sclerosis*

Etiology of T2 or FLAIR Lesions	No. (%) of Patients
Definite migraine	34 (37)
Age-related	11 (12)
Other neurological disease	10 (11)
Hypertension	9 (10)
Nonspecific headache	7 (8)
No cause identified	20 (22)
Total	**91**

Carmosino MJ. Arch Neurol 2005

Table 1 | **Studies assessing the DIS MS criteria in other neurological disorders**

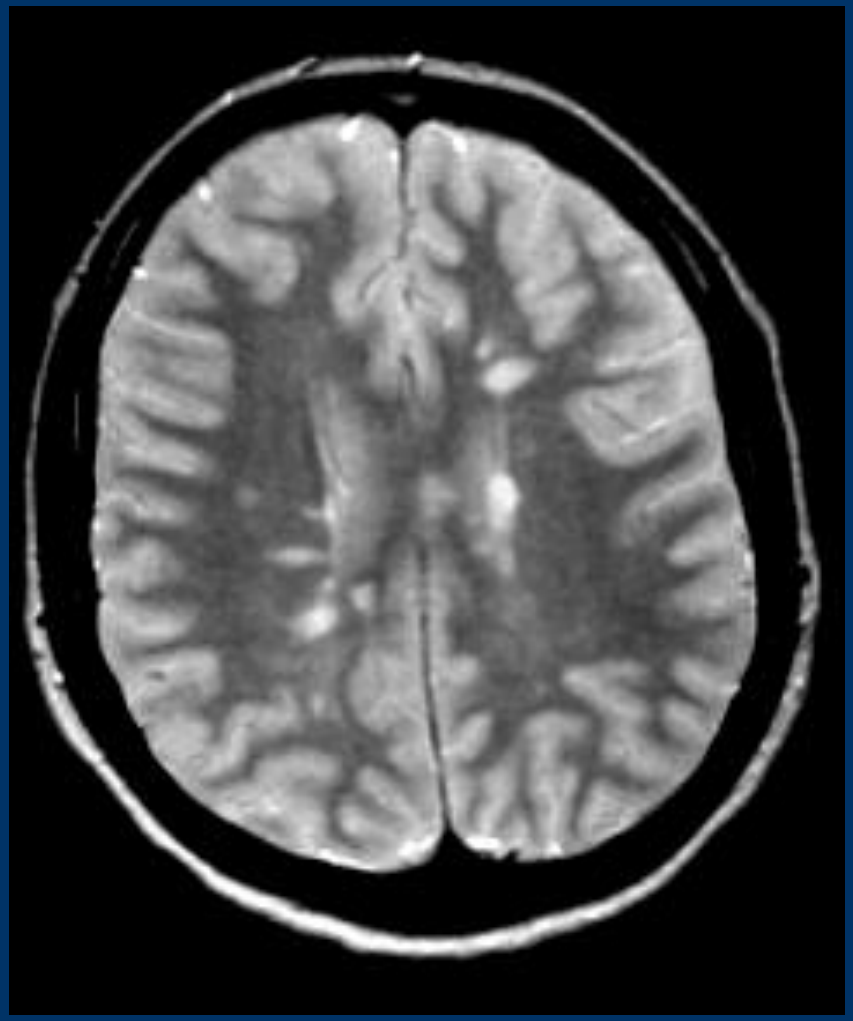
Disorder	Number of patients	Percentage of lesions that meet criteria		Refs.
		Barkhof criteria	McDonald 2010 criteria	
Migraine	44	NA	9	9
	168	7.1	34.5	8
	32	NA	34	10
Anti-AQ4 antibody-associated NMOSD	31	12.9	NA	13
	26	15.9	NA	11
	67	13	NA	12
Anti-MOG antibody-associated NMOSD	21	14.3	NA	13
	26	26.9	NA	14
Neuro-Behçet disease	84	13.1	NA	15
Primary CNS vasculitis	24	50	NA	16
Secondary vasculitis	25	58	NA	16
SLE or Sjögren syndrome	16	17	NA	16

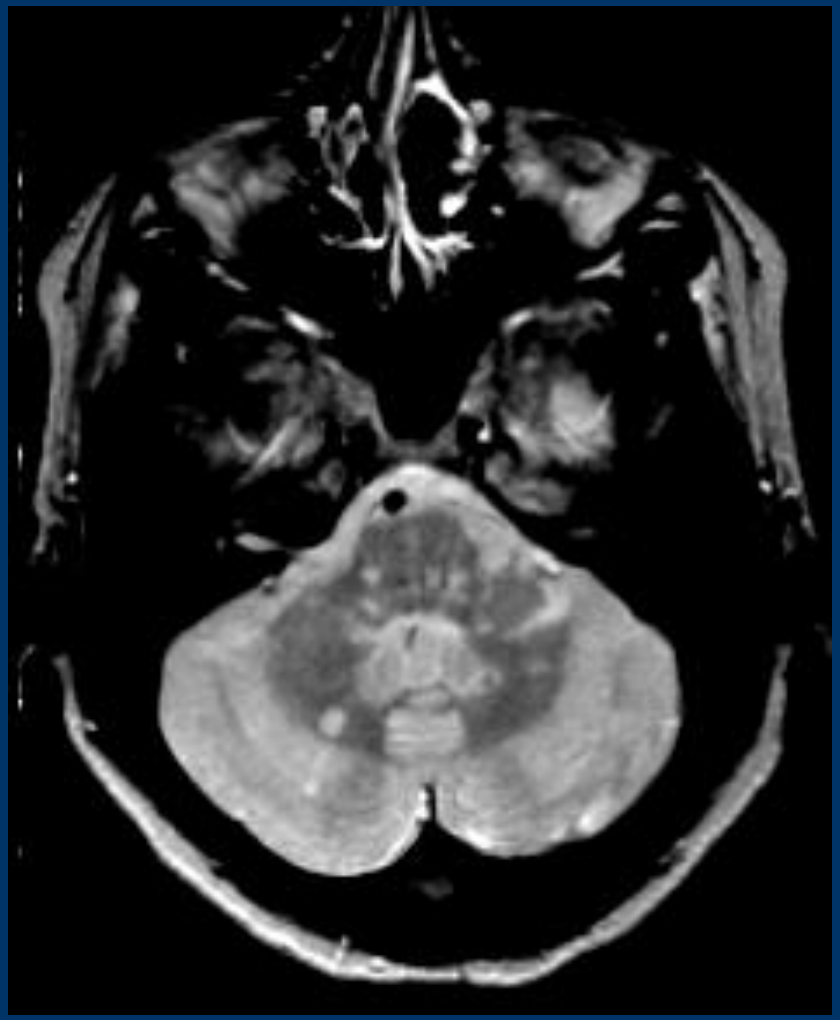
Geraldes & MAGNIMS, Nat Rev Neurol 2018

Prevalence of WM disorders

- hereditary – individual disorders rare
  - as a group less uncommon
- acquired WM disease
  - vascular 1-5/10
  - MS 1-2/1000
  - neuro-SLE 5/100.000
  - Lyme 1/100.000
- highest *a priori* chance for vascular lesions

MS – periventricular & infratentorial





MC question 1

Periventricular lesions can bee seen in

1. Multiple sclerosis (MS)
2. Neuromyelitis optica (NMO)
3. Cerebrovascular disease (CVD)
4. All of the above

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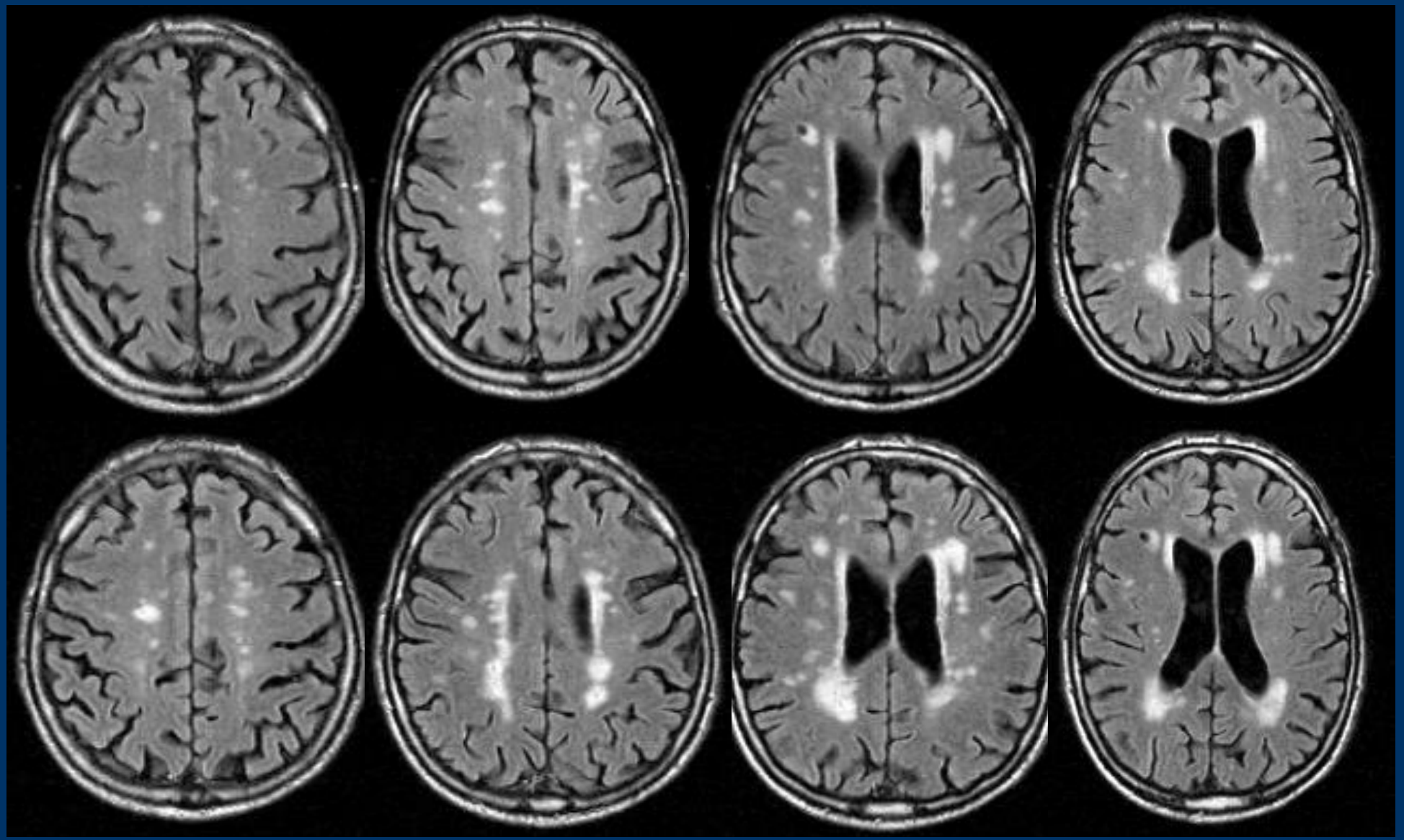
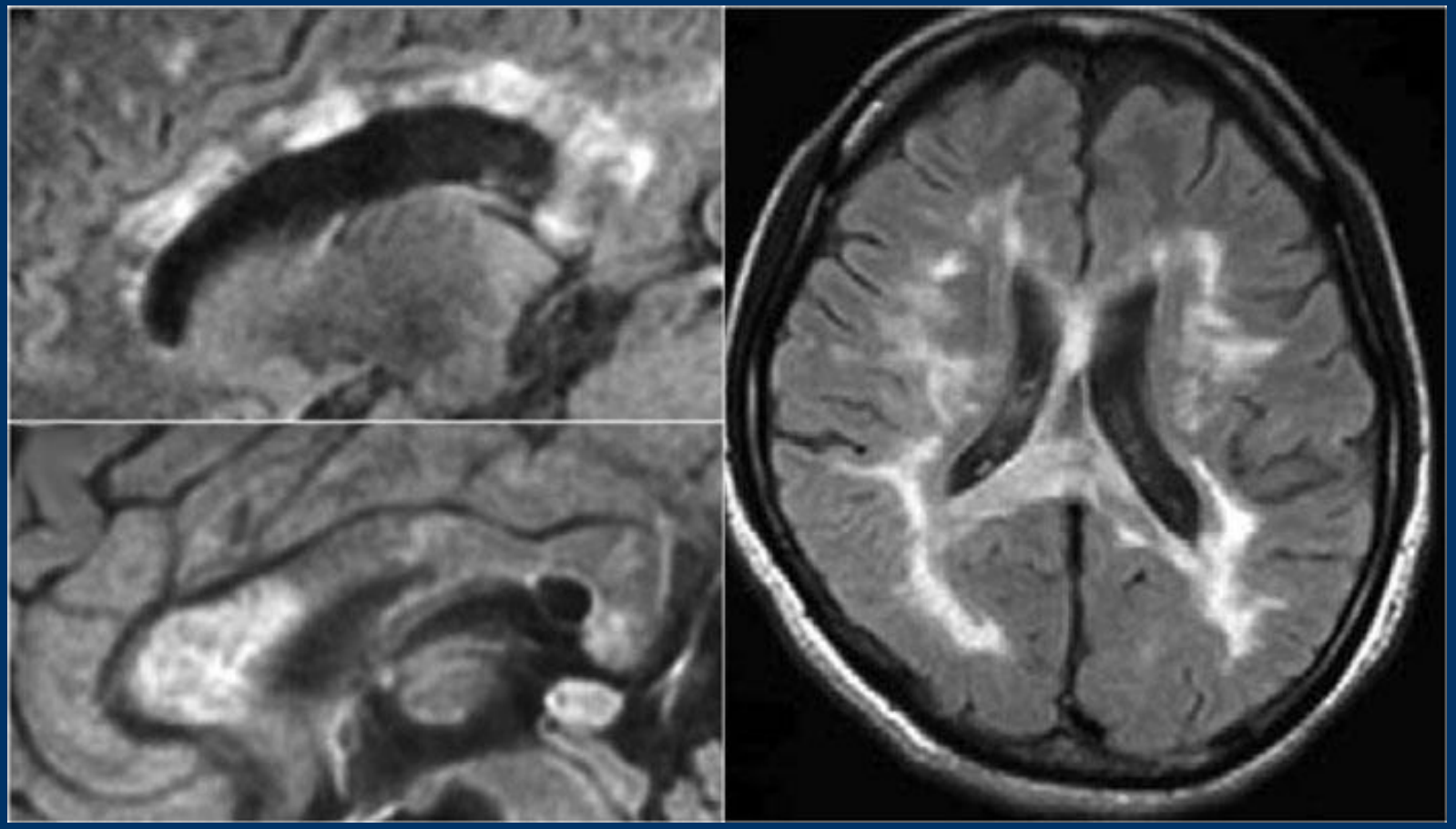
SVD - periventricular involvement



