Cervical myelitis: a practical approach to its differential diagnosis on MR imaging

Zervikale Myelitis: praktische differentialdiagnostische Aspekte im MRT

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ABSTRACT

Background Differential diagnosis of non-compressive cervical myelopathy encompasses a broad spectrum of inflammatory, infectious, vascular, neoplastic, neurodegenerative, and metabolic etiologies. Although the speed of symptom onset and clinical course seem to be specific for certain neurological diseases, lesion pattern on MR imaging is a key player to confirm diagnostic considerations.

Methods The differentiation between acute complete transverse myelitis and acute partial transverse myelitis makes it possible to distinguish between certain entities, with the latter often being the onset of multiple sclerosis. Typical medullary MRI lesion patterns include a) longitudinal extensive transverse myelitis, b) short-range ovoid and peripheral lesions, c) polio-like appearance with involvement of the anterior horns, and d) granulomatous nodular enhancement prototypes.

Results and Conclusion Cerebrospinal fluid analysis, blood culture tests, and autoimmune antibody testing are crucial for the correct interpretation of imaging findings. The combination of neuroradiological features and neurological and laboratory findings including cerebrospinal fluid analysis improves diagnostic accuracy.

Key Points:
- The differentiation of medullary lesion patterns, i. e., longitudinal extensive transverse, short ovoid and peripheral, polio-like, and granulomatous nodular, facilitates the diagnosis of myelitis.
- Discrimination of acute complete and acute partial transverse myelitis makes it possible to categorize different entities, with the latter frequently being the overture of multiple sclerosis (MS).
- Neuromyelitis optica spectrum disorders (NMOSD) may start as short transverse myelitis and should not be mistaken for MS.
- The combination of imaging features and neurological and laboratory findings including cerebrospinal fluid analysis improves diagnostic accuracy.
- Additional brain imaging is mandatory in suspected demyelinating, systemic autoimmune, infectious, paraneoplastic, and metabolic diseases.

ZUSAMMENFASSUNG

Hintergrund Die Differentialdiagnose der nicht durch eine Kompression bedingten zervikalen Myelopathie umfasst ein breites Spektrum von inflammatorischen, infektiösen, vaskulären, neoplastischen, neurodegenerativen und metabolischen Ätiologien. Obschon Symptombeginn und klinischer Verlauf für einzelne neurologische Erkrankungen spezifisch erscheinen, nimmt die MRT eine Schlüsselrolle in der Bestätigung diagnostischer Überlegungen ein.

Methode Durch die Unterscheidung von akut kompletter und akut partieller transversaler Myelitis können verschiedene Entitäten differenziert werden, letztere öfter als Ouvertüre einer Multiplen Sklerose. Charakteristische medulläre Läsionsmuster sind a) die longitudinal extensive transversale Myelitis, b) kurzstreckige ovale und peripher im Rückenmarksquerschnitt gelegene Läsionen, c) polio-ähnliche Signalalterationen in den Vorderhörnern und d) granulomatösnodulär anmutende Kontrastmittelanreicherungen.

Ergebnisse und Schlussfolgerung Die Fusion von charakteristischen medullären Läsionsmustern sowie klinischen und laborchemischen Befunden einschließlich der Liquoranalyse verbessert die differentialdiagnostische Abklärung und Zuordnung zervikaler Myelitiden.

Kernaussagen:
- Die Unterscheidung verschiedener Läsionsmuster wie longitudinal extensiv transversal, kurzstreckig ovoid und peripher gelegen, polio-ähnlich und granulomatös-nodulär erleichtert die Differenzierung von Myelitiden.
- Die Differenzierung zwischen akut kompletter und akut partieller transversaler Myelitis trennt verschiedene Ätiologien, letztere oft als Ouvertüre einer multiplen Sklerose (MS).
- Die Neuromyelitis-optica-Spektrum-Erkrankung (NMOSD) kann als kurzstreckige Myelitis beginnen und darf nicht mit einer MS verwechselt werden.
- Die Fusion von neuroradiologischen, neurologischen und laborchemischen Befunden einschließlich Liquoranalyse erhöht die diagnostische Sicherheit.
- Bei Verdacht einer demyelinisierenden, systemisch autoimmunen, infektiösen, paraneoplastischen oder metabolischen Ätiologie ist eine zusätzliche zerebrale Bildgebung essentiell.

Zitierweise
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Introduction

Differential diagnosis of non-compressive cervical myelopathy includes immune-mediated inflammatory, infectious, parainfectious, vascular, neoplastic, neurodegenerative, and metabolic etiologies [1–4] (▶ **Tab. 1**). Key feature questions to reach the diagnosis in a clinical approach encompass the speed of symptom onset, e. g., acute, subacute, or chronic, and the course of the disease, i. e., monophasic, relapsing, or chronic progressive [5–7]. In addition, cerebrospinal fluid (CSF) analysis, serum inflammatory markers, infection serology and knowledge of preexisting conditions are necessary [1, 2, 4, 6].

Following the diagnostic criteria of the Transverse Myelitis Consortium Working Group, the nadir of neurologic symptoms due to acute transverse myelitis (ATM) develops within 4 hours to 21 days [8]. Evidence of inflammation is demonstrated by CSF pleocytosis or elevated immunoglobulin G (IgG) index or pathological enhancement on T1-weighted images (WI) after contrast medium administration. Distinction of acute complete transverse myelitis (ACTM) and acute partial transverse myelitis (APTM) makes it possible to categorize different entities [2, 9, 10]. In ACTM, the cord cross-section of the spinal cord is extensively or even completely affected [9]. Consequently, pronounced neurological deficits such as tetraparesis, sensory deficits, and dysfunction of the bladder and bowel are present [8–10]. Unlike ACTM, APTM shows less extensive and mainly peripheral medullary involvement and presents with mild and incomplete neurological symptoms such as dissociated sensory loss (▶ **Tab. 2**) [8–10]. Chronic inflammatory myelopathies are infrequent and progressive over several weeks or even months, e. g., primary progressive multiple sclerosis (PPMS) or paraneoplastic myelopathy [11–16].

Although symptom onset and clinical course seem to be characteristic for certain neurological diseases, additional MR imaging plays a pivotal role in narrowing down or even confirming diagnostic considerations [1, 3, 6, 16, 17]. Analysis of sagittal lesion extent along with the cross-sectional lesion pattern of the spinal cord such as involvement of grey and/or white matter, restriction to specific anatomical structures, symmetry of signal abnormal-

▶ **Tab. 1** Etiology of non-traumatic cervical myelopathy.

Extradural *Compression*	**Pathology**	**Disease**
	Neoplasm/tumor	▪ Vertebral metastasis ▪ Bone neoplasm
	Infection	▪ Spondylodiscitis
		▪ Epidural abscess
	Spinal canal stenosis	
	Medial disc herniation	
	Bleeding	▪ Epidural hematoma
		▪ Subdural hematoma
Intradural *vascular*		
	Venous congestion, infarct	Dural AV – fistula ▪ Spinal ▪ Cerebral (Cognard grade V)
	Venous congestion, bleeding	▪ Perimedullary ("true") AVM
	Superficial siderosis	▪ Infratentorial SS (type I and type II)
Compression	Neoplasm	▪ Meningioma, other
Intramedullary *vascular*		
	Bleeding	▪ AVM ▪ Cavernoma
	Venous congestion, infarct	▪ AVM
	Infarct	▪ ASA infarct, SCA infarct, other

▶ **Tab. 1** (Continuation)

Inflamma-tory/immuno-associated	Demyelination	▪ MS ▪ ADEM ▪ NMOSD ▪ MOGAD ▪ GFAP-associated disease ▪ Other
	Idiopathic	
	Systemic auto-immune disease (SAD)	▪ SLE, Behet disease, other
	Neurosarcoidosis	
	Postvaccination	
	Graft-versus-host disease	
	Parainfectious	▪ Bacterial, viral, parasitic, fungal (agent often not identified)
Inflammatory/ not auto-immu-ne-associated	Infectious acute myelopathy	▪ bacterial, viral, parasitic, fungal
	Infectious chronic myelopathy	▪ HIV, tuberculosis, syphilis, other
Metabolic diseases	SCDSC	▪ Vit. B12 -, Vit. E – deficiency ▪ Cu $^{2+}$, N_2O, Zn, other
Hereditary/ degenerative diseases	Motoneuron disease	▪ SMA, ALS, Hirayama diseases
	Spinal tract degeneration	▪ Friedreich's ataxia, (adult) PGBD, LBSL, other
Paraneoplastic		
Neoplastic	Intramedullary neoplasm	▪ Ependymoma, glioma, other
		▪ Neoplastic meningiosis
Syringomyelia		▪ (non-) communicating, e. g., Arnold-Chiari malformation

ADEM: Acute disseminated encephalomyelitis; ALS: Amyotrophic lateral sclerosis; ASA: Anterior spinal artery; AVM: Arteriovenous malformation; GFAP: Glial-fibrillary-acidic-protein-antibody-IgG-associated disease; HIV: Human Immunodeficiency Virus; LBSL: Leukoencephalopathy with brainstem and spinal cord involvement and lactate elevation; MOGAD: Myelin-Oligodentrocyte-Glycoprotein-Antibody-IgG-associated disease; MS: Multiple sclerosis; NMOSD: Neuromyelitis Optica Spectrum Disorders; PGBD: Polyglycosan body disease; SCA: Sulco-commissural artery; SCDSC: Subacute combined degeneration of the spinal cord; SMA: Spinal muscle atrophy; SS: Superficial siderosis

ities on T2 WI, and shape of contrast enhancement is crucial [1–3, 5, 6, 16, 17].

Yet, it is worth noting that despite unequivocal neurological findings and CSF changes indicative of inflammation, spine MRI may be unremarkable [18–21].

In this review, we present the most common and important non-compressive spinal cord disorders at the cervical level and an approach to differential diagnostic considerations of myelitis, not only from a radiological perspective, based on various typical lesion patterns.

Longitudinal extensive transverse myelitis (LETM)

LETM, which is typically associated with ACTM, shows sagittal extension over three or more contiguous vertebral body segments and was defined by Wingerchuk et al. [22] in 2007 in order to differentiate spinal cord involvement in neuromyelitis optica (NMO) from MS [3, 16, 22–24]. Besides immune-mediated demyelinating diseases such as neuromyelitis optica spectrum disorders (NMOSD), myelin oligodendrocyte-glycoprotein (MOG-IgG) associated disease (MOGAD) [25–31], autoimmune glial fibrillary acidic protein (GFAP-IgG) astrocytopathy [28, 32–36], and acute disseminated encephalomyelitis (ADEM) [37–39], other etiologies can also cause LETM (▶ **Tab. 3**) [2, 3, 5, 28, 40–53].