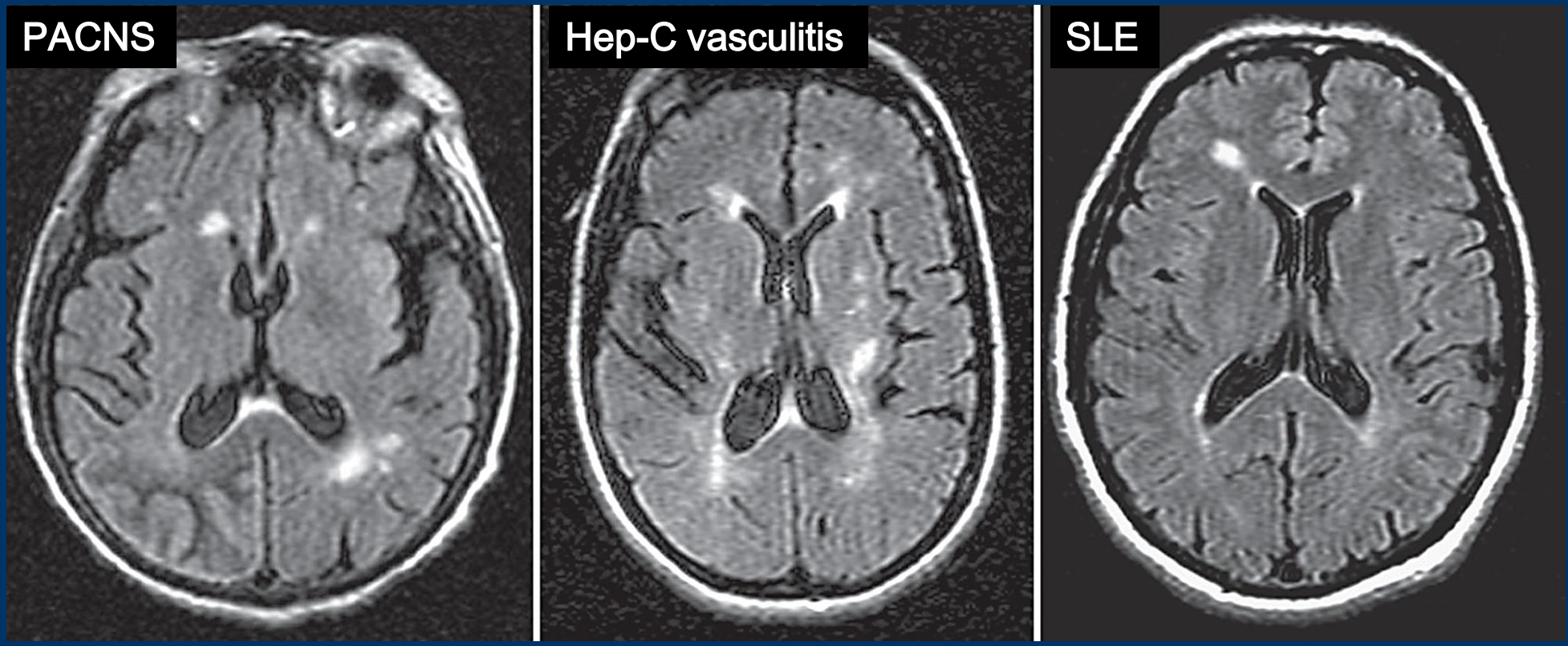
*5039676

PV lesions in NMO



Courtesy dr. Nakamura – www.radiologyassistent.nl

PV lesions in OND



Barkhof criteria distinguish CDMS (60%) from SLE / Sjogren's syndrome (17%, p = 0.0173)
but not from primary CNS vasculitis (50%, p = 0.7376) or secondary CNS vasculitis (58%, p = 1.0)
Three other MRI criteria were superior: any ovoid periventricular T2 lesions,
any perpendicular periventricular T2 lesions, any T2 lesions larger than 6 mm.

Kim S. MSJ 2013

Panel 2: Recommended 2016 MAGNIMS MRI criteria to establish disease dissemination in space in multiple sclerosis

Dissemination in space can be shown by involvement* of at least two of five areas of the CNS as follows:

- Three or more periventricular lesions
- One or more infratentorial lesion
- One or more spinal cord lesion
- One or more optic nerve lesion
- One or more cortical or juxtacortical lesion†

*If a patient has a brainstem or spinal cord syndrome, or optic neuritis, the symptomatic lesion (or lesions) are not excluded from the criteria and contribute to the lesion count. †This combined terminology indicates the involvement of the white matter next to the cortex, the involvement of the cortex, or both, thereby expanding the term juxtacortical lesion.

Filippi M, Lancet Neurology 2016

Specificity McD criteria in OND

- 28 / 377 referred cases ultimately alternative Dx
  - 16 ischemic cerebrovascular disease (CVD)
  - 4 angiitis/vasculitis, 3 multisystem atrophy (MSA), 5 other single Dx
- matched with 28 typical definite MS cases

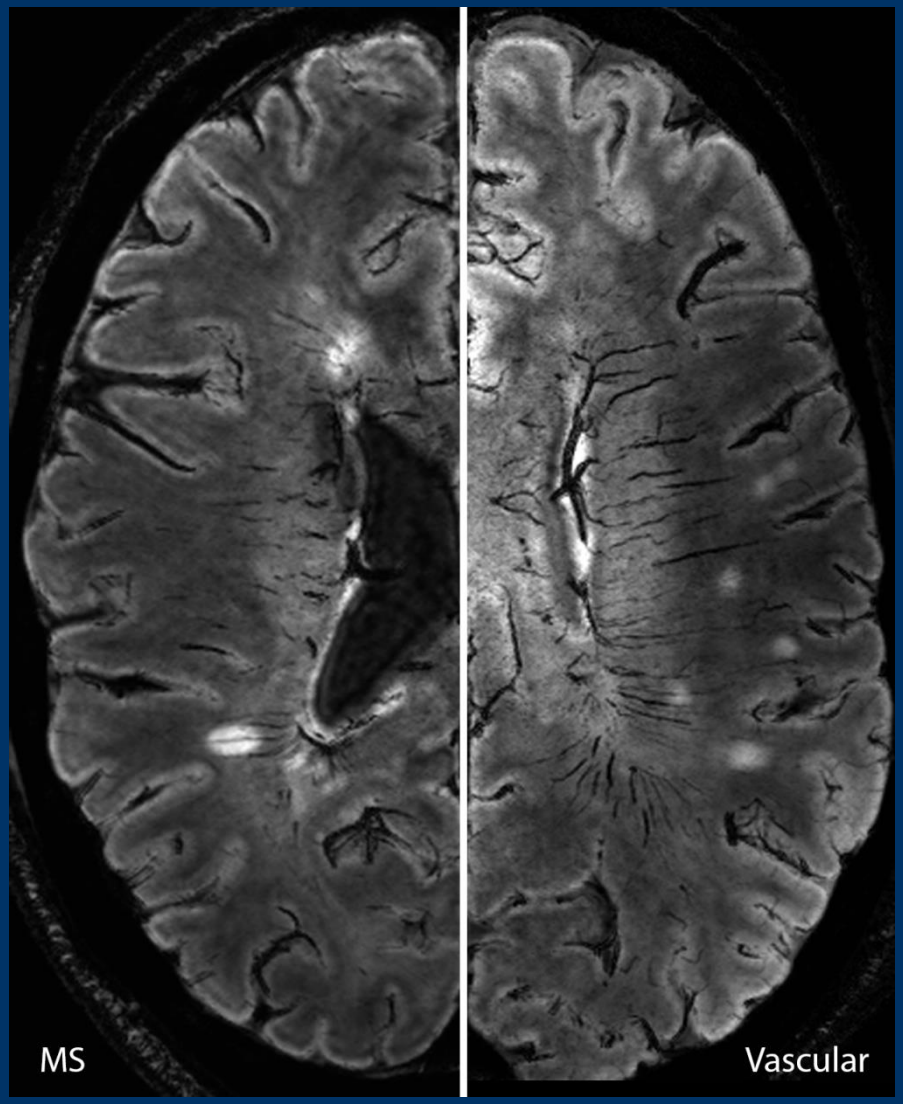
Table 2. MRI Characteristics

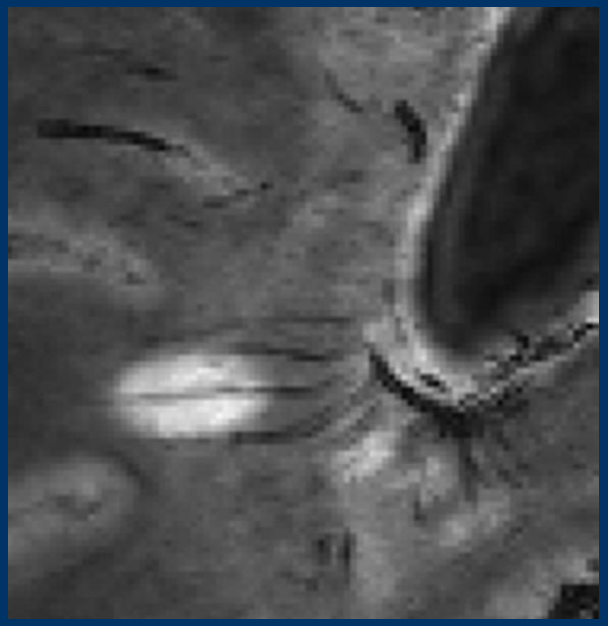
Imaging Findings	OND (n = 28)	MS (n = 28)	Odds Ratios MS vs OND (95% CI)
Nine or more T2 lesions	14 (50%)	22 (79%)	3.7 (1.1–11.8)
Infratentorial lesion present	7 (25%)	18 (64%)	5.4 (1.7–17.1)
Juxtacortical lesion present	4 (14%)	15 (54%)	6.9 (1.9–25.2)
Three periventricular lesions present	5 (18%)	21 (75%)	13.8 (3.8–50.2)
Three or more T2 lesions	20 (71%)	26 (93%)	5.2 (1–27.2)

MRI = magnetic resonance imaging; OND = other neurological disease; MS = multiple sclerosis; CI = confidence interval.

Nielsen JM, Ann Neurol 2005

FLAIR* @ 7T – perivenous lesions

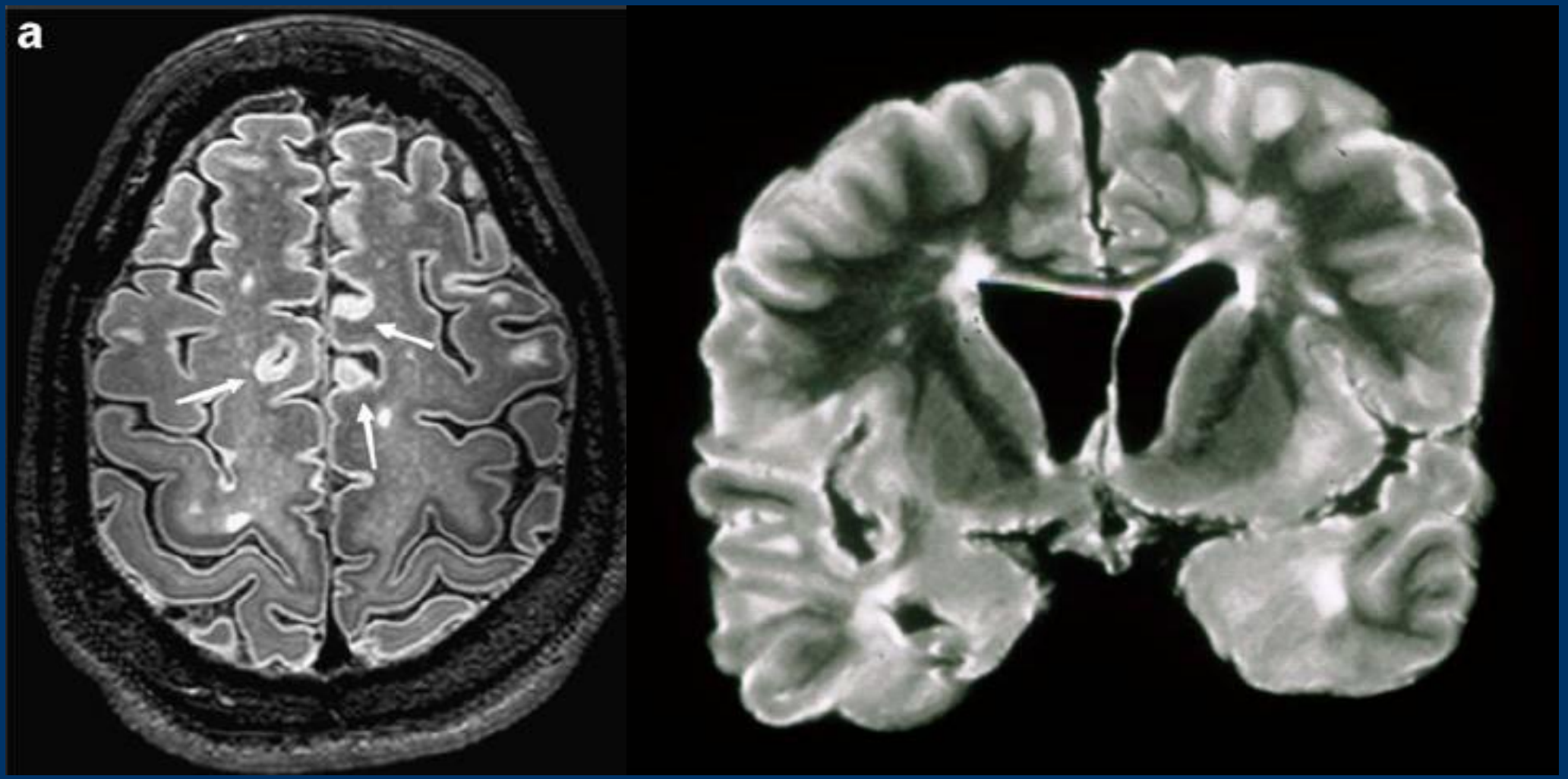




More work needed to translate to 3T and below
Needs better criteria for interpretation

Kildonk I, Eur Radiol 2013

Cortico-juxtacortical lesions in MS



MC question 2

Cerebrovascular lesions do NOT affect

1. Centrum semi-ovale
2. Brain stem
3. U-fibres
4. Basal ganglia

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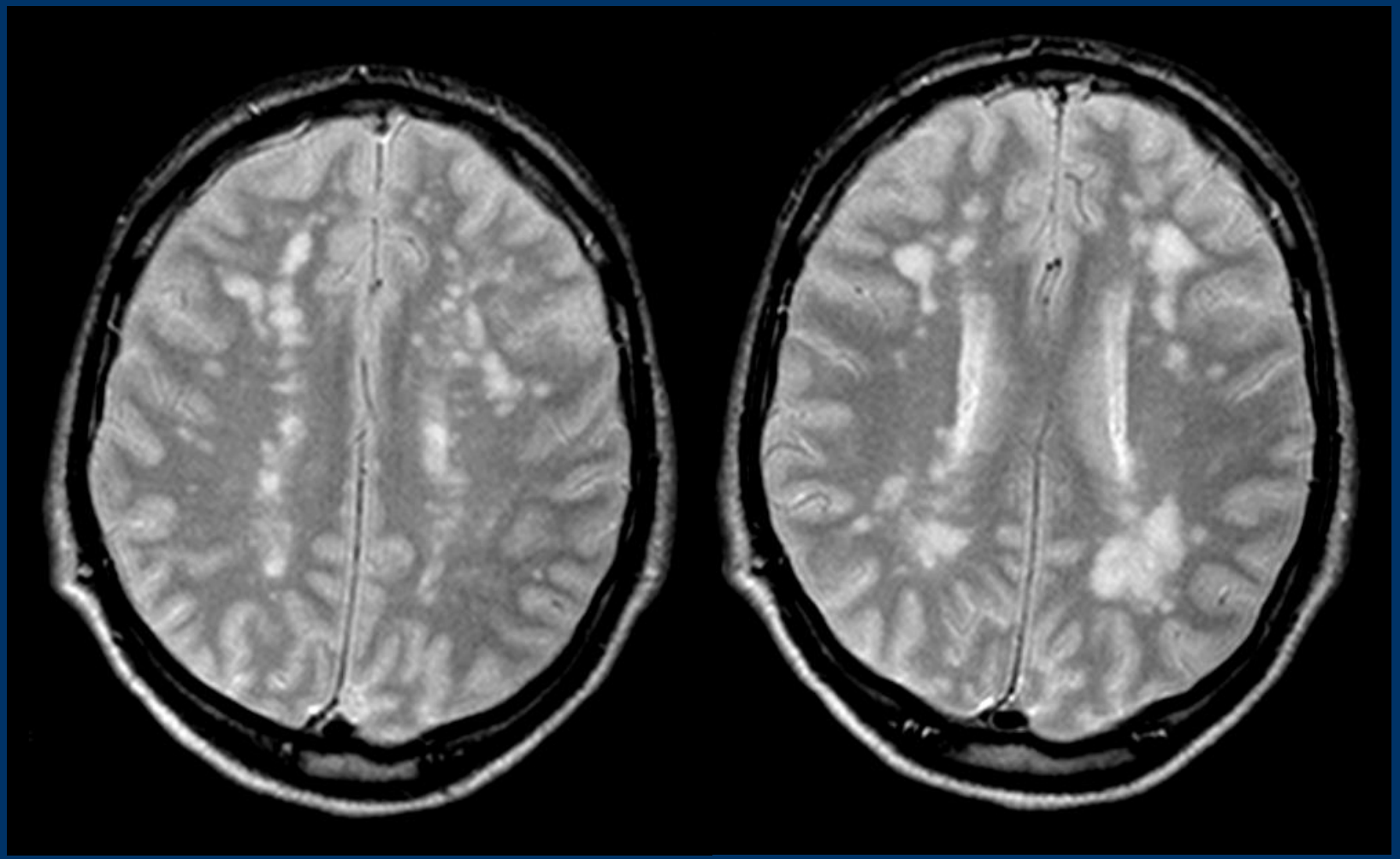
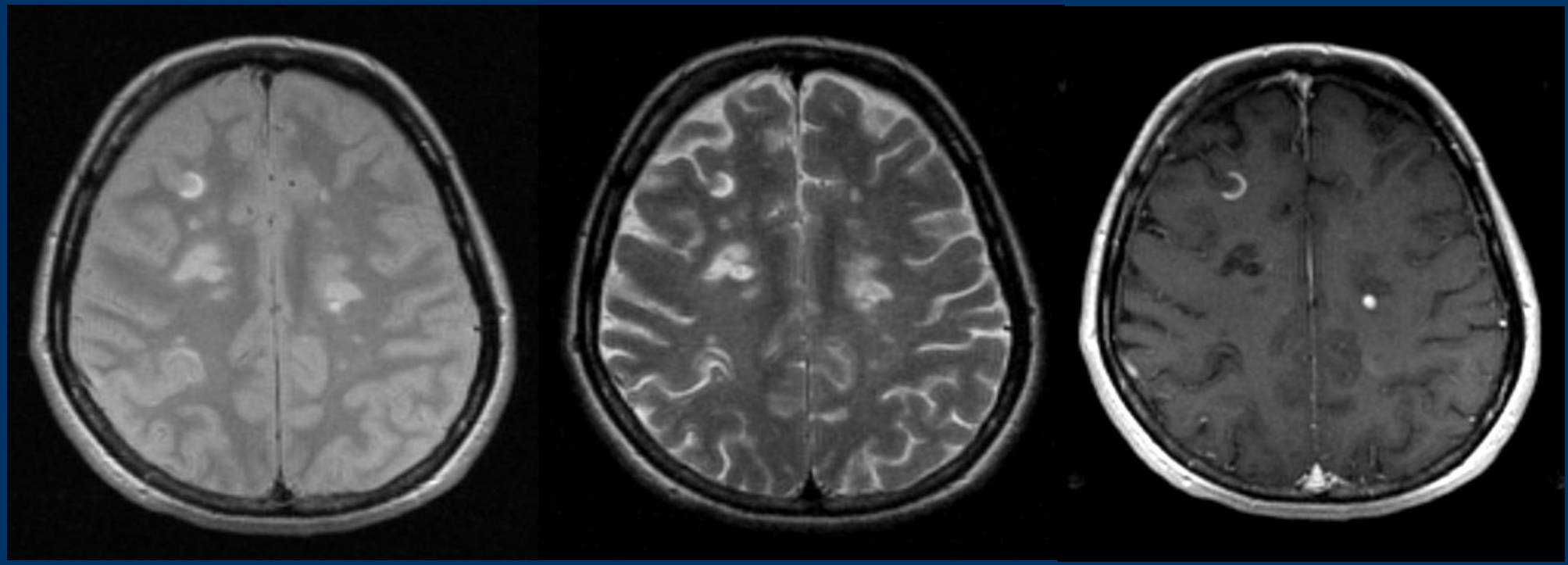
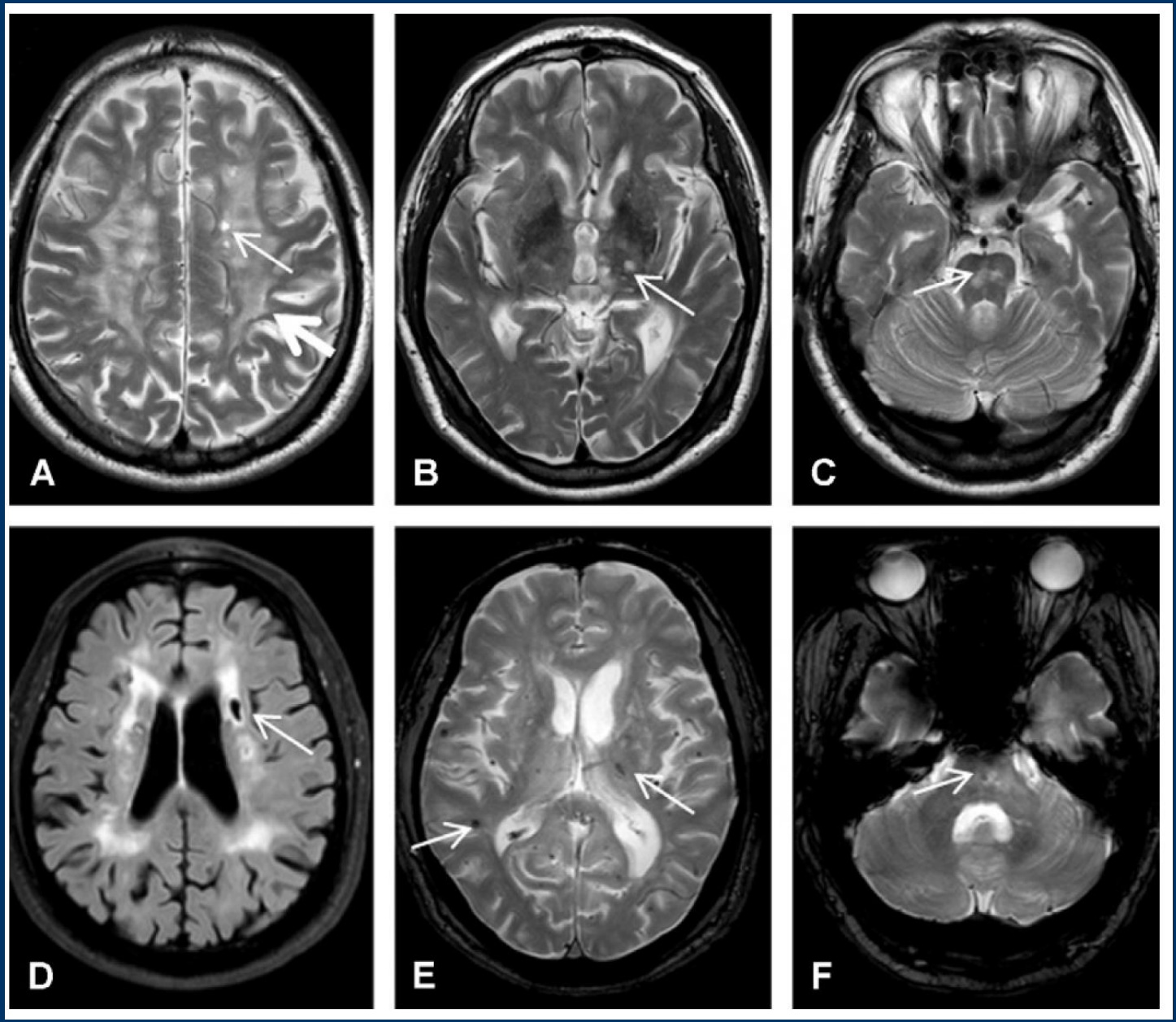
SVD – sparing of U-fibers