Immune-mediated diseases

Neuromyelitis Optica Spectrum Disorders (NMOSD)

Evidence of aquaporin-4 antibodies (AQP4-ab) in LETM confirms the diagnosis of NMOSD [23]. In patients with a negative or an equivocal AQP4-ab status, a specific lesion pattern on MRI and additional core criteria in neurological assessment are required to establish the diagnosis of NMOSD [23]. In NMOSD, cerebral hyperintense lesions on T2-weighted MRI sequences are frequently located in regions with high AQP4 expression, e. g., periependymal surfaces of the third and fourth ventricle. Besides involvement of the area postrema in the dorsal medulla, the brainstem and cerebellum, also diencephalic lesions, long corticospinal tract lesions and confluent subcortical or deep white matter lesions are neuroimaging characteristics in NMOSD [23, 54–56]. Moreover, optic neuritis (ON) involves extensively one- or both-sided anterior visual pathways, potentially including the optic chiasm [23, 57].

Spinal cord lesions should involve at least 70 % of the grey matter and contrast enhancement should be present (▶ **Fig. 1**) [3, 17, 23, 57, 58]. Although no specific enhancement pattern has been defined, ring-like enhancement on axial images favors the diagnosis of NMOSD [3, 6, 59]. Hypointense signal changes on T1 WI, swelling and brighter spotty lesions (BSL) are additional imaging findings [23, 42, 57, 58]. Especially on axial T2 WI, a BSL is a circumscribed extraordinarily hyperintense, i. e., brighter signal change than usual T2 hyperintense spinal cord lesions. To discri-

▶ **Tab. 2** Acute transverse myelitis: clinical and neuroradiological findings [7, 8].

	TMCWG	ACTM	APTM
Neurological findings	Sensory, motor, or autonomic dysfunction	Moderate to severe para- or tetraparesis and sensation deficits, often symmetrical, autonomic dysfunction (bladder/bowel)	Mild paresis and/or sensation deficits (uni- or bilateral)
	Bilateral signs and/or symptoms (not necessarily symmetric), clearly defined sensory level	Bilateral extensor plantar responses (Babinski sign), exaggerated tendon reflexes, often distinct bilateral sensory level	Possible extensor plantar responses (Babinski sign) and exaggerated tendon reflexes, sensory signs attributable to a sensory level or MR lesion, unilateral sensation deficits (dissociated)
CSF	Pleocytosis or elevated IgG index	Pleocytosis, OCB often negative, possibly elevated protein content	Pleocytosis (typically < 50 cells), OCB often positive, possibly elevated protein content
	and/or		
MRI	Enhancement on pc T1 WI	Often sagittal extension ≥ 3 vertebral segments (LETM)	Typically sagittal extension < 2 vertebral segments (e. g., STM, ovoid peripheral located lesions)
		Involvement of grey and white matter	Often exclusively white matter involvement
Disease course	Progression to nadir: 4 hours – 21 days after symptom onset, frequently incomplete remission	Frequently partially persisting deficits; relapse uncommon	Frequently complete remission; definite MS: ~ 30 % in 5-year follow-up
Exclusion criteria	Former radiation, vascular disease, proven infection with known agents, SAD, NMO, brain abnormalities suggestive for demyelinating disease		

Abbreviations: ACTM: Acute complete transverse myelitis; APTM: Acute partial transverse myelitis; CSF: Cerebrospinal fluid; LETM: Longitudinal extensive transverse myelitis; MS: Multiple sclerosis; NMO: Neuromyelitis optica; OCB: Oligoclonal bands; pc: Post-contrast; SAD: Systemic autoimmune disease; STM: Short transverse myelitis; WI: Weighted images

minate NMOSD from MS, BSL are a helpful imaging feature [42, 60–62]. Hypointense T1 signal changes might be the correlate of necrotizing demyelination on histological examination, reflecting the frequently complicated course in NMOSD with incomplete remission and persistent neurological deficits [17, 63].

However, in 25 out of 176 patients (~14 %) with myelitis and AQP4-ab, the longitudinal involvement was less than three vertebral body segments, i. e., short transverse myelitis (STM) (▶ **Fig. 1e–h**) [64]. NMOSD-related STM shows a central lesion pattern, hypointense signal changes on T1WI, and absence of oligoclonal bands (OCB) in CSF as compared with more peripherally located lesions in STM without AQP4-ab, e. g., in relapsing remitting MS (RRMS) [10, 17, 64–66]. After short segment involvement, however, follow-up scans over 5.4 years revealed relapses with the presence of LETM in 92 % of cases [64]. While immune-modulating therapies are recommended immediately in CIS or MS, they are potentially damaging in NMOSD [13, 64, 65]. Therefore, knowledge of this neuroradiological overture in NMOSD is crucial.

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD)

MOGAD is an autoimmune-mediated demyelinating disease positive for IgG against MOG in 40 % of AQP4-Ab-negative patients with initial suspicion of NMOSD [25–27, 67, 68]. There is strict perivenous demyelination, patients are often younger, and the disease course usually is monophasic and has a more favorable outcome [25]. Spinal cord involvement in MOGAD often manifests as LETM with predominant central location (H-sign) on T2WI (▶ **Fig. 1i–l**). Postcontrast (pc) T1WI may disclose slight or even no parenchymal contrast enhancement, whereas leptomeningeal enhancement is more frequent [3, 17, 25, 26]. Comparison of lesion evolution across MOGAD, AQP4-NMOSD, and MS shows that clinically leading MS-associated spinal lesions are preferentially located in the cervical region and to a lesser extent in the thoracic region of the spine [3, 17]. By contrast, thoracic spinal cord involvement is more common in AQP4 - NMOSD and MOGAD. In MOGAD, in particular, the conus medullaris is also frequently involved [3, 25]. In a recent study by Sechi E et al. [17], it was shown that clinical attacks occurred in 58 % of patients

▶ **Tab. 3** MRI features of myelitis.

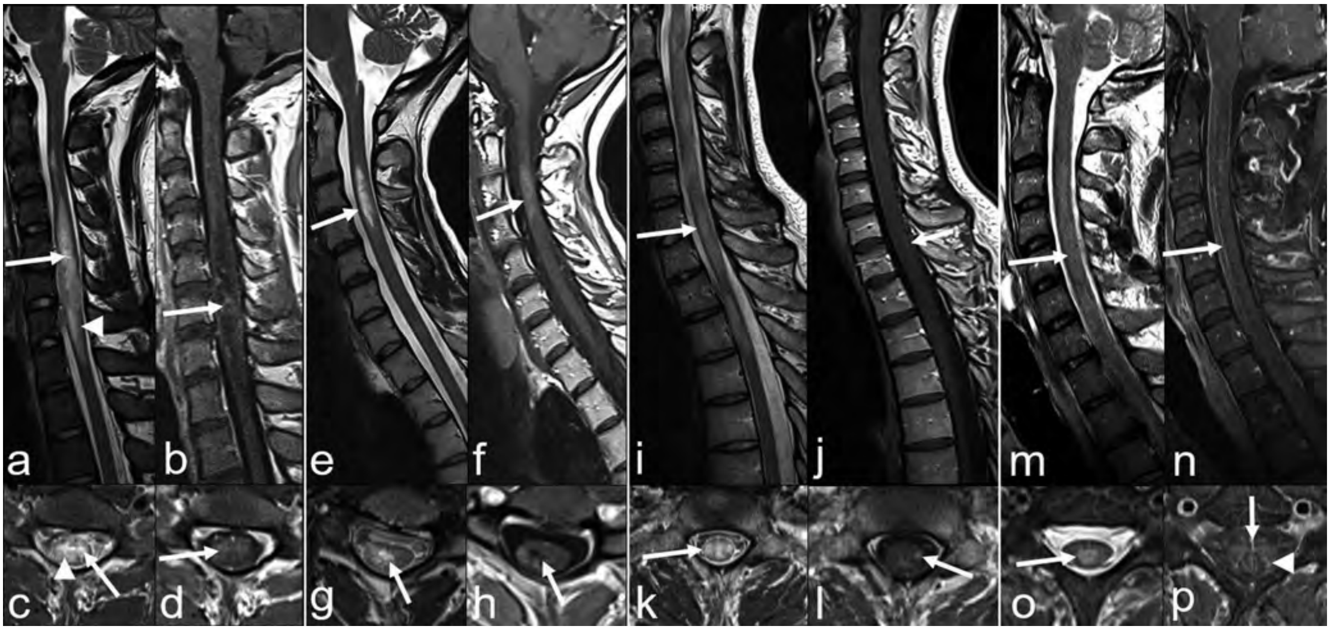
Lesion type	MRI	Etiology	Differential diagnosis
LETM	▪ ≥ 3 vertebral segments ▪ involvement of grey and white matter ▪ often central accentuation ▪ often swelling (T2 WI) ▪ partially T1 signal ↓	▪ NMOSD, MOGAD, ▪ GFAP ▪ ADEM ▪ SAD (e. g., SLE) ▪ idiopathic ▪ agent-related	vascular: ▪ infarction ▪ dural AV fistula ▪ AVM Syringomyelia Tumor metabolic, e. g. SCDSC hereditary/degen. Cord compression
	T1 WI post-contrast: ▪ trident sign ▪ pancake enhancement	 ▪ Neurosarcoidosis ▪ Neurosarcoidosis, spondylotic myelopathy	
	▪ ring enhancement	NMOSD	
	T2 WI: H-sign	MOGAD	
Oval and peripheral	▪ < 2 vertebral segments ▪ preferentially white matter ▪ interface to CSF compartment ▪ possible swelling (T2 WI) ▪ possible enhancement	CIS (dd: MS) NMOSD (14 %: STM), Neurosarcoidosis, primary Sjögren-Syndr., other	
Polio-like lesion	▪ preferentially grey matter/anterior horns ▪ often symmetric ("snake or owl eyes") ▪ rarely swelling (T2 WI) T1 WI post-contrast: ▪ Possible enhancement	Poliovirus, Picornaviridiae (e. g., Enterovirus D 68), Flaviviridae (e. g., TBEV), other	Hopkins syndrome Infarction (low flow, hemodynamic) Motoneuron disease: ▪ SMA ▪ ALS
Granulomatous	▪ Possible swelling ▪ Possible T2 signal ↓		
	T1 WI post-contrast: ▪ Possible nodular medullary enhancement ▪ leptomeningeal and radicular enhancement	Neurosarcoidosis, Tuberculosis, syphilis, parasitic infections, other	Neoplastic meningiosis

Abbreviations: ADEM: Acute disseminated encephalomyelitis; ALS: Amyotrophic lateral sclerosis; AVM: Arteriovenous malformation; CIS: Clinically isolated syndrome; GFAP: Glial-fibrillary-acidic-protein-antibody-IgG-associated disease; LETM: Longitudinal extensive transverse myelitis; MOGAD: Myelin-Oligodentrocyte-Glycoprotein-Antibody-IgG-associated disease; MS: Multiple sclerosis; NMOSD: Neuromyelitis Optica Spectrum Disorders; SCDSC: Subacute combined degeneration of the spinal cord; SMA: Spinal muscle atrophy; STM: Short transverse myelitis; TBEV: Tick-borne encephalitis virus

with MOGAD as part of an ADEM or with MRI evidence of multifocal central nervous system (CNS) lesions without encephalopathy. Myelitis with or without concomitant ON occurred in 32 %. Demyelinating spinal T2 lesions in MOGAD are more extensive compared with MS and are often initially associated with more severe deficits [3, 17, 29, 30]. However, MRI follow-up studies show complete lesion regression accompanied by an excellent clinical outcome in 79 % of cases, whereas regression of MS-associated lesions is often incomplete [17, 29, 30, 69]. AQP4 – NMOSD-related LETM is also initially associated with severe neurological deficits, but compared to MOGAD, often shows incomplete, approximately 60–80 % lesion reduction on follow-up [17, 70]. Frequent circumscribed spinal atrophy and varying degrees of neurologic residuals are likely consequences [17, 29, 63]. As opposed to MS, ON in MOGAD typically shows additional perineural enhancement on pc-T1WI besides more extensive one- or even both-sided involvement of the optic nerve [13, 57, 65].