MS – juxtacortical inflammation

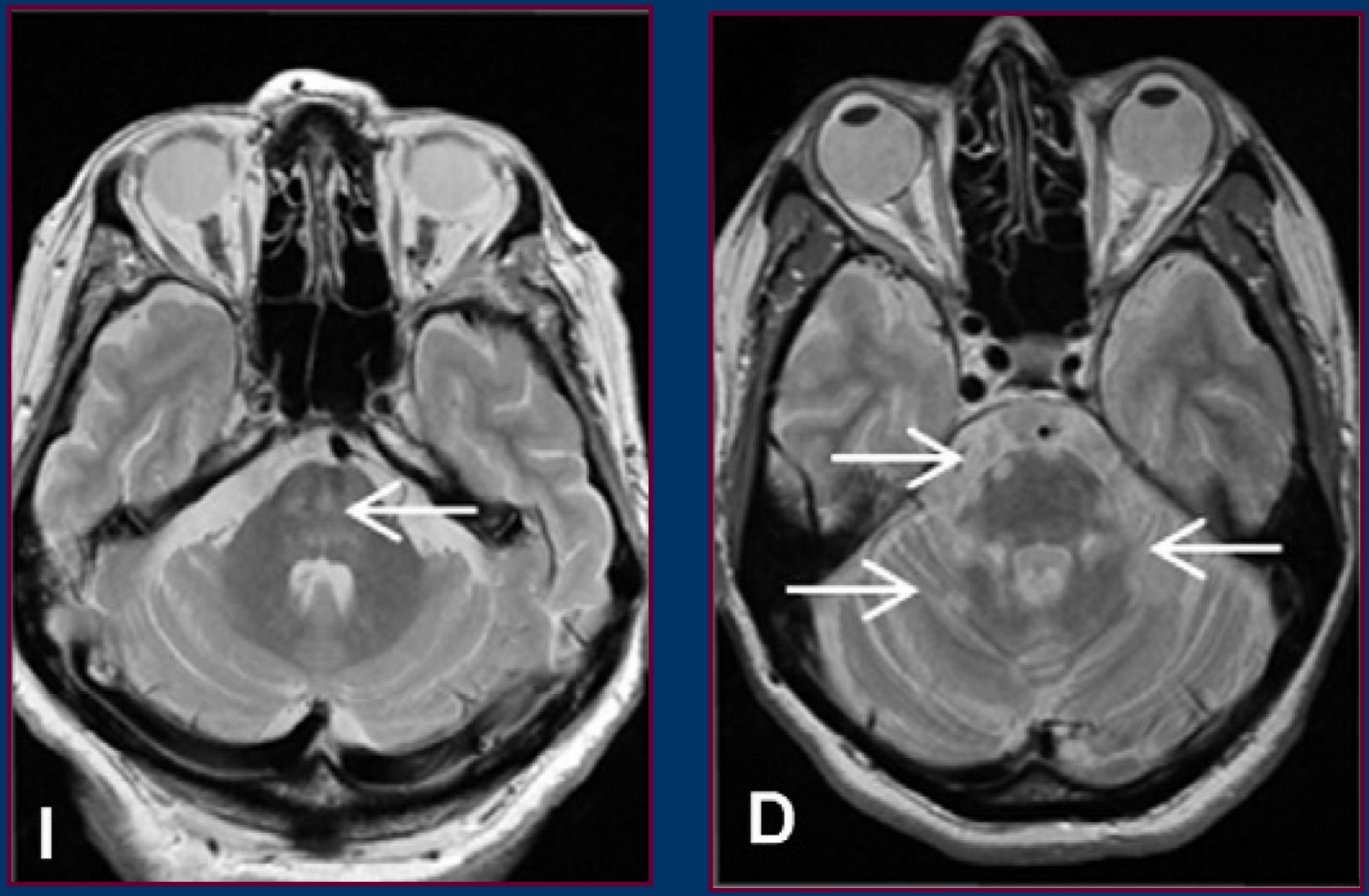


SVD



Sanchez-Alliaga & Barkhof. Handbook of Clinical Neurology

Brain stem lesions – SVD vs MS

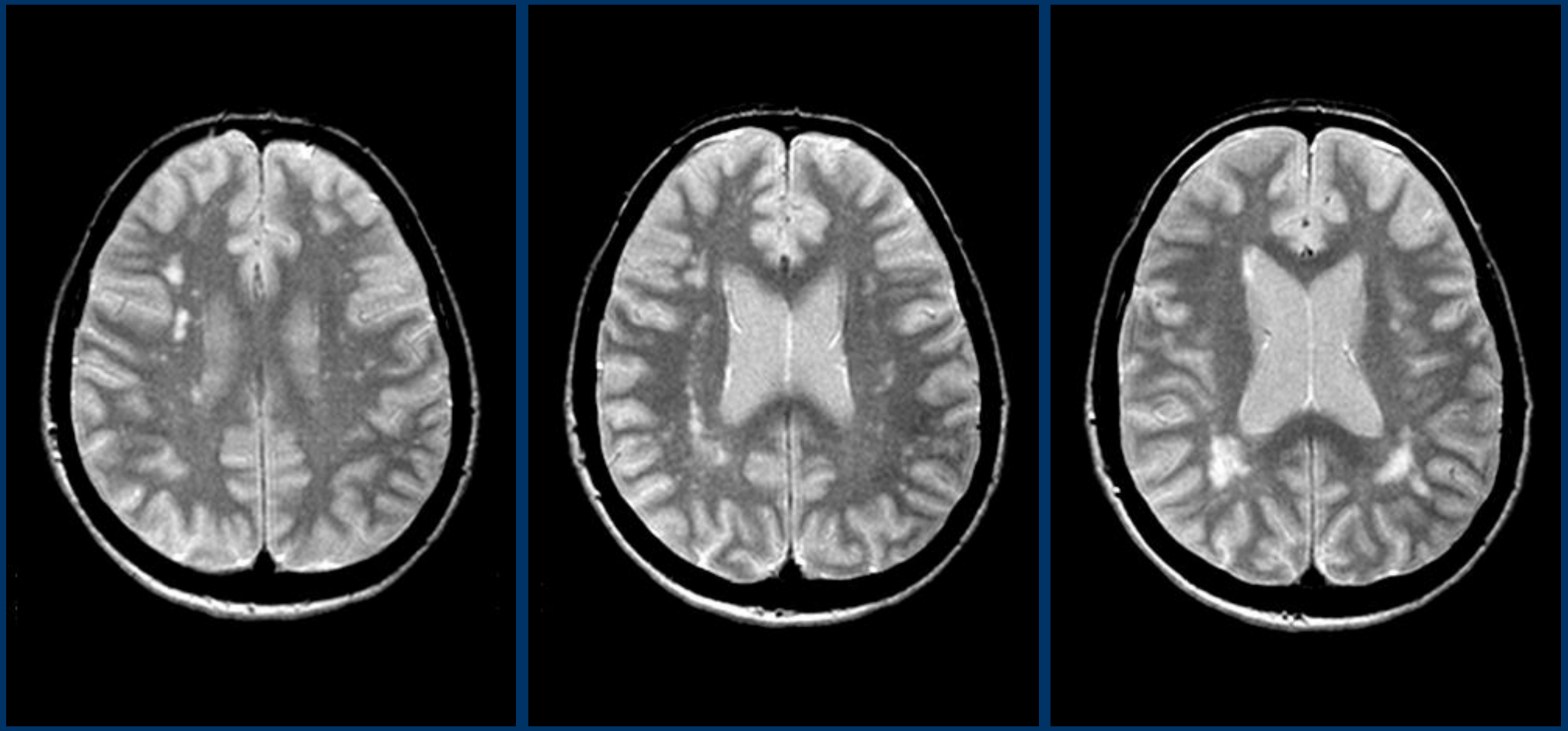


Sanchez-Alliaga & Barkhof. Handbook of Clinical Neurology

Table 2 | **MRI features that suggest cerebral small vessel disease**

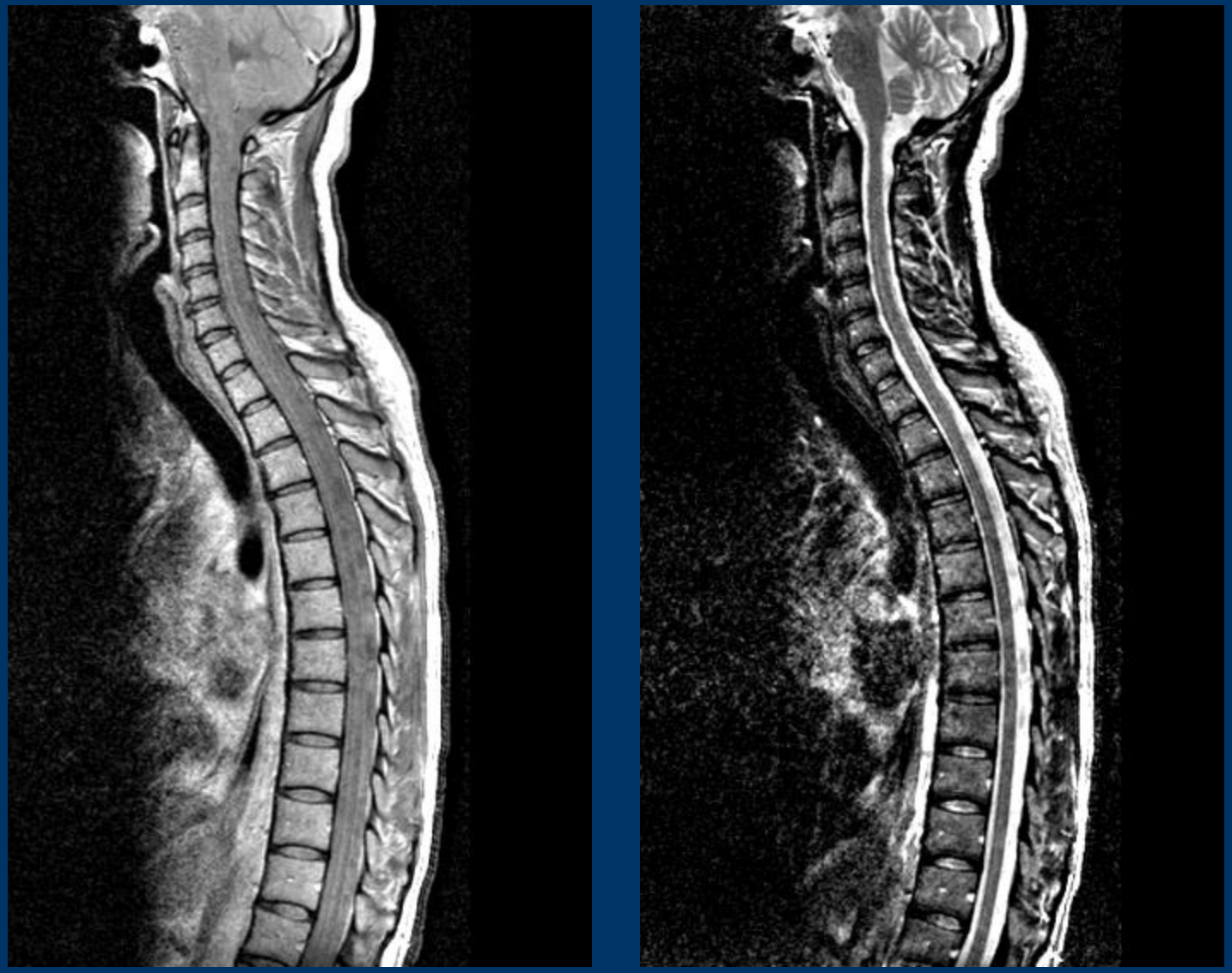
Small vessel disease type	Differentiating features
CADASIL[a]	WMLs in the external capsule and temporal poles, and lacunae in the basal ganglia and central pons
COL4A1 mutations[a]	Arterial dilatation and/or aneurysms, porencephaly and microbleeds
Fabry disease[a]	Vertebrobasilar arterial dolichoectasia, pulvinar T1 hyperintensity, and infarcts
Arteriosclerotic or related to age and vascular risk factors	• Lesions (microbleeds and lacunae) in perforating artery territory (basal ganglia, brainstem) • Symmetrical, poorly demarcated deep WMLs that spare U-fibres • Central pontine diffuse white matter changes and infarcts • Spared spinal cord
Cerebral amyloid angiopathy (sporadic and hereditary)	Lobar microbleeds and macrobleeds, convexity subarachnoid haemorrhages and/or cortical siderosis
Inflammatory or immune-mediated (for example, vasculitis associated with connective tissue disorders or primary systemic vasculitis with cerebral involvement) and infectious vasculitis	Meningeal enhancement, lacunae, microbleeds, territorial infarcts, pseudotumoural lesions in the basal ganglia and/or brainstem, and longitudinal extensive transverse myelitis
Other (for example, post-radiation angiopathy)	Diffuse WMLs, sometimes with cavitation owing to coagulative necrosis; distal artery thinning detectable with angiography

Small vessel disease or MS?

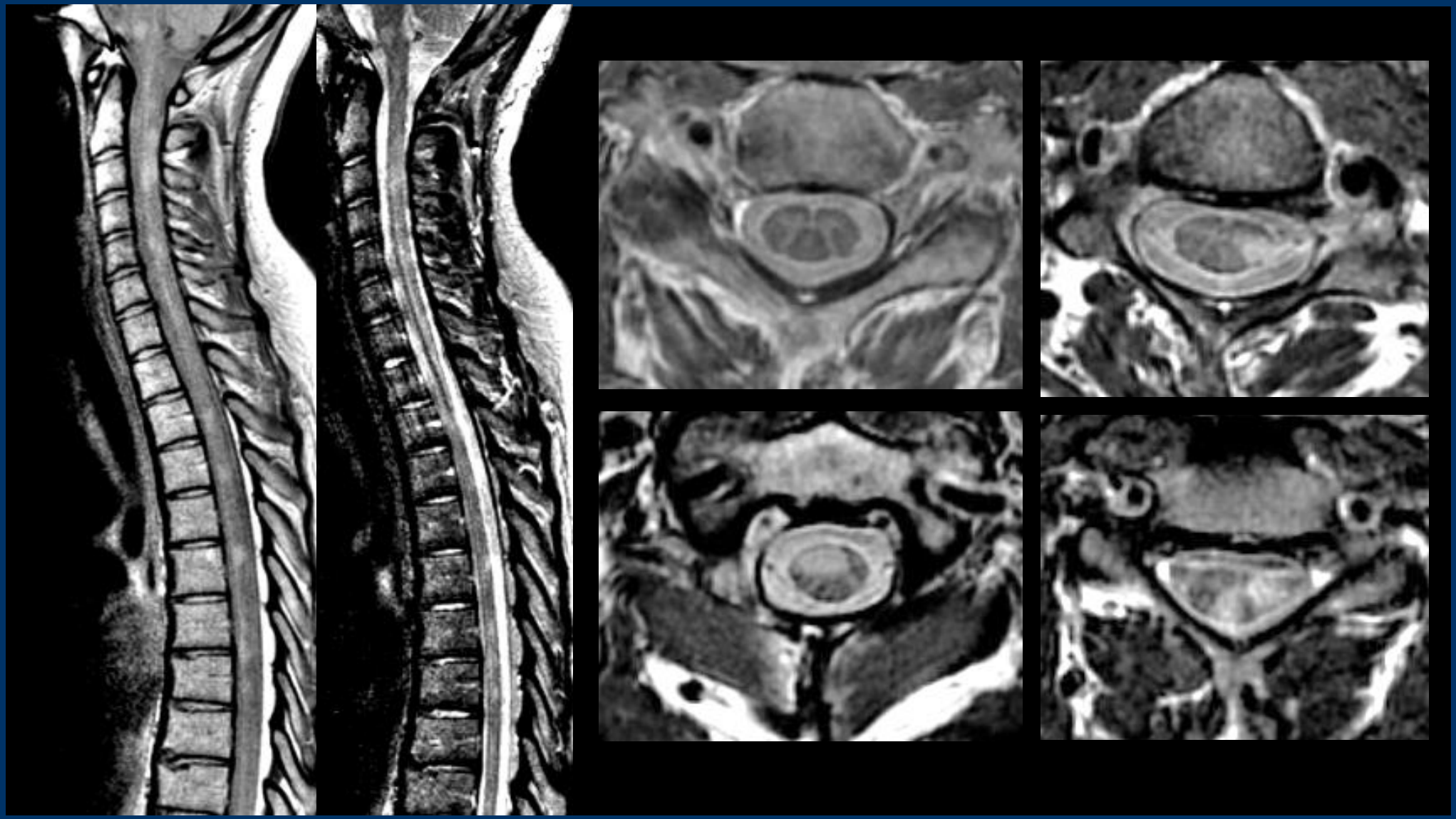


*2982144

not MS…..



MS – multifocal short lesions



Spinal cord abnormality – MS vs OND

	Spinal cord normal	Spinal cord abnormal
OND (n = 43)	39	4
MS (n = 25	2	23

Z=-7.01, p<0.0001

*OND: hyperhomocysteinemia, vasculitis

Bot JCJ. *Radiology* 2002

Involvement	MS	SVD
• CC	typical	rare
• U-fiber	often	rare
• Infratentorial	always	late
• Temporal lobe	often	never*
• Gad-enhancement	common	never
• Asymmetric	never	possible
• Black holes	typical	rare
• Cystic lesions	never	lacunes
• Spinal cord	frequent	never

www.radiologyassistant.nl

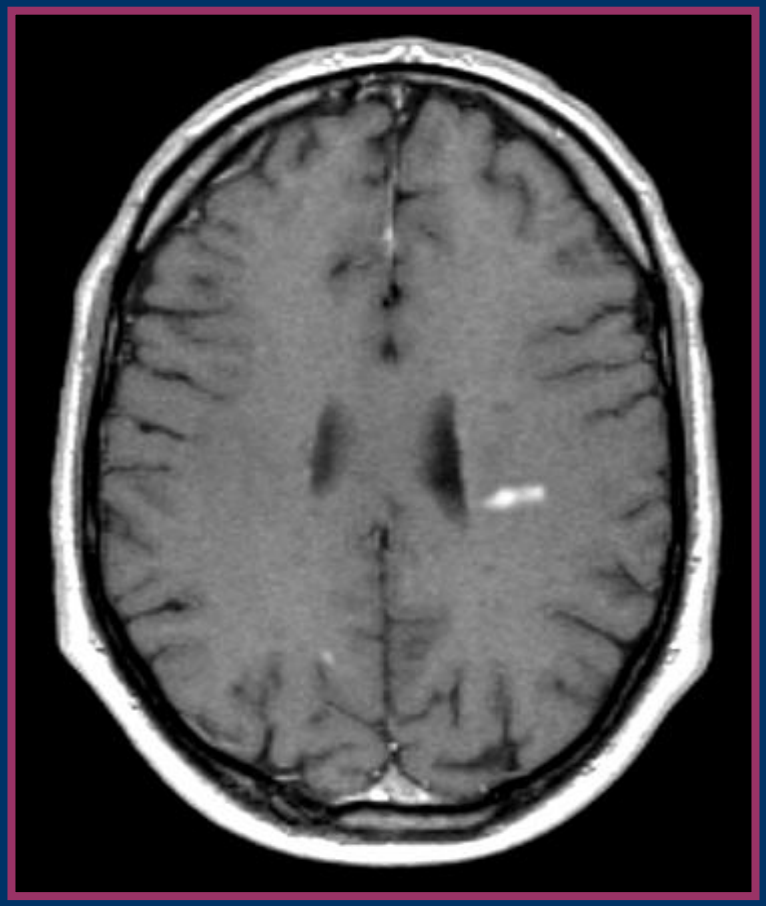
*except CADASIL

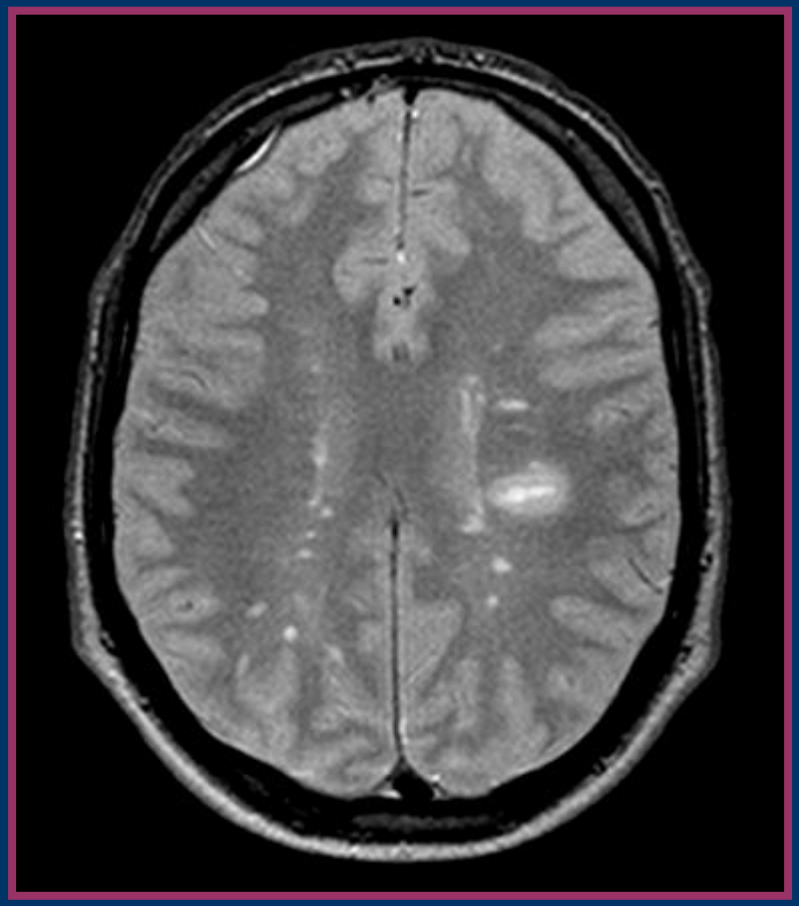
Table 3 | **MRI observations that differentiate between multiple sclerosis and the indicated disorders**

Type of observation	Observations	Possible disorders
Brain MRI		
Lesion location	Central and diencephalic (thalamus, basal ganglia and hypothalamus)	NMOSD, other autoantibody-mediated diseases (for example, anti-MA2 antibody encephalopathy[171]), ADEM, Susac syndrome, neurosarcoidosis, infection (for example, Whipple disease), metabolic disorders (for example, hyponatraemia and thiamine deficiency) and mitochondrial disorders
	Adjacent to third and fourth ventricles or aqueduct; area postrema	NMOSD
	Involving or following corticospinal tracts	NMOSD, HTLV1 and globoid cell leukodystrophy
	Lack of temporal and lateral ventricle lesions, lack of Dawson fingers or lack of S-shaped U-fibre lesion	NMOSD, migraine and inherited leukodystrophies
	Posterior limb of internal capsule ('string of beads')	Susac syndrome
	Lateral geniculate body or optic radiations	Adrenoleukodystrophy
	Central pons	SVD and metabolic disease (for example, hyponatraemia)
	Brainstem pial FLAIR hyperintensity; tadpole atrophy (atrophy of the medulla and spinal cord with relative sparing of the pons)	Type II (late-onset) Alexander disease[153]
	Crescent-shaped lesions involving the middle cerebellar peduncles and adjacent pontine white matter	Progressive multifocal leukoencephalopathy
	Dentate nucleus (T2 hyperintensities)	Cerebrotendinous xanthomatosis
	Bilateral occipital white matter	PRES, X-linked adrenoleukodystrophy and globoid cell leukodystrophy

Lesion characteristics	Cerebrospinal fluid-like signal intensity	Dilated Virchow–Robin spaces
	Indistinct margins	NMOSD, ADEM and other antibody-mediated encephalopathies (for example, anti-$GABA_A$)
	Symmetrical lesions	NMOSD, ADEM, migraine and inherited leukodystrophies
	Punctate (<5 mm diameter), rarely confluent lesions	Migraine and SLE
	Oedematous and marbled callosal lesion with or without extension into cerebral hemispheres (the 'arch bridge sign')	NMOSD and lymphoma
	Central 'snowball'-shaped callosal lesion	Susac syndrome
	Callosal thinning	Adult-onset autosomal dominant leukodystrophy, vanishing white matter disease and Susac syndrome
	Extensive, confluent, tumefactive hemispheric white matter lesions	NMOSD, cerebral vasculitis, neuro-Behçet disease, infection and cancer
	Associated with silent infarcts and/or microbleeds	Migraine, dilated Virchow–Robin spaces, cerebral vasculitis, Susac syndrome, CADASIL, COL4A1, Fabry disease and fat embolism
	Associated with convexity haemorrhage	Reversible vasoconstriction syndrome in association with PRES and cerebral amyloid angiopathy
	Associated with cranial nerve and leptomeningeal contrast enhancement	Cerebral vasculitis, Susac syndrome, neurosarcoidosis and infection (for example, neuroborreliosis)
	Associated with dural masses	Neurosarcoidosis and cerebral vasculitis (for example, GPA)
	None between relapses or rare new lesions	NMOSD, ADEM and migraine
Lesion activity	Absence of contrast enhancement	Migraine and dilated Virchow–Robin spaces
	Punctate and curvilinear enhancement lesions in the pons	CLIPPERS
	Linear perivascular radial gadolinium enhancement extending outward from the ventricles and in the cerebellum	Glial fibrillary acidic protein antibody disease[162]
	Unusual enhancing patterns — poorly marginated, patchy, cloud-like, rare meningeal or linear of ependymal lateral ventricles	NMOSD, neurosarcoidosis and cancer
Optic nerve MRI		
Lesion characteristics	Long lesion, bilateral	NMOSD
	Posterior, chiasmatic	Anti-AQP4 antibody-associated optic neuritis
	Long lesion, anterior	Anti-MOG antibody-associated optic neuritis

Perivenous inflammation in MS





Dissemination in time (DIT)

MC question 3

The enhancement pattern in MS can be

1. Pancake-like
2. Punctate
3. Leptomeningeal
4. Open-ring

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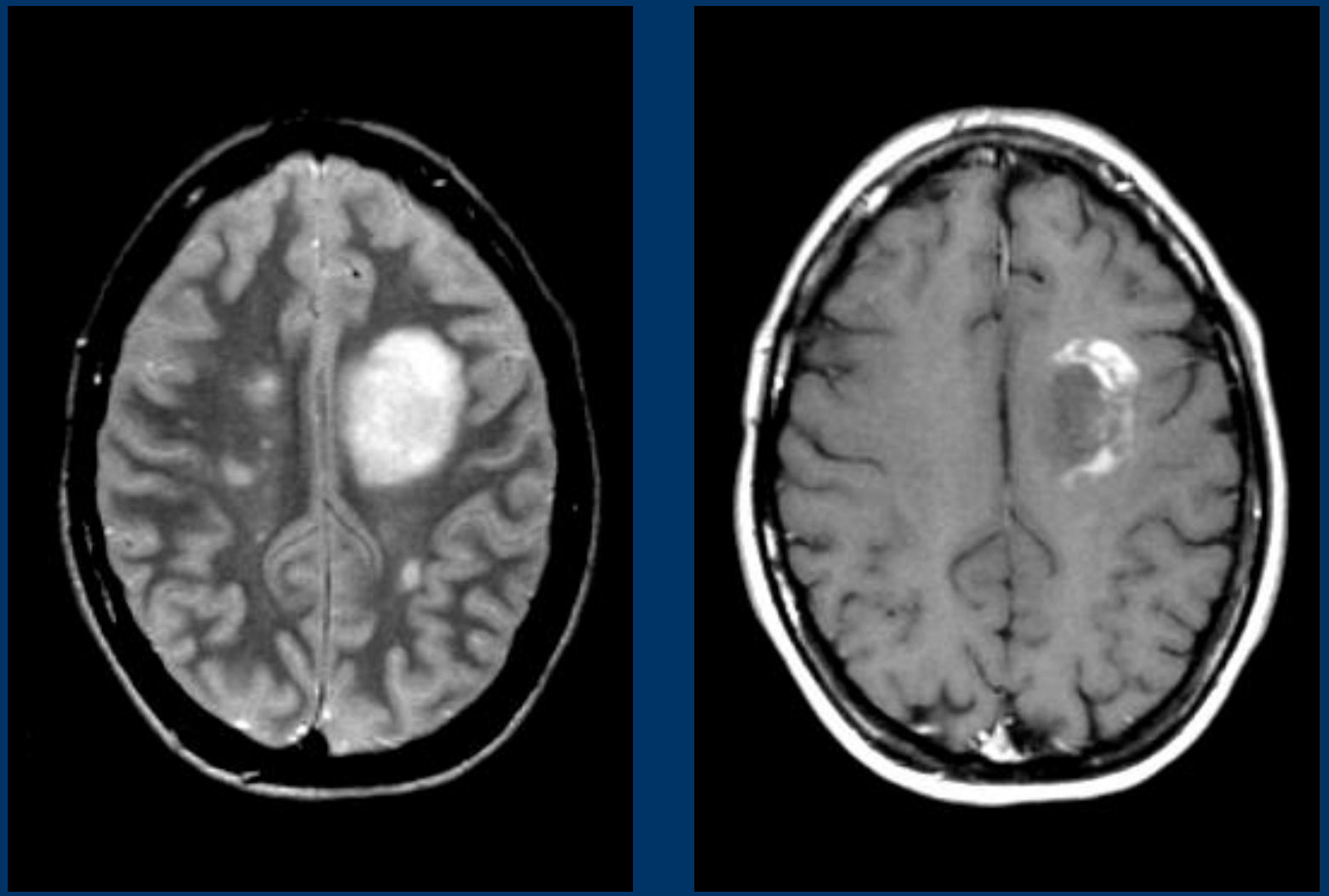
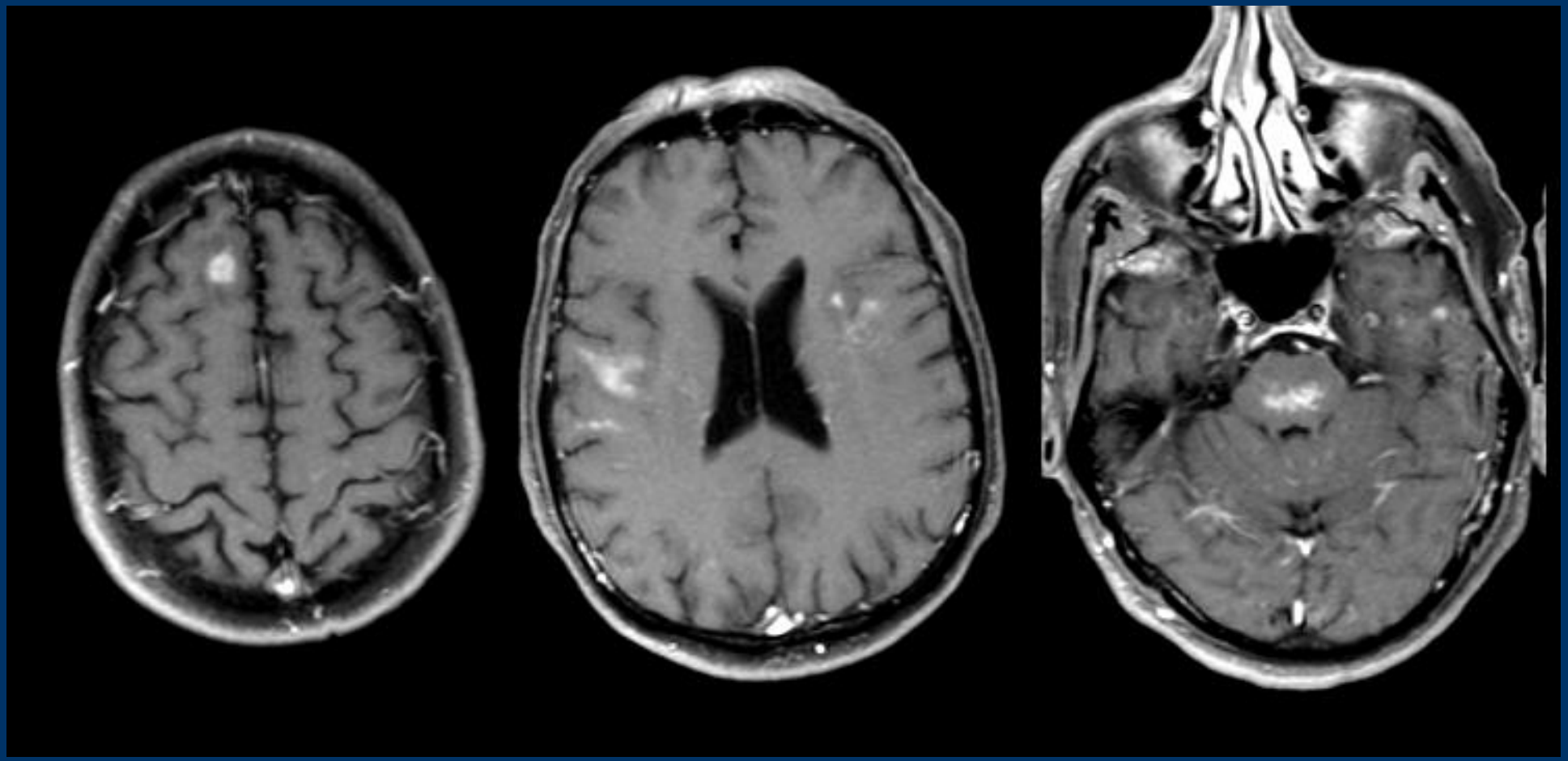
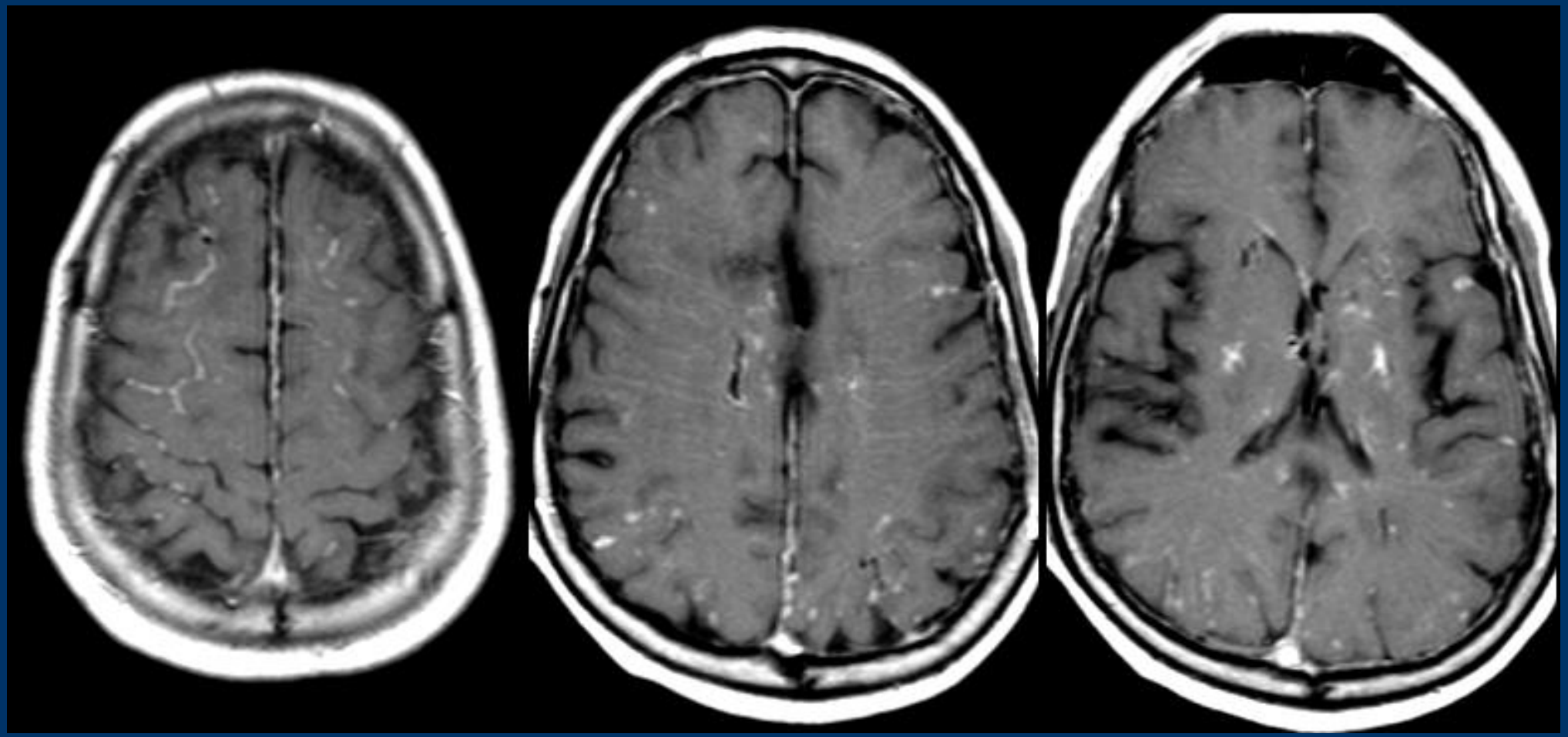
“open-ring” enhancement in MS