Autoimmune glial fibrillary acidic protein (GFAP) IgG astrocytopathy

Autoimmune GFAP astrocytopathy is a corticosteroid-responsive inflammatory disorder with meningoencephalitis, with or without myelitis, associated with GFAP-IgG ab [32–36]. Clinical manifestations are heterogeneous, often with acute or subacute onset accompanied by fever, headache, encephalopathy, ataxia, abnormal vision, psychosis, autonomic dysfunction and spinal cord symptoms [33, 36]. Due to the presence of other autoimmune antibodies, diagnosis is sometimes challenging. In about 22 % of cases, a possibly causative malignancy, most often an ovarian teratoma, is present [36]. However, concomitant primary CNS lymphoma should also be considered [36, 71]. CSF analysis indicates pleocytosis, elevated protein content with OCB in 50 % of cases [26, 67]. Cranial MRI commonly shows lesions in the subcortical white matter, the basal ganglia, hypothalamus, brainstem, and cerebellum. In about 66 % of patients, contrast enhancement with a characteristic linear radial perivascular



▶ **Fig. 1** Comparison of different autoimmune mediated myelitis often manifesting as longitudinal extensive transverse myelitis (LETM). **a–d**: LETM in Neuromyelitis optica spectrum disorders (NMOSD) positive for aquaporin – 4 antibodies (AQP-4 AB) showing distinct swelling (**a, b**) and brighter spotty lesions (**a, c**: arrow) within extensive hyperintense signal changes on T2 WI (**a, c**: arrowhead) and inhomogeneous contrast enhancement (**b, d**: arrow); also note the decrease in T1 signal intensity. **e–h**: Short transverse myelitis (STM) (**e, f**: arrow) in NMOSD positive for AQP-4 AB involving especially grey matter (**g, h**: arrow) and inhomogeneous contrast enhancement (**f, h**: arrow). **i–l**: Myelin oligodendrocyte-glycoprotein (MOG-IgG) associated disease (MOGAD) revealing LETM with inhomogeneous hyperintense signal changes (**i, k**: arrow) on T2 WI, slight swelling (**i**), affecting grey and white matter (**k**) with slight patchy contrast enhancement on post-contrast (pc) T1-weighted images (WI) (**j, l**: arrow). **m–p**: Glial fibrillary acidic protein antibody (GFAP-IgG) associated LETM showing nearly homogeneous central accentuated T2 hyperintense signal changes (**m, o**: arrow) with slight contrast enhancement in the anterior fissure (**n, p**: arrow) and intramedullary (**p**: arrowhead) on pc T1 WI.

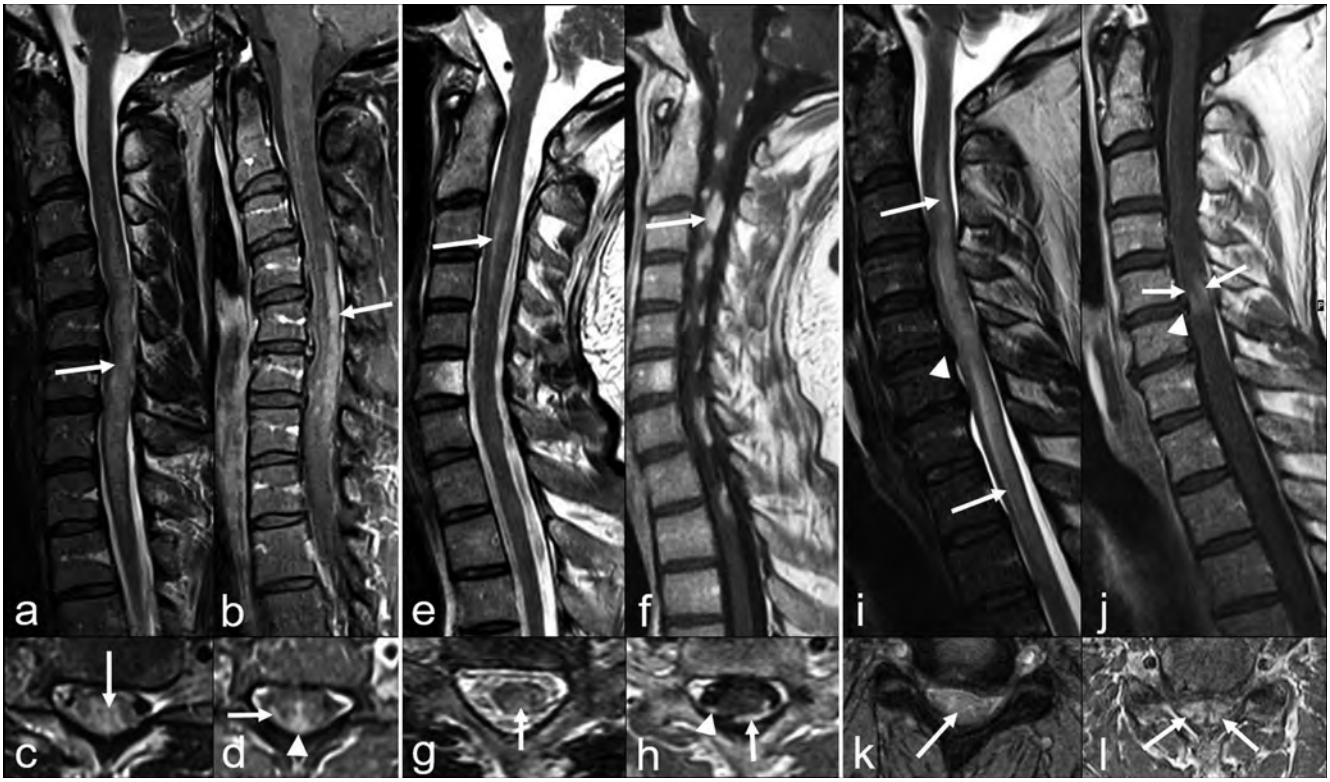
pattern perpendicular to the ventricles is seen [36]. Involvement of the spinal cord varies between 22 % and 68 % [3, 17, 31], occurs in about 11 % alone [3], and is often associated with a subacute course. MRI usually shows LETM (> 80 %) on T2 WI [17, 28] accompanied by only slight swelling with punctuate (50 %) or leptomeningeal (40 %) enhancement on pc T1 WI and possible involvement of the central canal (30 %) (▶ **Fig. 1m–p**) [17, 36, 71, 72].

Neurosarcoidosis (NS)

CNS involvement in sarcoidosis occurs in approx. 5 % of the cases and may include the brain parenchyma, meninges, cranial nerves, pituitary gland, spinal cord, and spinal nerves [73]. In NS-associated myelopathy, four MRI phenotypes can be differentiated: (a) LETM as the most frequent lesion pattern, often together with crescent-shaped dorsal subpial contrast enhancement (trident sign), (b) spinal meningitis/-radiculitis with leptomeningeal and radicular pattern of contrast enhancement, occasionally nodular, (c) short tumefactive transverse myelitis, and (d) anterior myelitis associated with disc herniation (▶ **Fig. 2**) [1–3, 6, 73, 74]. Myelopathy in NS may be challenging due to an overlap with other inflammatory spinal cord disorders, e. g., NMOSD [58]. However, subpial contrast enhancement spanning at least two vertebral body segments and persisting over months despite high-dosage corticosteroid treatment is suggestive for NS [3, 5, 58]. Especially in short tumefactive transverse myelitis, the granuloma may exhibit hypointense signal changes on T2WI with distinct enhancement on pc-T1WI and may mimic a spinal cord tumor [2, 3, 73]. Differential diagnoses of the meningoradicular MRI phenotype include bacterial infections, e. g., tuberculosis and syphilis, and parasitic infections, e. g., neurotoxocariasis, as well as meningeal carcinomatosis [1–4, 6, 16]. Moreover, cross-sectional pancake-like contrast enhancement caudal to the maximum spinal stenosis in spondylotic myelopathy may also be present in NS-associated myelitis [75].

Systemic autoimmune diseases (SAD)

In 16.5 % of cases, LETM is the start of CNS involvement in SAD [76]. Two subtypes of SLE-associated myelitis are attributed to the preferential involvement of grey and white matter, respectively [76]. (a) Predominant inclusion of grey matter typically presents with a fulminant course within 6 hours together with severe and occasionally persisting neurological deficits. Histological studies disclosed perivascular inflammation with necrosis and infarction, possibly with restricted diffusion on DWI [2, 3]. (b) Predominant inclusion of white matter causes subacute and frequently mild para- or tetraparesis and sensory deficits accompanied by bladder and bowel dysfunction. Altogether, AQP4-ab