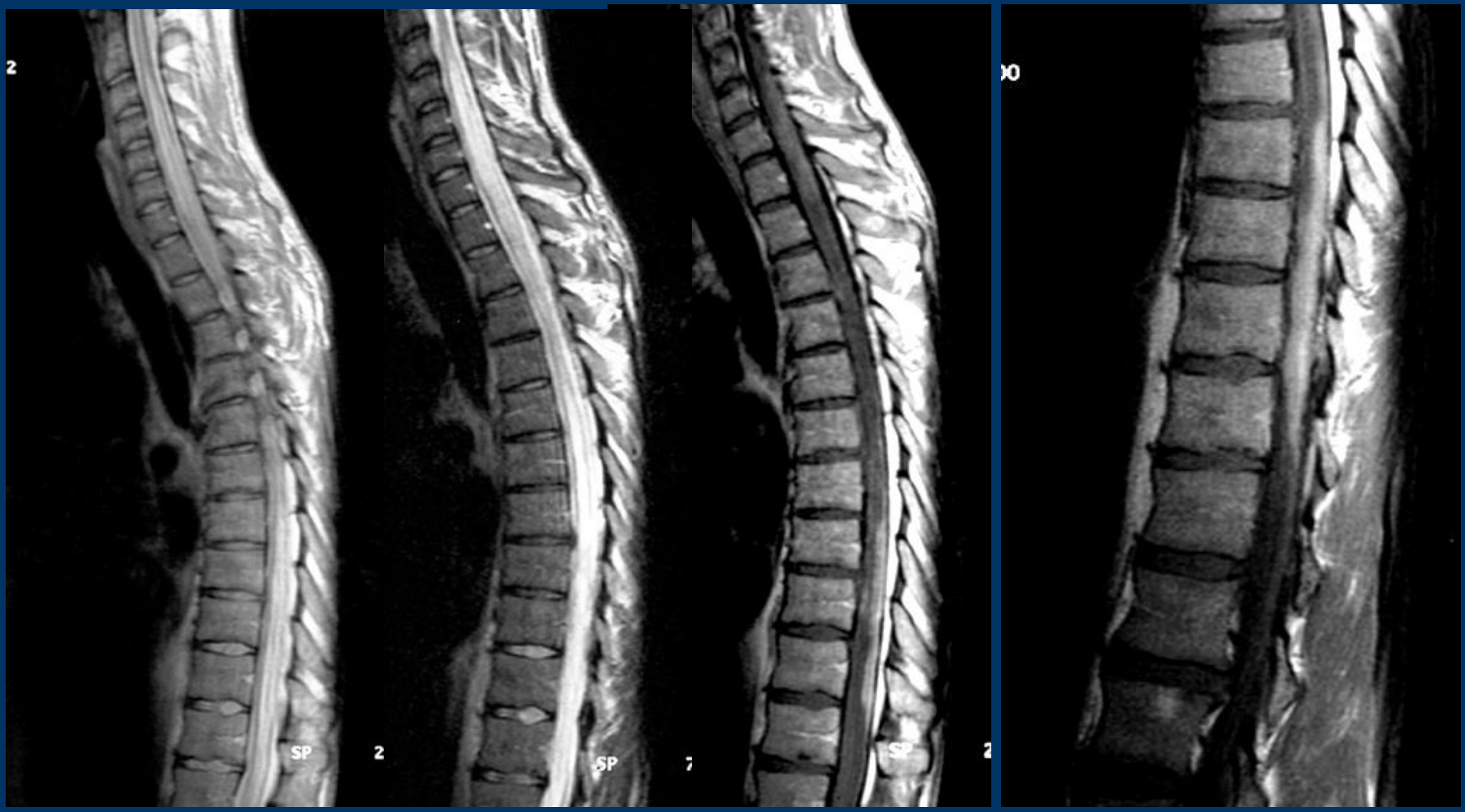
CNS angiitis: punctiform enhancement



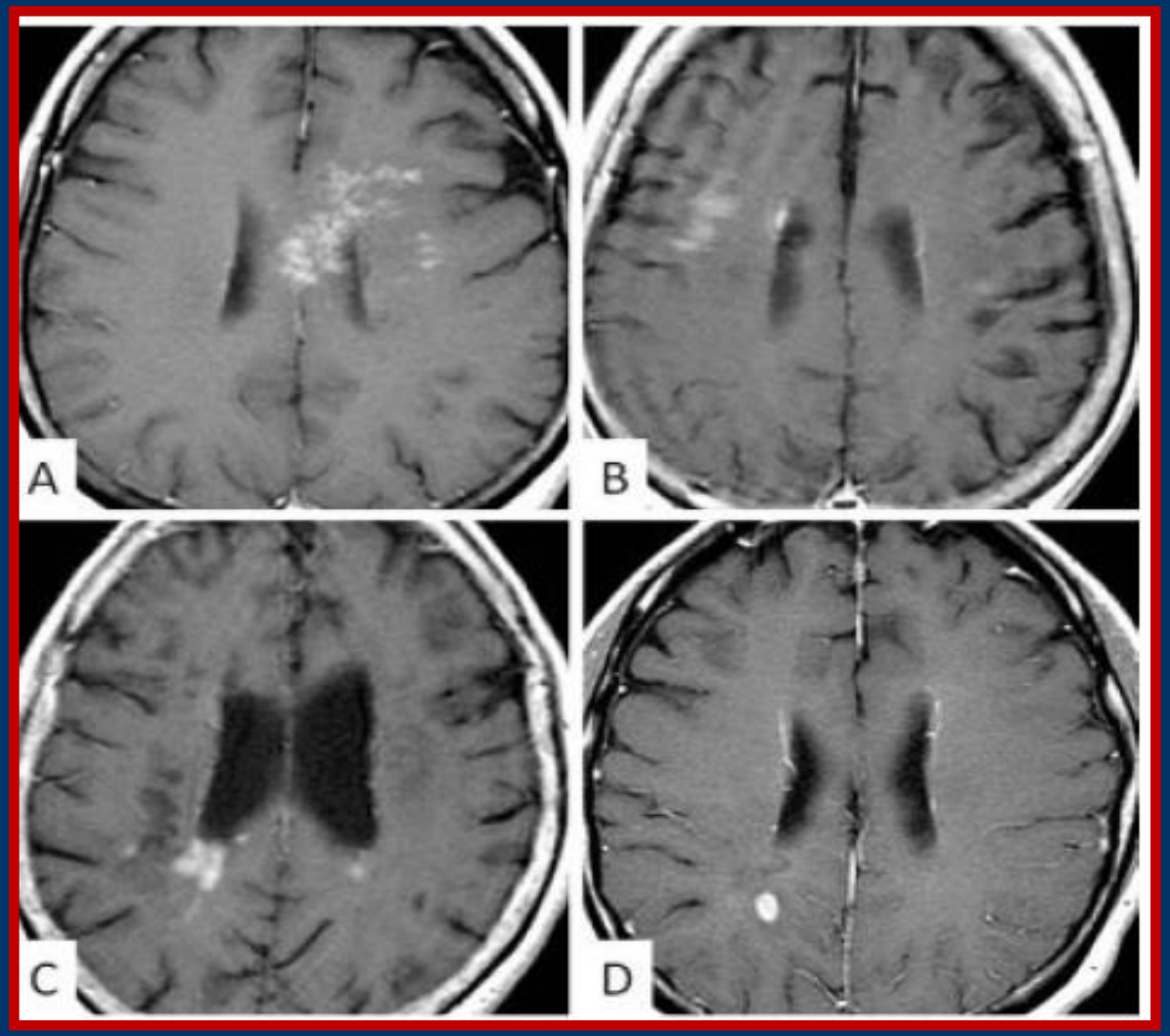
Sarcoid: meningeal enhancement



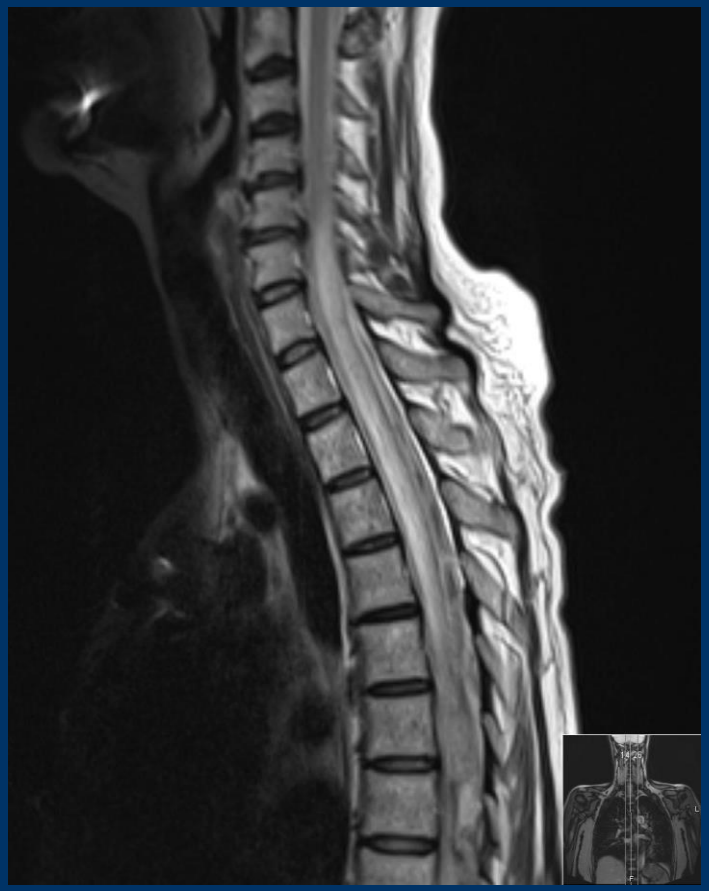
Sarcoid: persistent subpial enhancement

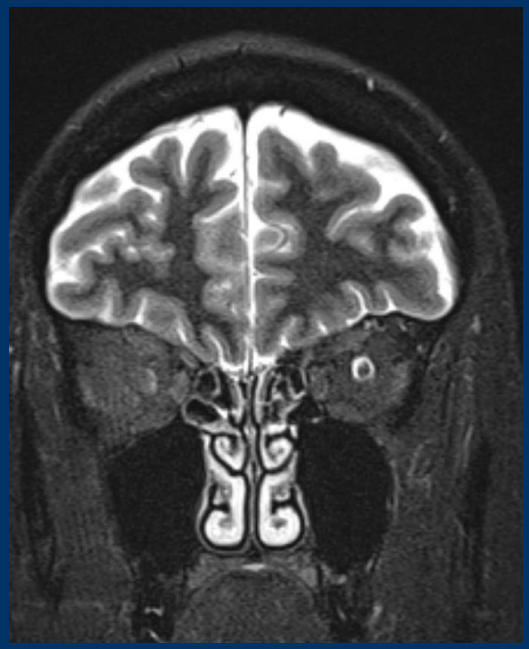


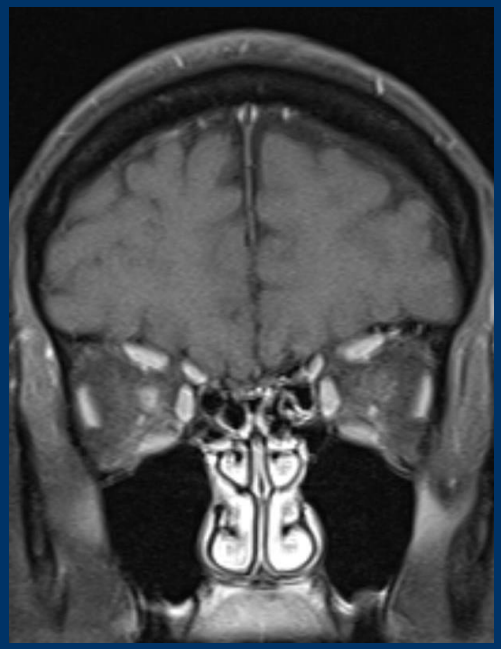
NMO – Cloud-like Enhancement



Longitudinally extensive myelitis (LETM)