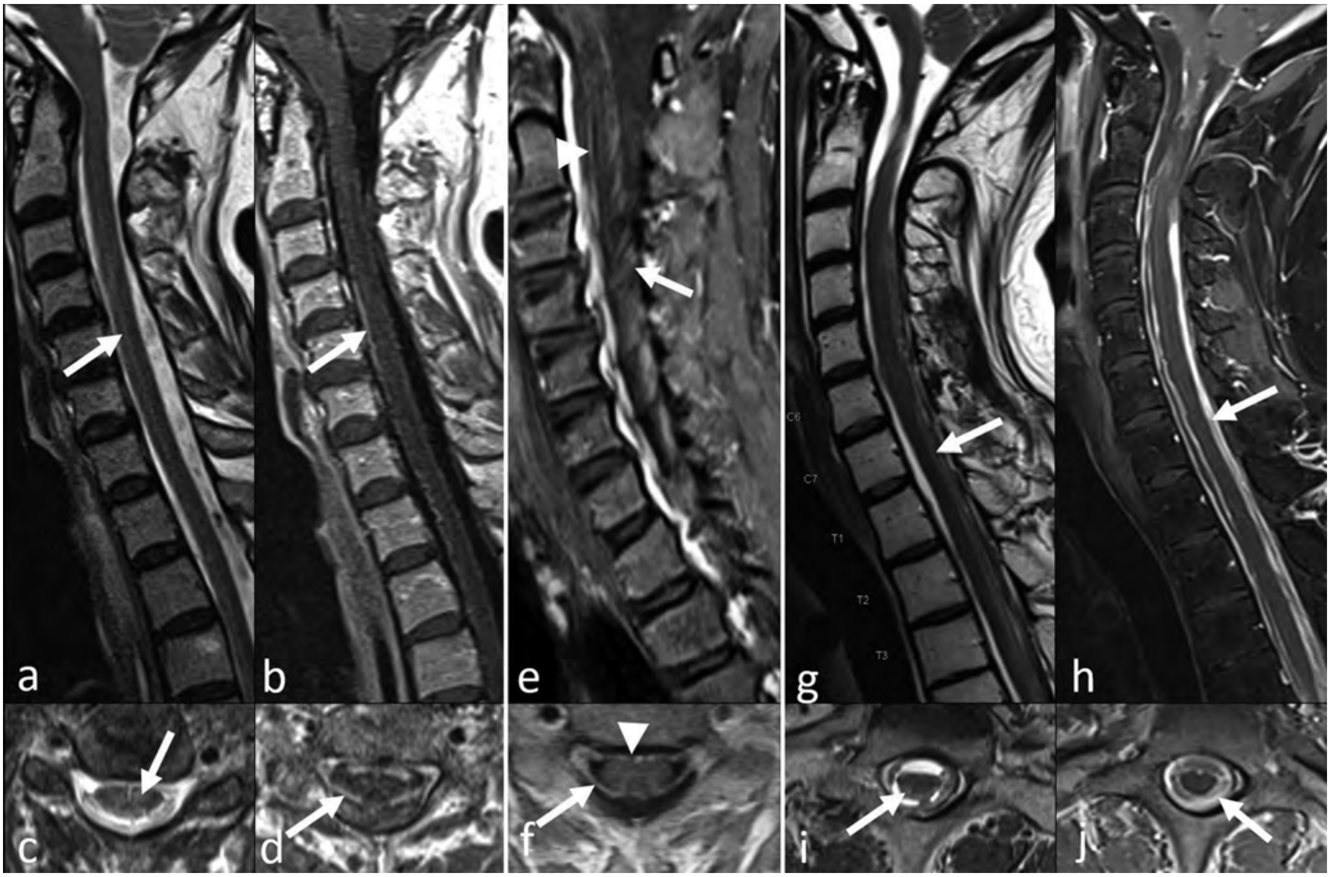


▸ **Fig. 2** MRI phenotypes in sarcoidosis-associated myelopathy. **a–d**: Longitudinal extensive transverse myelitis (LETM) spanning from C3 – Th 1/2 with hyperintense dorsal and central cord involvement on T2-weighted images (WI) (**a, c**: arrow), pronounced dorsal subpial contrast enhancement (**b, d**: arrow) and trident sign on axial T1 WI after contrast medium administration (d: arrowhead). **e–h**: Nodular leptomeningeal **(f, h**: arrow) and radicular (**h**: arrowhead) enhancement on post-contrast (pc) T1 WI; LETM showing inhomogeneous hyperintense signal changes on T2 WI with grey matter involvement (**e, g**.: arrow). **i–l**: LETM spanning from C2 – Th 2 (**i, k**: arrows) with anterior and posterior linear subpial (**j**: arrows) and intramedullary pancake-like (**l**: arrows) contrast enhancement in close proximity to degenerative cervical spine changes (**i, j**: arrowhead).

was positive in 57 % of cases and 55 % had a history of ON, highlighting the fact that the clinical symptoms of NMOSD and SAD overlap [2, 3, 23, 39]. However, it is noteworthy that repetitive spine MR imaging may be negative despite CSF evidence of inflammation, i. e., pleocytosis and raised protein level [18]. In such cases, neurological examination typically demonstrates the involvement of specific spinal cord tracts [18]. In addition, ▸ **Tab. 1** summarizes other immunological entities causing myelitis.

Infectious and parainfectious diseases

Up to 40 % of pediatric patients suffering from LETM show serological and clinical evidence of previous viral infection, whereas the rate of parainfectious etiology varies between 6 % and 45 % in adults [4, 10, 37]. Differentiation between parainfectious and infectious myelitis is challenging, and viral agents are more common than bacterial, fungal, or parasitic pathogens. Despite extensive laboratory testing, however, the causative agents often remain unidentified [28, 38, 41]. Viral infections are more frequently accompanied by an antigen-derived excessive immune reaction in the spinal cord due to molecular mimicry [37, 38]. Microbial superantigen-mediated infections can lead to a fulminant course within several hours [6, 38, 41]. Therefore, knowledge of the individual immunological state, current environment, and recent travel abroad is essential. A rare complication of bacterial meningitis is rapid progressive LETM due to spinal cord invasion of pathogens via venous pathways [77]. Other dramatic consequences are infarctions, hemorrhage, and abscess formation. Although not specific, extensive contrast enhancement of nerve roots is common in infectious meningoradiculitis (▸ **Fig. 3**) [1, 2, 4]. However, this pattern of contrast medium uptake is also seen in Guillain-Barré syndrome, meningiosis carcinomatosa, and infarction of the conus medullaris, i. e., positive anterior cauda equine sign [78]. Neuroborreliosis is a meningoradiculitis with only occasionally detectable spinal cord swelling and possible slight T2 hyperintense signal changes that do not correspond to myelitis [1, 10]. A likely explanation may be impaired venous outflow and spinal cord edema. A detailed description of the wide range of viral-, bacterial-, fungal- and parasite-induced types of ATM goes beyond the scope of this review and is reported elsewhere [1, 2, 4, 6, 79].



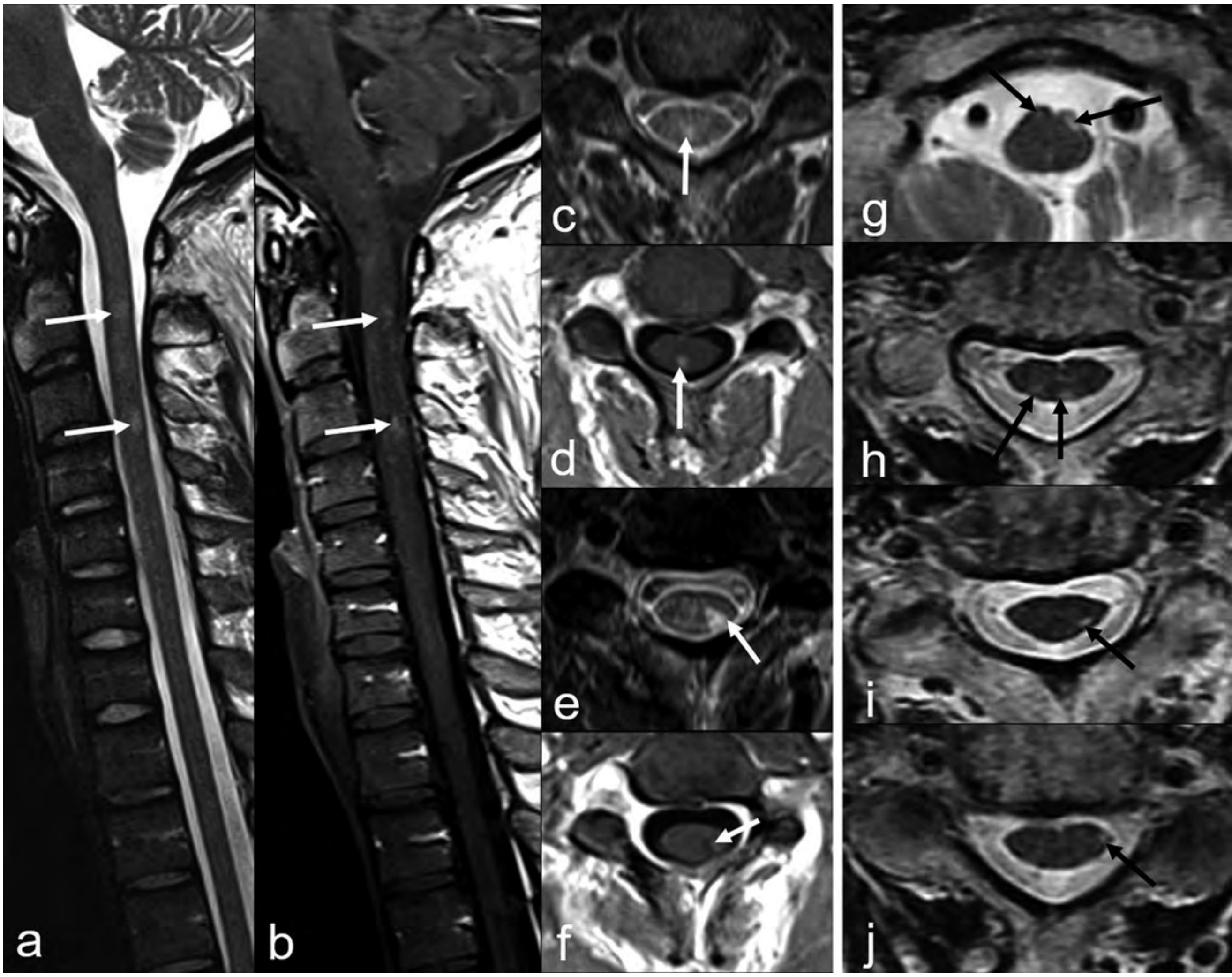
▶ **Fig. 3 a–d** Longitudinal extensive transverse myelitis (LETM) with centrally accentuated hyperintense signal changes on T2-weighted images (WI) (**a, c**: arrow) and pial and radicular contrast enhancement (**b, d**: arrow) in tuberculous meningomyeloradiculitis. **e–f**: Sagittal and axial post-contrast T1 WI showing pronounced radicular (**e, f**: arrow) and pial enhancement (**e, f**: arrowhead) in neuroborreliosis. **g–j**: Meningeosis gliomatosa in a 38-year-old man. Sagittal (**g**) and axial (**i**) T2 WI disclosing hyperintense signal changes in the dorsolateral cord circumference (**g, i**: arrow) and distinct contrast enhancement of the leptomeninges (**h, j**: arrow).

Short segment ovoid and peripherally located partial transverse myelitis

Typically, these lesions span less than two vertebral body segments and are predominately located in the white matter at the circumference with broad contact to the pial surface and subarachnoid space [1–3, 17, 65, 66, 80]. This manifestation of APTM is the prototype of spinal cord involvement in MS [65]. CSF analysis frequently shows pleocytosis and OCB. Acute lesions may show slight swelling on T2WI and eccentric or homogeneous enhancement on pc-T1WI (▶ **Fig. 4a, b**) [65, 81]. The dorsolateral circumference of the spinal cord at the cervical level is preferentially affected as a likely consequence of increased biomechanical stress due to its attachment by the denticulate ligaments and the notable mobility of the cervical spine [82]. Although the lesions are usually smaller than in myelitis caused by NMOSD, MOGAD, or GFAP, and clinical symptoms corresponding to APTM are less pronounced, they usually show only a moderate size reduction of 30–70 % on follow-up imaging [17]. In order to confirm the diagnosis of MS, additional brain MRI is mandatory in order to demonstrate dissemination in space according to the McDonald criteria [65, 66, 83].

Polio-like lesions

Besides the nearly eradicated poliovirus, other picornaviruses and some flaviviruses may cause grey matter myelitis with a frequently symmetrical lesion pattern in the anterior horns ("snake eyes", "owl eyes") causing acute flaccid paralysis [3, 10, 84]. In addition, Listeria monocytogenes may invade the CNS via nerve roots and has a similar predilection for grey matter [10]. These lesions span longitudinally over several vertebral body segments. In the acute stage, enhancement of the anterior nerve roots on pc T1WI may occur [84]. In addition, the start of spinal NMOSD may show T2 hyperintense signal changes in the anterior horns [24, 84, 85]. Due to the increased vulnerability of the motor neurons in the anterior horns with respect to oxygen reduction, especially hemodynamic infarctions with selective parenchymal necrosis may exhibit the same lesion pattern (▶ **Fig. 5**) [19, 86]. Restricted diffusion may be detectable in the acute stage followed by contrast enhancement on pc T1WI in the subacute phase [78]. As a watershed zone of the spinal cord is located in the mid-cervical region [87–89], the motor neurons of nerve roots C3-C6 are usually affected with consecutive "man-in-the-barrel syndrome" on neurological examination [90, 91]. However, neurodegenera-



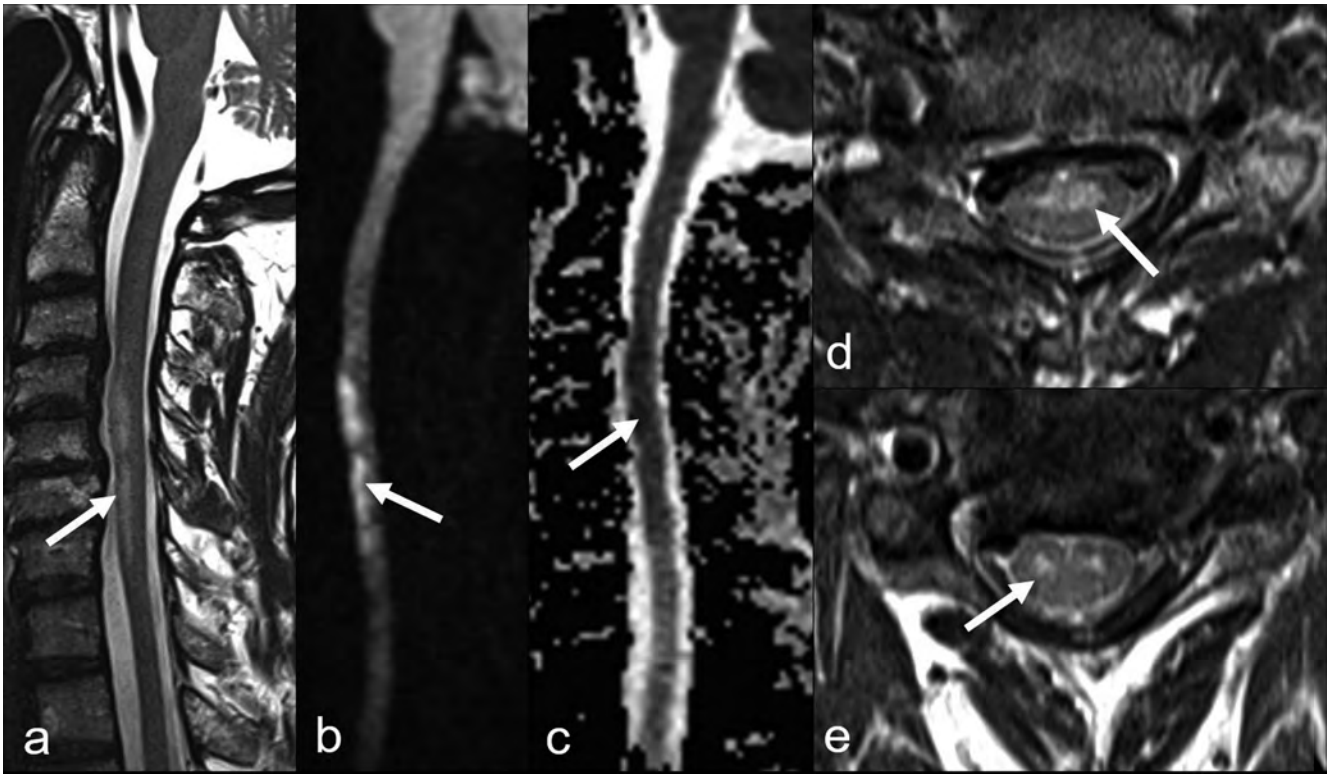
▶ **Fig. 4** Spinal cord lesions due to multiple sclerosis (MS) in relapsing remitting MS (RRMS) (**a–f**) and primary progressive MS (PPMS) (**g–j**). **a–f**: T2-weighted images (WI) disclosing two peripherally located short segment hyperintense lesions with ovoid shape (**a, c, e**: arrow) and different contrast enhancement on post-contrast T1 WI (**b, d, f**: arrow). **g–j**: multiple small hyperintense lesions (**g–j**: black arrows) on axial T2 WI predominantly at the border to the subarachnoid space.

tive diseases involving the second (lower) motor neurons, e. g., spinal muscular atrophy, amyotrophic lateral sclerosis, and atopic myelitis with focal amyotrophy and raised IgE levels (Hopkins syndrome) may also cause a similar lesion pattern on T2WI (▶ **Fig. 6**) [85, 92, 93].

Differential diagnosis of non-inflammatory myelopathies

After exclusion of extradural spinal cord compression, especially from degenerative spondylosis or metastatic vertebral tumors, the heterogeneous etiopathogenesis of non-inflammatory myelopathies includes vascular, metabolic, neoplastic and neurodegenerative diseases (▶ **Tab. 1**) (▶ **Fig. 6–8**) [1–4, 16]. The longitudinal extent on sagittal T2WI with variable hyperintense signal changes often spans several vertebral body segments [1–4, 6]. Involvement of specific spinal cord tracts, the presence of perimedullary T2 flow voids, or evidence of restricted diffusion may narrow down the differential diagnosis [1–4, 6].

Subacute combined degeneration of the spinal cord (SCDSC) depicts the alteration of the dorsal and/or lateral columns that are most commonly due to vitamin B12 deficiency (funicular myelosis) [1–3, 5, 6, 16, 94]. Other infrequent causes are deficiencies of vitamin E or copper, and exposure to nitrous oxide (N_2O) leading to vitamin B12 deficiency [94]. However, neurodegenerative diseases, e. g., Friedreich's ataxia and adult polyglycosan body disease (PGBD), paraneoplastic syndromes, and vacuolar myelopathy in people living with human immunodeficiency virus may mimic SCDSC (▶ **Tab. 1,** ▶ **Fig. 6**) [12, 14, 44, 95]. Moreover, coronavirus disease 2019 with concomitant myelopathy may also predominantly involve dorsal and lateral spinal tracts [96, 97].



▶ **Fig. 5** Anterior spinal artery syndrome. Pencil-like T2 hyperintense lesion (**a**: arrow) with restricted diffusion (**b**: diffusion-weighted imaging; b = 1000 sec/mm²; **c**: apparent diffusion coefficient map; arrow) especially in the anterior spinal cord (**a, d**: arrow); "snake-eye" lesion pattern in the anterior horns (**e**: arrow) with hyperintense T2 signal changes in the vascular border zone.

Vascular spinal cord diseases

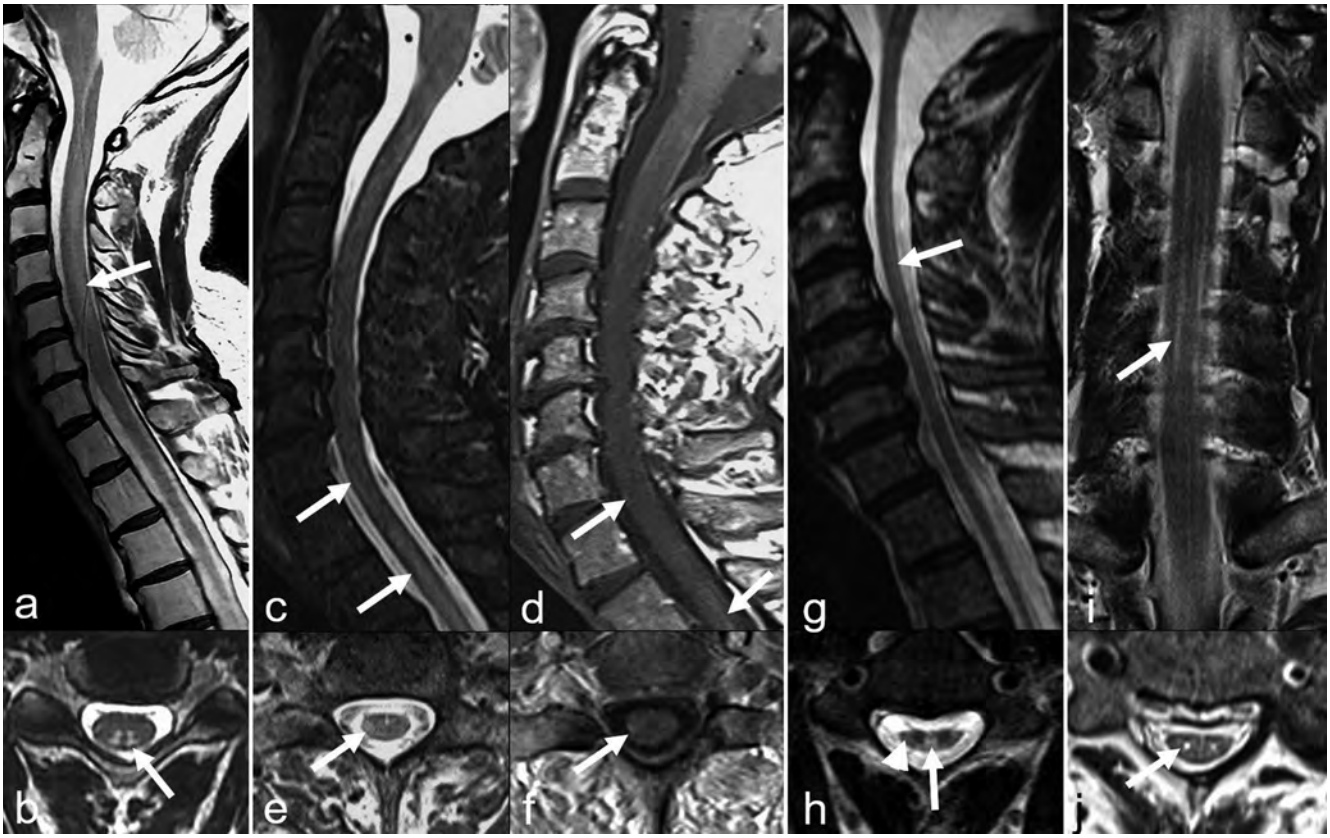
Perimedullary T2 flow voids due to enlarged veins are a diagnostic clue for the identification of spinal arteriovenous malformations (AVM) and dural AV fistulas (DAVF) [1, 2, 98–100]. Besides rarely "true" intramedullary AVMs (▶ **Fig. 8a–e**), well-treatable DAVFs also cause volume and pressure overload of the venous drainage system resulting in so-called congestive myelopathy [99, 101]. MRI shows T2 hyperintense signal changes, possible spinal cord swelling, and inhomogeneous patchy enhancement on pc T1WI [99, 101]. However, intracranial DAVFs with drainage into the perimedullary veins of the cervical spine, i. e., classified as Cognard type V, should be considered (▶ **Fig. 8f–i**) [100, 102]. Whereas the initial clinical course usually presents with slowly progressive or fluctuating symptoms, mild neurological deficits can be misleading as sudden deterioration due to concomitant ischemic aggravations may occur [5, 98, 99]. Hematomyelia and spinal subarachnoid hemorrhage may happen as a complication of "true" spinal AVM [98].