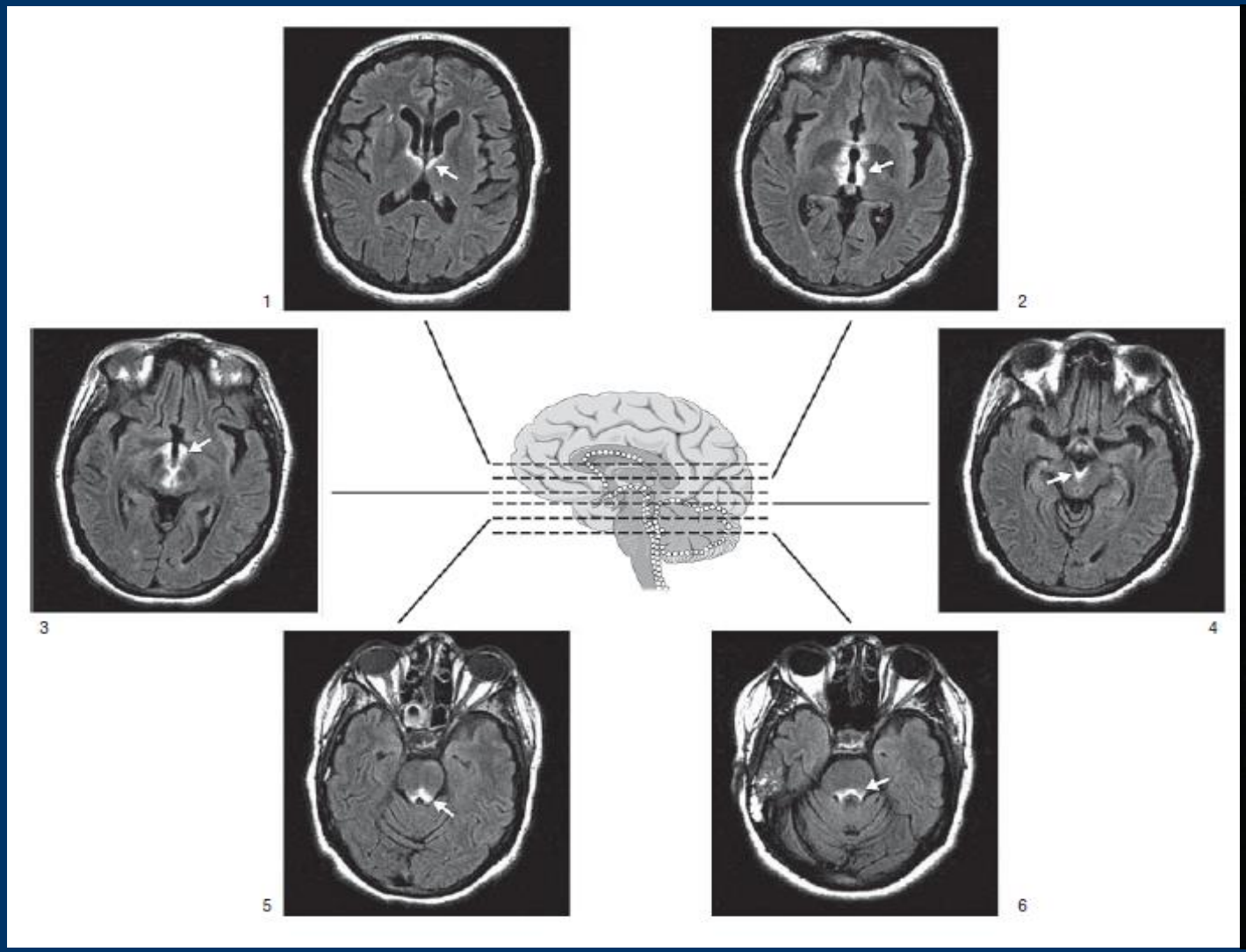


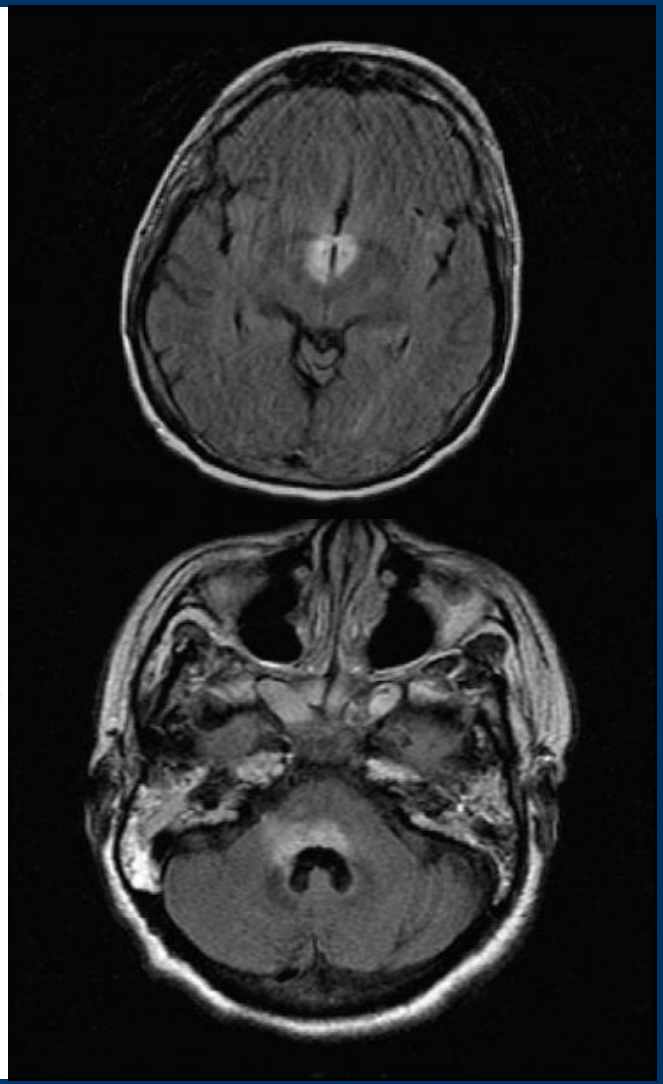
Neuromyelitis optica (NMO)
AQP4-AB titer 1:1024

Type of observation	MRI observations	Possible disorders
Spinal cord MRI		
Lesion location	Conus involvement	Anti-MOG antibody-associated transverse myelitis
	Thoracic involvement	NMOSD, HTLV1 myelopathy and arteriovenous fistulae
	Centrally symmetrically placed with grey and white matter involvement	NMOSD
	Posterior columns or spinothalamic tracts	Metabolic (for example, vitamin B_{12} and copper deficiency), infection (for example, HIV and *Treponema pallidum*), adrenoleukodystrophy and DARS-associated encephalopathy[172]
Lesion characteristics	T1 hypointensity	NMOSD
	Bright spotty lesions	NMOSD
	Patchy nodular or central canal contrast enhancement; trident sign	Neurosarcoidosis
	Pencil-like, 'snake-like' or 'owl's eye' T2 hyperintensities of the anterior horns of the grey matter on axial images associated with T2 hyperintensities of the dorsal part of the vertebrae in the affected region	Spinal cord infarction
	T2 increased perimedullary flow voids; vascular	Dural arteriovenous fistulae
	Pancake-like gadolinium enhancement or spindle-shaped lesion	Spondylotic myelopathy
	Nerve root and leptomeningeal contrast enhancement	Neurosarcoidosis and infection
	Lesion that affects three or more vertebral segments	NMOSD, ITM, ADEM, SLE, Sjögren syndrome, neuro-Behçet disease, neurosarcoidosis, spinal cord infarction, dural arteriovenous fistulae, paraneoplastic, spondylotic myelopathy and glial fibrillary acidic protein antibody disease[162]
	No lesions	Migraine, dilated Virchow–Robin spaces and SVD

Geraldes & MAGNIMS, Nat Rev Neurol 2018

NMO – lesion distribution

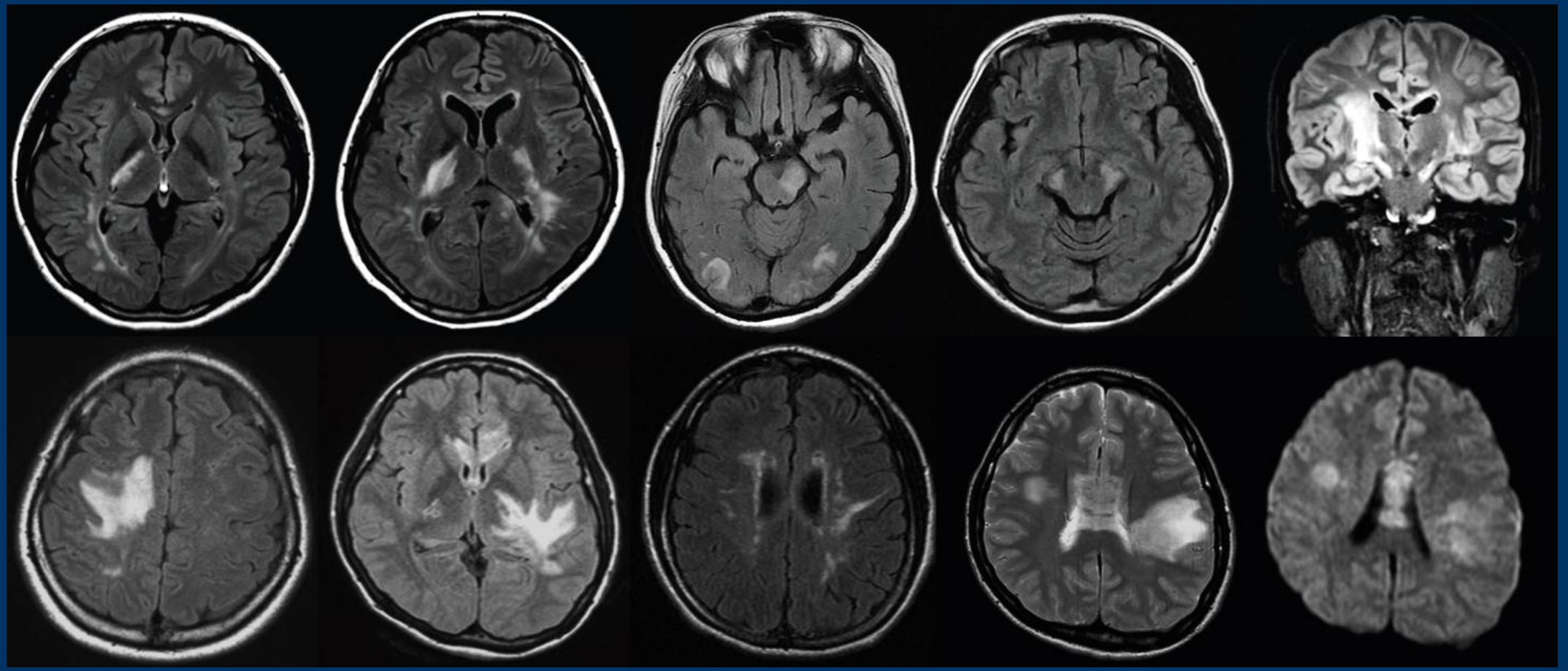




Pittock et al. Arch Neurol 2006

Wang et al. Eur J Radiol 2011

NMO – atypical brain lesions



Kim W, Mult Scler Int 2012

Table 4 | **Red-flag imaging features summarized by the iMIMICs mnemonic**

Letter	Meaning	Minimum essential MRI sequences
M	• Meningeal enhancement	2D axial or 3D contrast-enhanced T1 weighted
I	• Indistinct lesions • Increasing lesions	Sagittal 2D or 3D T2-weighted FLAIR
M	• Macrobleeds • Microbleeds	2D axial T2*-weighted gradient echo
I	• Infarcts	2D axial, 3D T1 weighted and DWI
C	• Cavities • Complete ring enhancement	2D axial or 3D contrast-enhanced T1 weighted
S	• Symmetrical lesions • Sparing of U-fibres	2D axial or coronal or 3D FLAIR
	• Siderosis	2D axial T2*-weighted gradient echo or FLAIR
	• Spinal cord extensive lesions	Sagittal dual echo (proton-density and T2-weighted) and/or fast spin echo, contrast-enhanced T1-weighted spin echo and axial 2D and/or 3D T2 and contrast-enhanced T1 weighted fast spin echo

Geraldes & MAGNIMS, Nat Rev Neurol 2018

Take home messages

- misdiagnosis not uncommon
  - clinical and radiological sources
- a priori chance of SVD extremely high
  - 3PV lesions better safeguard
  - JC/IC and SC lesions rare
- red flags on MRI - iMIMICS
  - peculiar enhancement pattern
  - no spinal cord involvement or LETM
  - large cerebral lesions

UCL

Magnims
Magnetic Resonance Imaging in Multiple Sclerosis

VU university medical center

UCL

Magnims
Magnetic Resonance Imaging in Multiple Sclerosis

VU university medical center

Key points

- MRI is crucial in the diagnosis of multiple sclerosis (MS), revealing the dissemination in space and time of white matter lesions (WMLs) and helping to rule out alternative diagnoses
- WMLs with a distribution similar to that seen in MS can occur in many disorders, from common age-related vascular disease and migraine to neuromyelitis optica spectrum disorders and rarer conditions
- The distribution of WMLs can help to differentiate MS from antibody-mediated CNS disorders
- The proportion of lesions that exhibit the central vein sign and the presence of cortical lesions can be useful in differentiating MS from some of its mimics
- Meningeal enhancement, indistinct (ill-defined) lesions that increase in size over time, macrobleeds and microbleeds, infarcts, cavities, symmetrical lesions that spare U-fibres, siderosis and extensive spinal cord lesions suggest diagnoses other than MS
- We suggest the mnemonic iMIMICs to remember the atypical MRI features that indicate a diagnosis other than MS

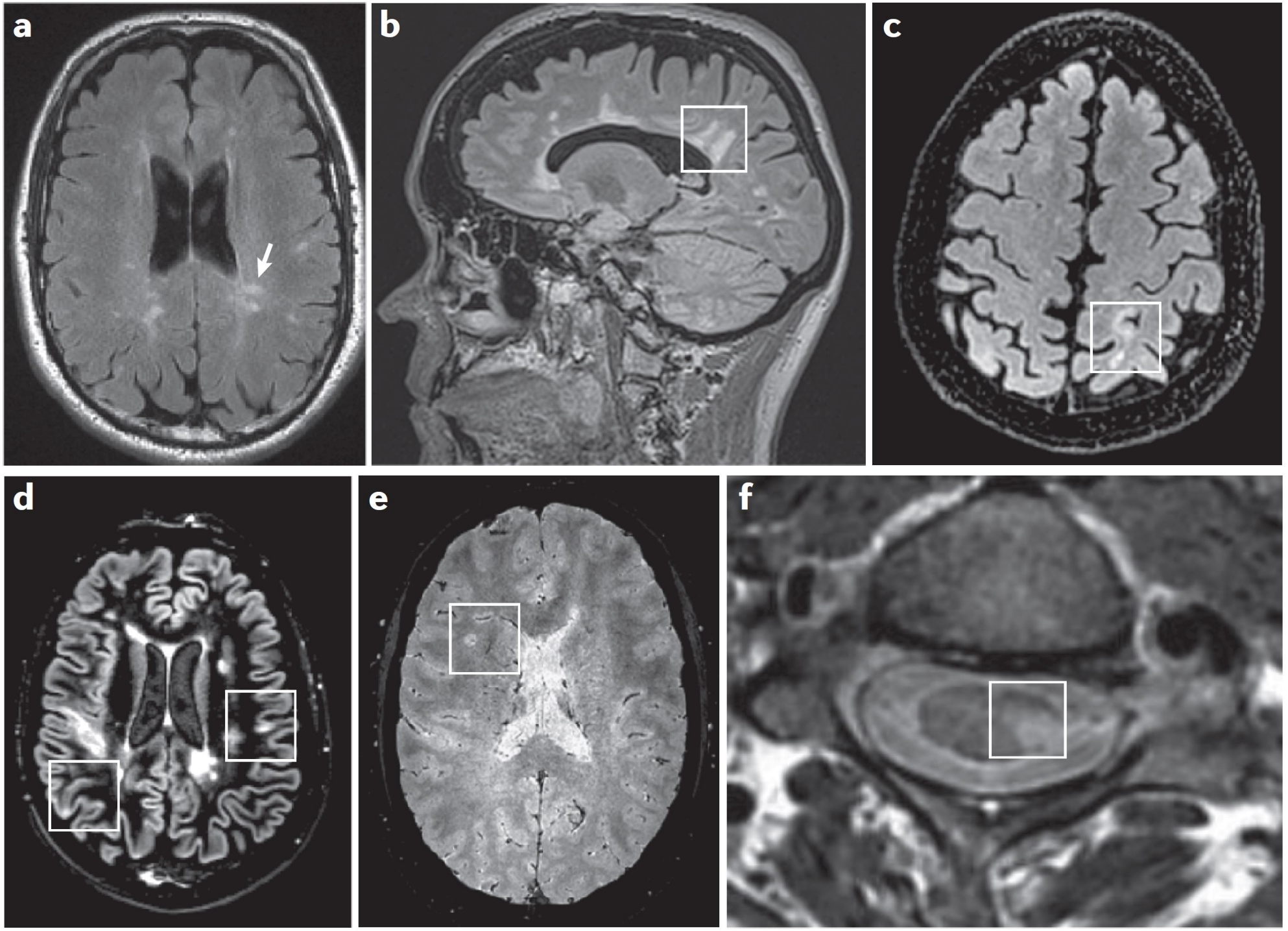

a
b
c
d
e
f

Table 13.1

Summary of magnetic resonance imaging (MRI) appearance of multiple sclerosis (MS) lesions