The clinical start of the anterior spinal artery syndrome (▶ **Fig. 5**) is accompanied by bilateral severe radiating pain in the shoulders and arms [6, 78]. The median time to reach the nadir of neurologic symptoms in spinal cord infarcts is 1 hour, but the mean is approximately 8 hours [6, 78, 103]. In a similar way, fulminant myelitis can cause a maximum of neurological deficits within 4 hours and therefore may mimic spinal ischemia [6, 8]. Furthermore, only 20 % of patients suffering from spinal cord infarction show intramedullary signal conversion on T2 WI within the first 15 hours, without taking DWI into account [20]. However, evidence of intramedullary diffusion restriction is not specific for spinal ischemia because it also occurs in fulminant myelitis, particularly at the start of the inflammatory process [2, 3, 21]. However, detailed description of the different types of spinal cord infarcts and their heterogeneous etiologies is beyond the scope of this review and reference is made to the relevant literature.

▶ **Fig. 9** presents an algorithm with different spinal cord lesion types, additional MRI features, medical history, and the most likely differential diagnosis.

In conclusion, differential diagnosis of non-compressive cervical myelopathy encompasses a broad spectrum of inflammatory, infectious, vascular, neoplastic, neurodegenerative, and metabolic etiologies. The combination of imaging features, clinical course, neurological and laboratory findings, and the knowledge of preexisting conditions improves diagnostic accuracy.

In myelitis, the differentiation of ACTM and APTM makes it possible to distinguish certain entities, the latter frequently being the start of MS. Characteristic lesion patterns in spinal MRI include LETM, short-range ovoid and peripheral lesions, polio-like lesions, and granulomatous nodular enhancement prototypes. However, CSF analysis, blood culture tests, and AQP4-, MOG- and GFAP-ab testing are pivotal for proper interpretation of imaging findings.



► **Fig. 6** Tract/medullary anatomical structures-associated myelopathy. **a, b**: Subacute combined degeneration of the spinal cord (SCDSC) due to vitamin B12 deficiency showing hyperintense signal changes of the dorsal columns (arrow) on T2-weighted images (WI). **c–f**: Paraneoplastic myelopathy in a 55-year-old man suffering from progressive ataxic gait and neuropathic pain. Hyperintense bilateral (r > l) signal changes of the anterolateral tracts in the lower cervical and thoracic cord (**c, e**: arrows) with slight enhancement on post-contrast T1 WI (**d, f**: arrows). **g, h**: Sagittal (**g**) and axial (**h**) T2 WI showing distinct cord atrophy and hyperintense signal changes in the dorsal (**g, h**: arrow) and anterolateral columns (**h**: arrowhead) due to adult polyglucosan body disease (PGBD). **i, j**: Spinal muscle atrophy (SMA) causing hyperintense signal changes in the anterior horns on T2 WI (**i, j**: arrow); note "snake eye" configuration (**j**).

Especially in autoimmune-associated myelitis, additional cerebral imaging is mandatory. Perimedullary flow voids are a key feature of spinal AVM, and more frequently of DAVF, which can cause varying degrees of venous congestion.

Conflict of Interest

The authors declare that they have no conflict of interest.

References

[1] Goh C, Desmond PM, Phal PM. MRI in transverse myelitis. J Magn Reson Imaging 2014; 40: 1267–1279

[2] Weidauer S, Wagner M, Nichtweiß M. Magnetic Resonance Imaging and Clinical Features in Acute and Subacute Myelopathies. Clin Neuroradiol 2017; 27: 417–433

[3] Cacciaguerra L, Sechi E, Rocca MA et al. Neuroimaging features in inflammatory myelopathies: A review. Front Neurol 2022; 13: 993645

[4] Yokota H, Tali ET. Spinal Infections. Neuroimag Clin N Am 2023; 33: 167–183

[5] Schmalstieg W, Weinshenker BG. Approach to acute or subacute myelopathy. Neurology 2010; 75: S2–S8

[6] Mariano R, Flanagan EP, Weinshenker GB et al. A practical approach to the diagnosis of spinal cord lesions. Pract Neurol 2018; 18: 187–200

[7] Eckstein C, Saidha S, Levy M. A differential diagnosis of central nervous system demyelination: beyond multiple sclerosis. J Neurol 2012; 259: 801–816

[8] The Transverse Myelitis Consortium Working Group Members. Proposed diag-nostic criteria and nosology of acute transverse myelitis. Neurology 2002; 59: 499–505

[9] Scott TF. Nosology of idiopathic transverse myelitis syndromes. Acta Neurol Scand 2007; 115: 371–376

[10] Nichtweiß M, Weidauer S. Acute transverse myelitis: Clinical features, patho-physiology, and treatment options. In: Minagar A (ed). Neuroinflammation. London, San Diego, Cambridge, Oxford: Elsevier Academic Press; 2018: 141–161

[11] Weidauer S, Raab P, Hattingen E. Diagnostic Approach in Multiple Sclerosis with MRI: an Update. Clin Imaging 2021; 78: 276–285

[12] Zalewski L, Flanagan EP. Autoimmune and Paraneoplastic Myelopathies. Semin Neurol 2018; 38: 278–289

[13] Brownlee W, Hardy TA, Fazekas F et al. Diagnosis of multiple sclerosis: progress and challenges. Lancet 2017; 389: 1336–1346

[14] Flanagan EP, McKeon A, Lennon VA et al. Paraneoplastic isolated myelopathy: clinical course and neuroimaging clues. Neurology 2011; 76: 2089–2095

▶ **Fig. 7** Intramedullary tumors and syringomyelia. **a–d**: Biopsy-proven B-cell lymphoma with longitudinal extensive T2 hyperintense lesion involving the whole cross-section (**a, c**: arrow), cord swelling, and diffuse partially homogeneous enhancement on T1-weighted images (WI) after application of contrast medium (**b, d**: arrows). **e–h**: Ependymoma with an upper solid part (**e, g**: arrow) and pronounced inhomogeneous enhancement on post-contrast T1 WI (**f, h**: arrow); additional tumor cyst (**e, f**: arrowhead) with basal signal loss due to hemosiderin deposits (**e, f**: black arrow). **i–l**: Noncommunicating syrinx (**i–l**: arrow) due to Arnold Chiari malformation type I with herniation of the cerebellar tonsils (**I, j**: arrowhead) without contrast enhancement (**j, l**). Note pulsation-related T2 signal inhomogeneities in the syrinx (**I, k**) and shifting of the central canal (**k, l**: arrowhead).

[15] Flanagan EP, Keegan BM. Paraneoplastic myelopathy. Neurol Clin 2013; 31: 307–318

[16] Trebst C, Raab P, Voss EV et al. Longitudinal extensive transverse myelitis – it's not all neuromyelitis optica. Nat Rev Neurol 2011; 7: 688–698

[17] Sechi E, Krecke KN, Messina S et al. Comparison of MRI Lesion Evolution in Different Central Nervous System Demyelinating Disorders. Neurology 2021; 97: e1097–e1109

[18] Das S, Ray BK, Chakraborty AP et al. Persistent "MRI-negative" lupus myelitis-disease presentation, immunological profile and outcome. Front Neurol 2022; 13: 968322

[19] Sechi E, Krecke KN, Pittock SJ et al. Frequency and characteristics of MRI-negative myelitis associated with MOG autoantibodies. Mult Scler 2021; 27: 303–308

[20] Holland NR. Acute myelopathy with normal imaging. J Child Neurol 2013; 28: 648–650

[21] Tanenbaum LN. Clinical applications of diffusion imaging in the spine. Magn Reson Imaging Clin N Am 2013; 21: 299–320

[22] Wingerchuk DM, Lennon VA, Lucchinetti CF et al. The spectrum of neuromyelitis optica. Lancet Neurol 2007; 6: 805–815

[23] Wingerchuk DM, Banwell B, Bennett JL et al. International Panel for NMO Diag-nosis. International consensus diagnostic criteria for neuromyelitis optica spec-trum disorders. Neurology 2015; 85: 177–189

[24] Krampla W, Aboul-Enein F, Jecel J et al. Spinal cord lesions in patients with Neu-romyelitis optica: a retrospective long-term MRI follow-up study. Eur Radiol 2009; 19: 2535–2543

[25] Dubey D, Pittock SJ, Krecke KN et al. Clinical, radiologic, and prognostic features of myelitis associated with myelin oligodendrocyte glycoprotein autoantibody. JAMA Neurol 2019; 76: 301–309

[26] Jarius S, Ruprecht K, Kleiter I et al. MOG-IgG in NMO and related disorders: a multicenter study of 50 patients. Part 2: Epidemiology, clinical presentation, radiological and laboratory features, treatment responses, and long-term outcome. J Neuroinflammation 2016; 13: 280

[27] Hegen H, Reindl M. Recent developments in MOG-IgG associated neurological disorders. Ther Adv Neurol Disord 2020; 13: 1–20

[28] Oertel F, Scheel M, Chien C et al. Differential diagnostics of autoimmune inflammatory spinal cord diseases. Nervenarzt 2021; 92: 293–306

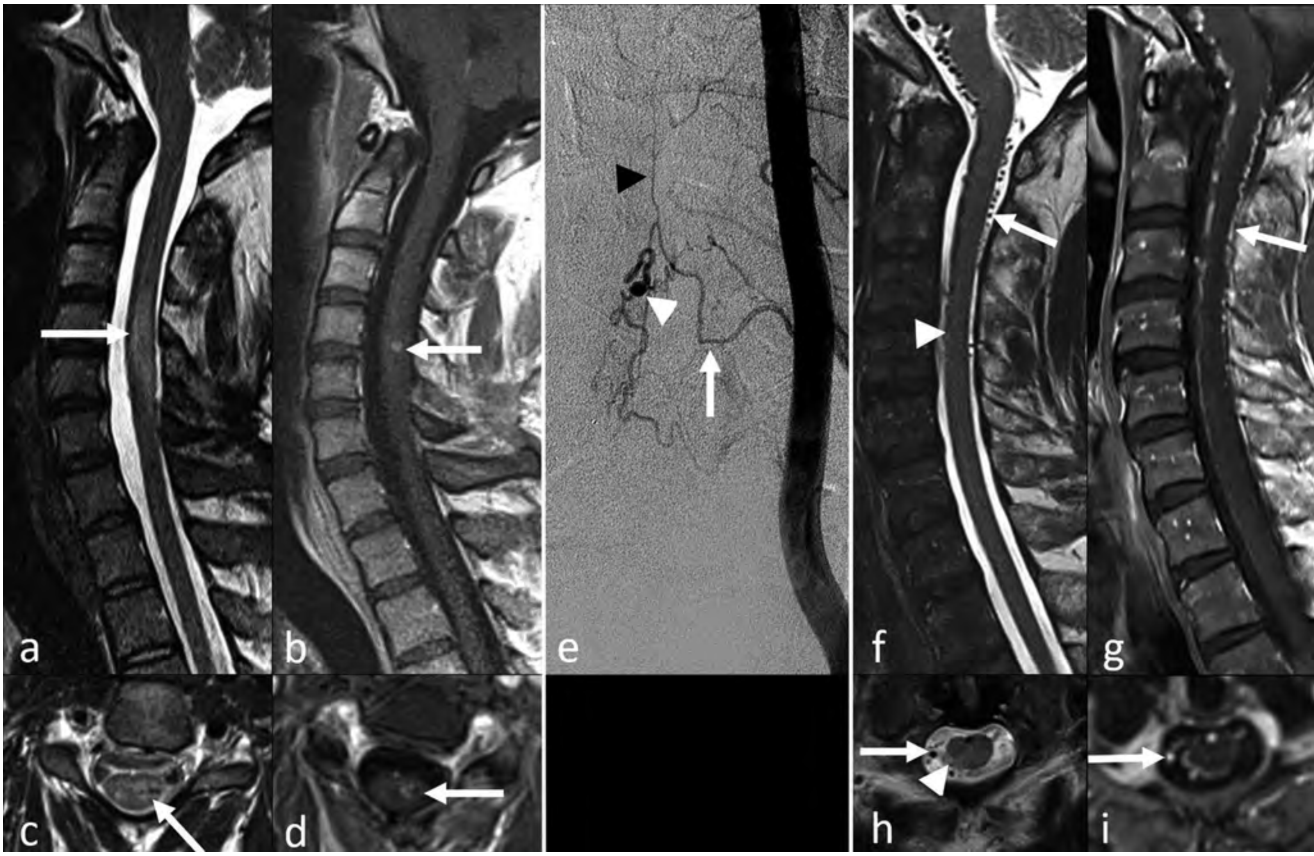
[29] Chien C, Scheel M, Schmitz-Hübsch T et al. Spinal cord lesions and atrophy in NMOSD with AQP4-IgG and MOG-IgG associated autoimmunity. Mult Scler 2019; 14: 1926–1936

[30] Denève M, Biotti D, Patsoura S et al. MRI features of demyelinating disease associated with anti-MOG antibodies in adults. J Neuroradiol 2019; 46: 312–318

[31] Reindl M, Waters P. Myelin oligodendrocyte glycoprotein antibodies in neurological disease. Nat Rev Neurol 2019; 15: 89–102

[32] Dubey D, Hinson SR, Jolliffe EA et al. Autoimmune GFAP astrocytopathy: prospective evaluation of 90 patients in 1 year. J Neuroimmunol 2018; 321: 157–163

[33] Fang B, McKeon A, Hinson SR et al. Autoimmune Glial Fibrillary Acidic Protein Astrocytopathy A Novel Meningoencephalomyelitis. JAMA Neurol 2016; 73: 1297–1307



▸ **Fig. 8 a–d** : Intramedullary arterio-venous malformation (AVM). Sagittal T2-weighted images (WI) (**a**) showing hyperintense myelopathy with slight cord swelling (**a**, arrow), nodular contrast enhancement on post-contrast (pc) T1 WI (**b, d**: arrow) and eccentric inhomogeneous signal changes on axial T2 WI (**c**: arrow). **e**: Digital subtraction angiography (DSA) of the left vertebral artery disclosed AVM with venous aneurysm (arrowhead) and prominent radiculomedullar branch C5 left (arrow); black arrowhead: anterior spinal artery. **f–i**: Intracranial dural AV fistula with additional drainage into dilated medullary veins (Cognard type V) (**f–i**: arrows) causing slight myelopathy (**f, h**: arrowhead).

[34] Flanagan EP, Hinson SR, Lennon VA et al. Glial fibrillary acidic protein immunoglobulin G as biomarker of autoimmune astrocytopathy: Analysis of 102 patients. Ann Neurol 2017; 81: 298–309

[35] Iorio R, Damato V, Evoli A et al. Clinical and immunological characteristics of the spectrum of GFAP autoimmunity: a case series of 22 patients. J Neurol Neurosurg Psychiatry 2018; 89: 138–146

[36] Shan F, Long Y, Qui W. Autoimmune Glial Fibrillary Acidic Protein Astrocytopathy: A Review of the Literature. Front Immunol 2018; 9: 2802

[37] Alper G. Acute Disseminated Encephalomyelitis. J Child Neurol 2012; 27: 1408–1425

[38] Pohl D, Alper G, Van Haren K et al. Acute disseminated encephalomyelitis. Up-dates on an inflammatory CNS syndrome. Neurology 2016; 87 (Suppl. 2): S38–S45

[39] Perez Giraldo GS, Ortiz Garcia JG. Immune-Mediated Disorders Affecting the Spinal Cord and the Spine. Curr Neurol Neurosci Rep 2021; 21: 3

[40] Garg RK, Paliwal VK. Spectrum of neurological complications following COVID 19 vaccination. Neurol Sci 2022; 43: 3–40

[41] Bennetto L, Scolding N. Inflammatory/post-infectious encephalomyelitis. J Neurol Neurosurg Psychiatry 2004; 75 (Suppl. 1): i22–i28

[42] Pekcevik Y, Mitchell CH, Mealy MA et al. Differentiating neuromyelitis optica from other causes of longitudinally extensive transverse myelitis on spinal magnetic resonance imaging. Mult Scler 2016; 22: 302–311

[43] Miller DH, Weinshenker BG, Filippi M et al. Differential diagnosis of suspected multiple sclerosis: a consensus approach. Mult Scler 2008; 14: 1157–1174

[44] McKeon A, Pittock SJ. Paraneoplastic encephalomyelopathies: pathology and mechanisms. Acta Neuropathol 2011; 122: 381–400

[45] Pourmoghaddas Z, Sadeghizadeh A, Tara SZ et al. Longitudinally extensive transverse myelitis as a sign of multisystem inflammatory syndrome following COVID-19 infection: A pediatric case report. J Neuroimmunol 2021; 360: 577704

[46] Pagenkopf C, Südmeyer M. A case of longitudinally extensive transverse myelitis following vaccination against Covid-19. J Neuroimmunol 2021; 358: 577606

[47] Sepahvand M, Yazdi N, Rohani M et al. Cervical longitudinally extensive myelitis after vaccination with inactivated virus-based COVID-19 vaccine. Radiol Case Rep 2022; 17: 303–305

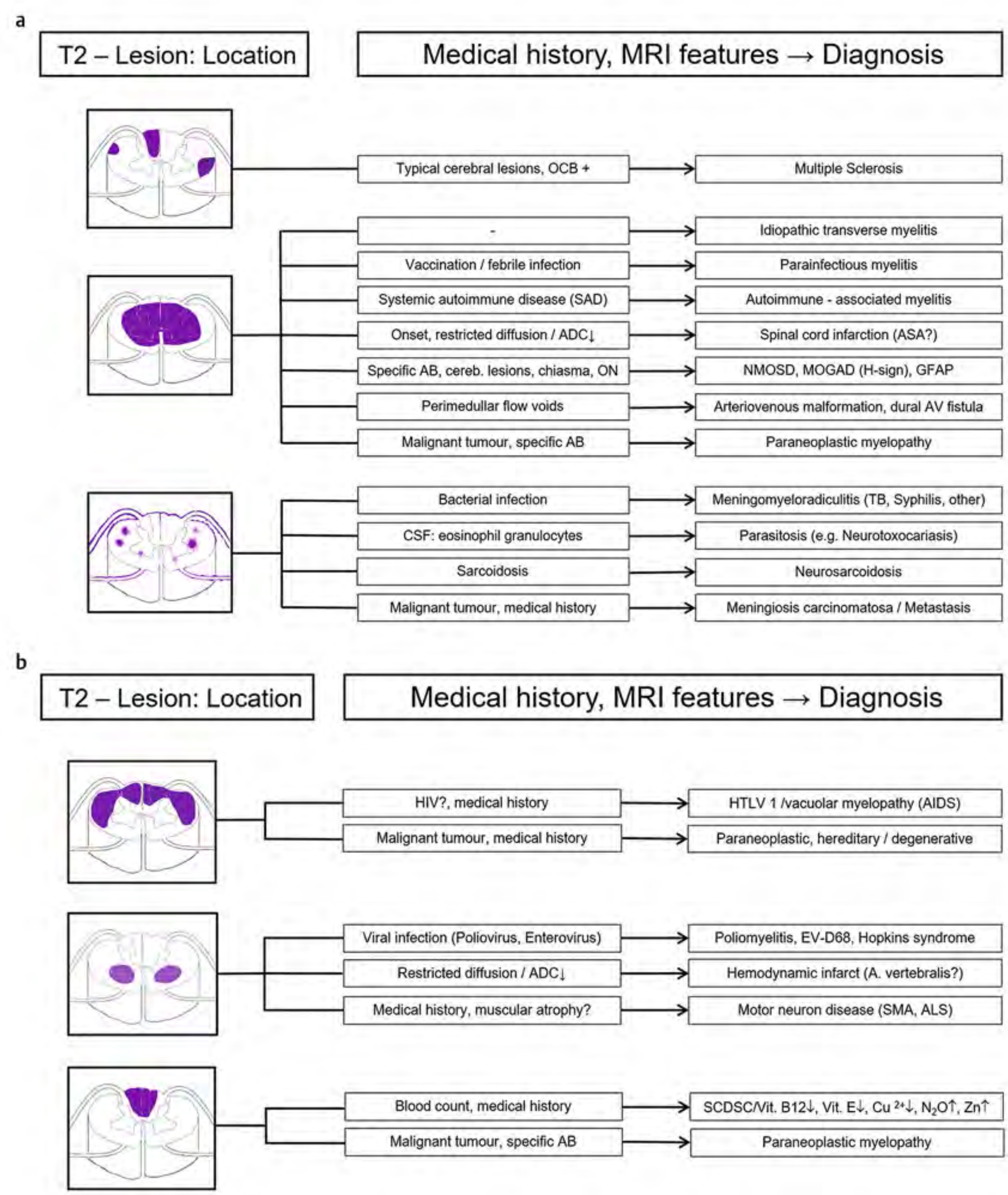
[48] Agmon-Levin N, Kivity S, Szyper-Kravitz M et al. Transverse myelitis and vaccines: a multi-analysis. Lupus 2009; 18: 1198–1204