	MRI characteristic of MS lesions
Location	Supratentorial: juxtacortical (involvement U-fibers); periventricular (corpus callosum, trigonum, temporal horns)
	Infratentorial: fourth ventricle, cerebellar peduncles, medulla oblongata, intra-axial segment of the trigeminal nerve and the pial and ventricular surface of the pons
	Cortical lesions (3D FLAIR, DIR). Basal ganglia infrequent
Morphology	Sharp margins, oval/round, perivenular (Dawson's fingers). Bilateral, slightly asymmetric
	Later stages may converge
Signal intensity	T1: Intermediate-low
	T2: Hyperintense
	Black holes: Signal intensity lower than the gray matter on T1
Enhancement	Nodular/homogeneous or ring-like. Frequent coexistence of enhancing/non-enhancing lesions
	Tumefactive demyelinating lesions: incomplete ring (open-ring pattern)
Optic neuritis	Hyperintense on STIR. May show enhancement
Spinal cord	Frequently cervical
	Short segment (less than two vertebral segments), less than half of the diameter of the spinal cord
	Commonly peripheral in the spinal cord, most frequently lateral and dorsal white-matter columns
	May enhance (and may present focal swelling)
	In PPMS diffuse subtle high T2/PD signal and atrophy

3D FLAIR, three-dimensional fluid attenuated inversion recovery; DIR, double inversion recovery; STIR, short T1 inversion recovery; PPMS, primary progressive multiple sclerosis.

Brain Ar Click for more information
Brain Dementia
Brain Epilepsy
Brain Ischemia - Acute Stroke
Brain Ischemia - Vascular territories
Brain Tumor - Systematic Approach
Brain Venous Thrombosis
Multiple Sclerosis
Sellar and Parasellar tumors
Spine - Cervical injury
Spine - Disc Nomenclature
Spine - Lumbar Disc Herniation
Spine - Myelopathy
Spine - Thoracolumbar injury
Spine injury - TLICS Classification

Multiple Sclerosis

Frederik Barkhof, Robin Smithuis and Mari

From the MR Center for MS Research, Radiology Department of the 'Vrije Universite
Hospital, Leiderdorp, the Netherland

Publicationdat

This review is
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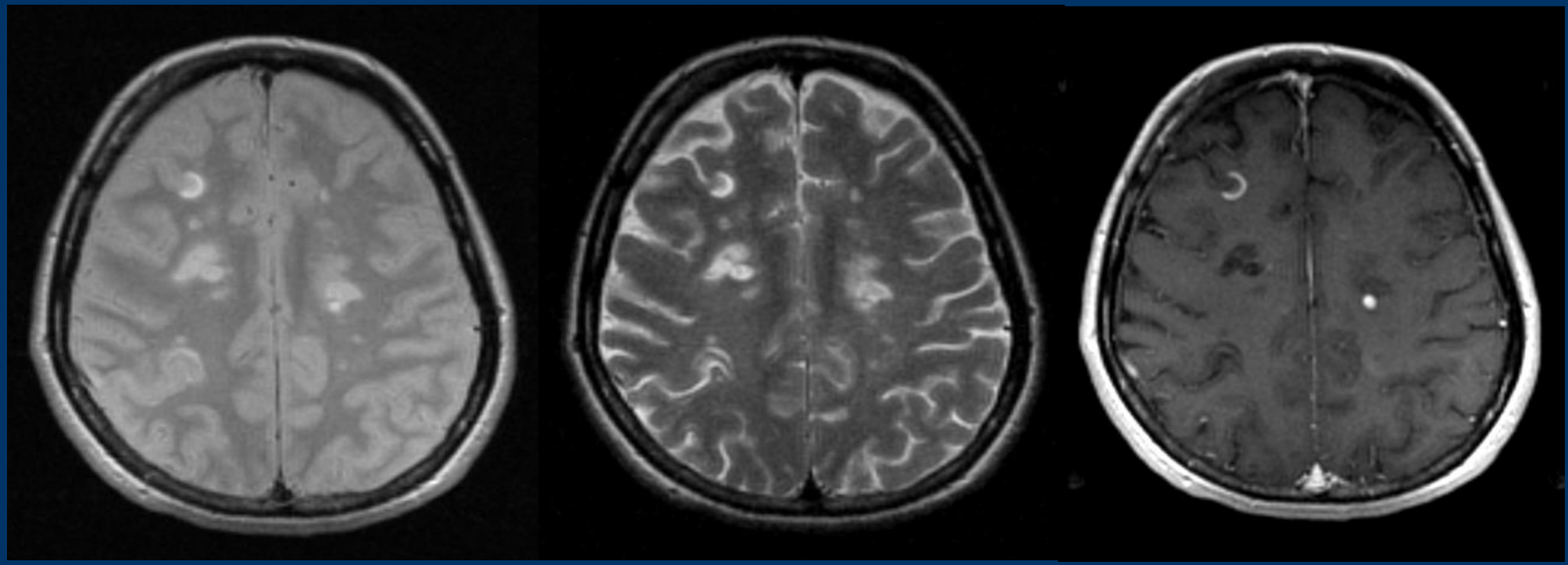
This presentat
of Multiple Scl

We will discus

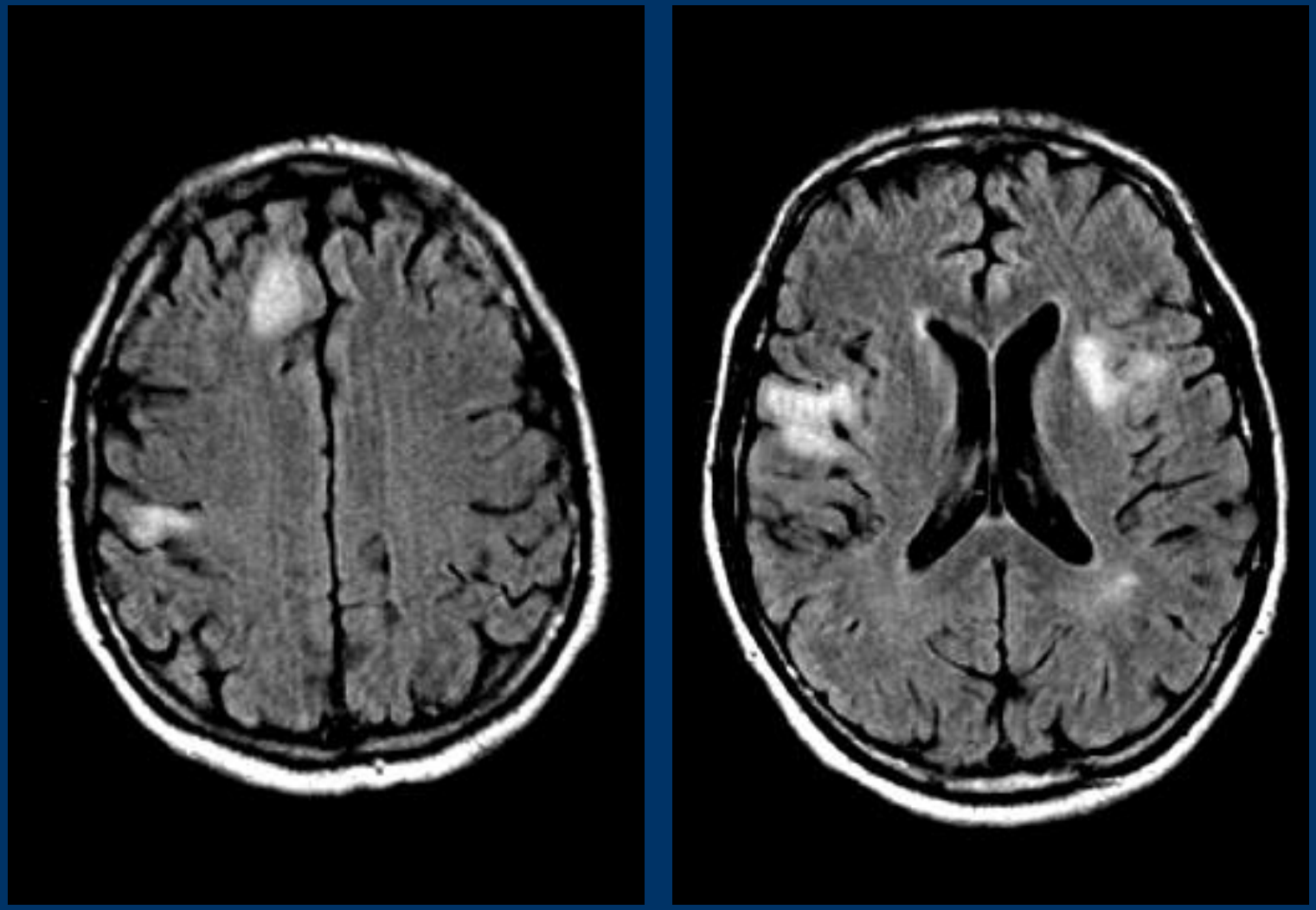
- Typical
- Role of
- How to
diseases
- The importance of the *a priori chance* for the differential diagnosis of white matter lesions.

Introduction

MS – juxtacortical inflammation



Primary CNS angiitis: FLAIR



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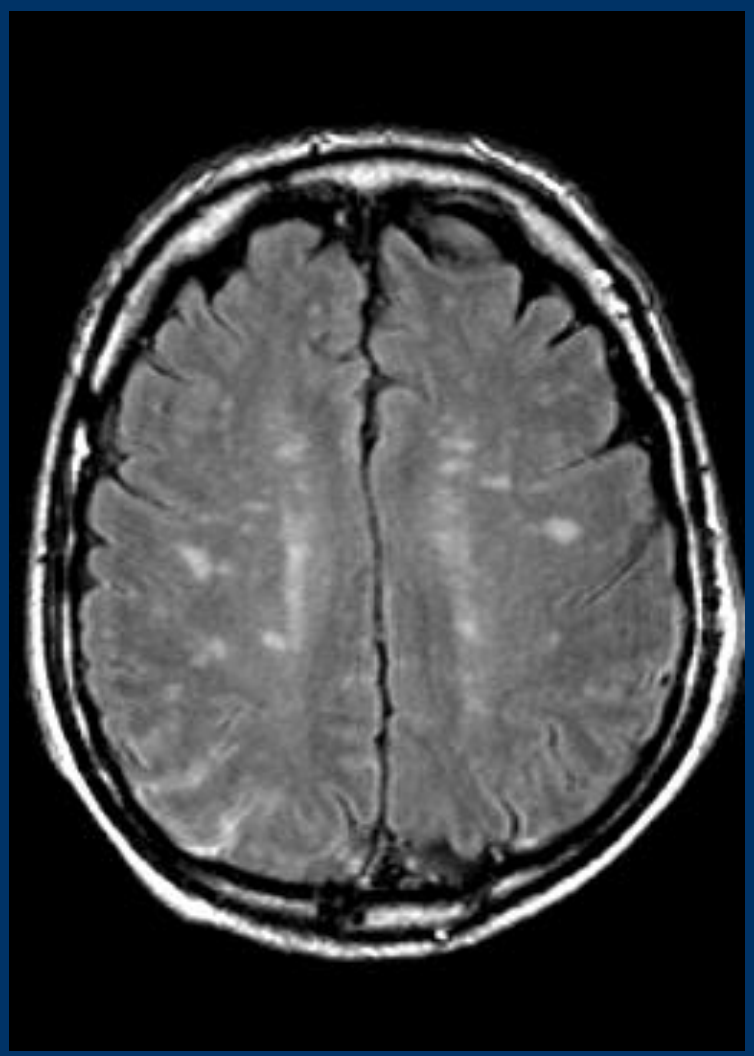
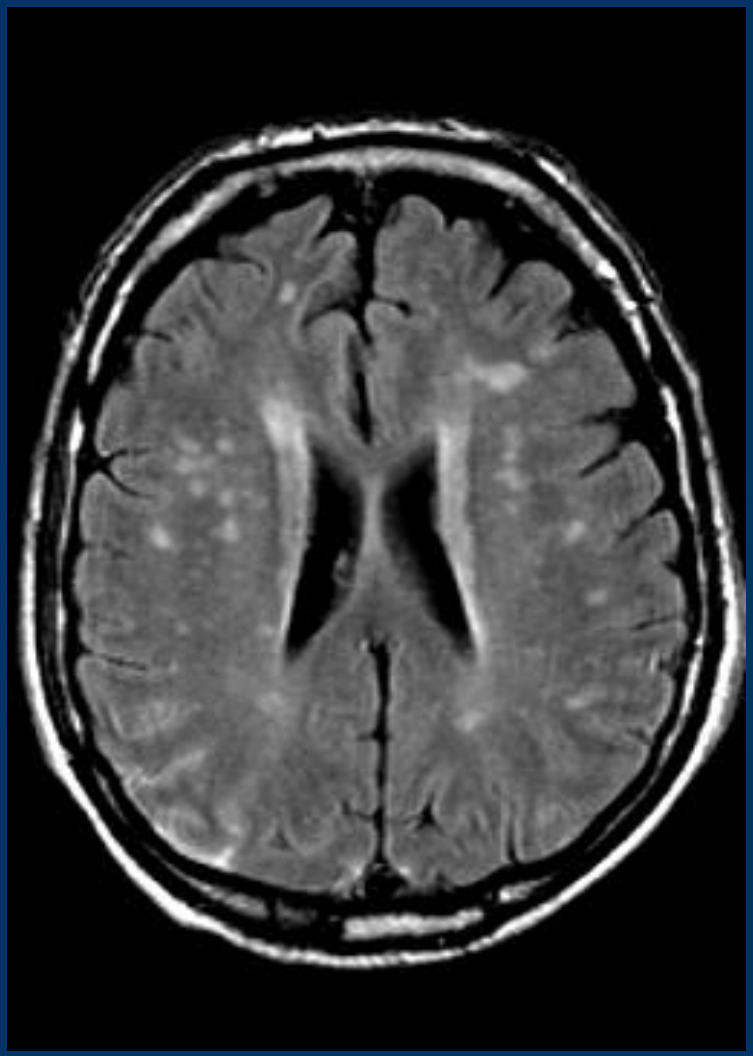
Sarcoid: T2 lesions





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