[49] Finke C, Schmidt W, Siebert E et al. Etanercept-associated myelitis. Oxf Med Case Reports 2015; 2015 (3): 220–221

[50] Picca A, Berzero G, Bihan K et al. Longitudinally extensive myelitis associated with immune checkpoint inhibitors. Neurol Neuroimmunol Neuroinflamm 2021; 8: e967

[51] Rath JJG, Ronday HK, Wirtz PW. Acute transverse myelitis in psoriatic arthritis. J Neurol 2010; 257: 457–458

[52] Richard S, Fruchtman S, Scigliano E et al. An immunological syndrome featuring transverse myelitis, Evans syndrome and pulmonary infiltrates after unrelated bone marrow transplant in a patient with severe aplastic anemia. Bone Marrow Transplant 2000; 26: 1225–1228



▶ **Fig. 9** Algorithm of different medullary lesion types, additional MRI features, medical history, and probable differential diagnosis. Abbreviations: AB: Antibody; ADC: Apparent diffusion coefficient; AIDS: Acquired immune deficiency syndrome; ALS: Amyotrophic lateral sclerosis; ASA: Anterior spinal artery; AV: arteriovenous; CSF: Cerebrospinal fluid; EV-D68: Enterovirus D68; GFAP: Glial-fibrillary-acidic-protein-antibody-IgG-associated disease; HIV: Human Immunodeficiency Virus; HTLV 1: Human T-cell lymphotropic virus 1; MOGAD: Myelin-Oligodentrocyte-Glycoprotein-Antibody-IgG-associated disease; NMOSD: Neuromyelitis Optica Spectrum Disorders; OCB: Oligoclonal bands; ON: Optic neuritis; SCDSC: Subacute combined degeneration of the spinal cord; SMA: Spinal muscle atrophy; TB: Tuberculosis.

[53] Rodrigues CEM, de Carvalho JF. Clinical, radiologic, and therapeutic analysis of 14 patients with transverse myelitis associated with antiphospholipid syndrome: report of 4 cases and review of the literature. Semin Arthritis Rheum 2011; 40: 349–357

[54] Kitley J, Waters P, Woodhall M et al. Neuromyelitis optica spectrum disorders with aquaporin-4 and myelin-oligodendrocyte glycoprotein antibodies: a comparative study. JAMA Neurol 2014; 71: 276–283

[55] Salama S, Khan M, Shanechi A et al. MRI differences between MOG antibody disease and AQP4 NMOSD. Mult Scler 2020; 26: 1854–1865

[56] Banks SA, Morris PP, Chen JJ et al. Brainstem and cerebellar involvement in MOG IgG-associated disorder versus aquaporin-4-IgG and MS. J Neurol Neurosurg Psychiatry 2020. doi:10.1136/jnnp-2020-325121

[57] Dutra BG, da Rocha AJ, Hoffmann Nunes R et al. Neuromyelitis Optica Spec-trum Disorders: Spectrum of MR Imaging Findings and Their Differential Diagnosis. Radiographics 2018; 38: 169–193

[58] Flanagan EP, Kaufmann TJ, Krecke KN et al. Discriminating long myelitis of Neuromyelitis Optica from sarcoidosis. Ann Neurol 2016; 79: 437–447

[59] Zalewsi NL, Morris PP, Weinshenker BG et al. Ring-enhancing spinal cord le-sions in Neuromyelitis optica spectrum disorders. J Neurol Neurosurg Psychiatry 2017; 88: 218–225

[60] Hyun JW, Kim SH, Jeong ICH et al. Bright spotty lesions on the spinal cord: an additional MRI indicator of neuromyelitis optica spectrum disorder? J Neurol Neurosurg Psychiatry 2015; 86: 1280–1282

[61] Hyun JW, Lee HL, Park J et al. Brighter spotty lesions on spinal MRI help differentiate AQP4 antibody-positive NMOSD from MOGAD. Mult Scler 2022; 28: 989–992

[62] Yonezu T, Ito S, Mori M et al. "Bright spotty lesions" on spinal magnetic resonance imaging differentiate neuromyelitis optica from multiple sclerosis. Mult Scler 2014; 20: 331–337

[63] Lucchinetti CF, Guo Y, Popescu BF et al. The pathology of an autoimmune as-trocytopathy: lessons learned from neuromyelitis optica. Brain Pathology 2014; 24: 83–97

[64] Flanagan EP, Weinshenker BG, Krecke KN et al. Short myelitis lesions in aqua-porin-4-IgG-positive neuromyelitis optica spectrum disorders. JAMA Neurol 2015; 72: 81–87

[65] Thompson AJ, Banwell BL, Barkhof F et al. Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria. Lancet Neurol 2018; 17: 162–173

[66] Wattjes M, Ciccarelli O, Reich DS et al. MAGNIMS–CMSC–NAIMS consensus recommendations on the use of MRI in patients with multiple sclerosis. Lancet Neurol 2021; 20: 653–670

[67] Jarius S, Paul F, Aktas O et al. MOG encephalomyelitis: international recommendations on diagnosis and antibody testing. J Neuroinflammation 2018; 15: 134

[68] Rinaldi S, Davies A, Fehmi J et al. Overlapping central and peripheral nervous system syndromes in MOG antibody-associated disorders. Neurol Neuroimmunol Neuroinflamm 2020; 8: e924

[69] Mariano R, Messina S, Kumar K et al. Comparison of clinical outcomes of transverse myelitis among adults with myelin oligodendrocyte glycoprotein antibody vs aquaporin-4 antibody disease. JAMA Netw Open 2019; 2: e1912732

[70] Cacciaguerra L, Valsasina P, Mesaros S et al. Spinal cord atrophy in neuro-myelitis optica spectrum disorders is spatially related to cord lesions and disability. Radiology 2020; 297: 154–163

[71] Xiao J, Chen X, Shang K et al. Clinical, neuroradiological, diagnostic and prognostic profile of autoimmune glial fibrillary acidic protein astrocytopathy: A pooled analysis of 324 cases from published data and a single-center retrospective study. J Neuroimmunol 2021; 360: 577718

[72] Liu L, Fang B, Qiao Z. Clinical Manifestation, Auxiliary Examination Features, and Prognosis of GFAP Autoimmunity: A Chinese Cohort Study. Brain Sci 2022; 12: 1662

[73] Murphy OC, Salazar-Camelo A, Jimenez JA et al. Clinical and MRI phenotypes of sarcoidosisassociated myelopathy. Neurol Neuroimmunol Neuroinflammation 2020; 7: e722

[74] Zalewski NL, Krecke KN, Weinshenker BG et al. Central canal enhancement and the trident sign in spinal cord sarcoidosis. Neurology 2016; 87: 743–744

[75] Flanagan EP, Krecke KN, Marsh RW et al. Specific pattern of gadolinium en-hancement in spondylotic myelopathy. Ann Neurol 2014; 76: 54–65

[76] Birnbaum J, Petri M, Thompson R et al. Distinct subtypes of myelitis in systemic lupus erythematosus. Arthritis Rheum 2009; 60: 3378–3387

[77] Kastenbauer S, Winkler F, Fesl G et al. Acute severe spinal cord dysfunction in bacterial meningitis in adults. MRI findings suggest extensive myelitis. Arch Neurol 2001; 58: 806–810

[78] Weidauer S, Nichtweiß M, Hattingen E et al. Spinal cord ischemia: aetiology, clinical syndromes and imaging features. Neuroradiology 2015; 57: 241–257

[79] Deshayes S, Bonhomme J, de La Blanchardière A. Neurotoxocariasis: a systematic literature review. Infection 2016; 44: 565–574

[80] Padilha I, Fonseca A, Pettengill A et al. Pediatric multiple sclerosis: from clinical basis to imaging spectrum and differential diagnosis. Pediatr Radiol 2020; 50: 776–792

[81] Tartaglino LM, Friedman DP, Flanders AE et al. Multiple sclerosis in the spinal cord: MR appearance and correlation with clinical parameters. Radiology 1995; 195: 725–732

[82] Oppenheimer DR. The cervical cord in multiple sclerosis. Neuropath Appl Neurob 1978; 4: 151–162

[83] Filippi M, Preziosa P, Banwell BL et al. Assessment of lesions on magnetic resonance imaging in multiple sclerosis: practical guidelines. Brain 2019; 142: 1858–1875

[84] Maloney JA, Mirsky DM, Messacar K et al. MRI findings in children with acute flaccid paralysis and cranial nerve dysfunction occurring during the 2014 Enterovirus D68 outbreak. AJNR Am J Neuroradiol 2015; 36: 245–250

[85] Lebouteux MV, Franques J, Guillevin R et al. Revisiting the spectrum of lower motor neuron diseases with snake eyes appearance on magnetic resonance im-aging. Eur J Neurol 2014; 21: 1233–1241

[86] Gelfan S, Tarlov IM. Differential vulnerability of spinal cord structures to anoxia. J Neurophysiol 1955; 18: 170–188

[87] Martirosyan N, Feuerstein J, Theodore N et al. Blood supply and vascular reactivity of the spinal cord under normal and pathological conditions. J Neurosurg Spine 2011; 15: 238–251

[88] Turnbull IM, Brieg A, Hassler O. Blood supply of cervical spinal cord in man. A microangiographic cadaver study. J Neurosurg 1966; 24: 951–965

[89] Thron A, Stoeter P, Schiessl J et al. Development of the Arterial Supply of the Spinal Cord Tissue Based on Radioanatomical and Histological Studies in Cattle. Clin Neuroradiol 2022; 32: 325–343

[90] Urban P, Gawehn J, Ringel K. "Man-in-the-barrel" syndrome. Clin Neuroradiol 2005; 15: 190–194

[91] Berg D, Mullges W, Klotzenburg M et al. Man-in-the-barrel syndrome caused by cervical spinal cord infarction. Acta Neurol Scand 1998; 97: 417–419

[92] Kira J, Isobe N, Kawano Y et al. Atopic myelitis with focal amyotrophy: a possible link to Hopkins syndrome. J Neurol Sci 2008; 269: 143–151

[93] Osoegawa M, Ochi H, Kikuchi H et al. Eosinophilic myelitis associated with atopic diathesis: a combined neuroimaging and histopathological study. Acta Neuropathol 2003; 105: 289–295

[94] Kumar N. Metabolic and toxic myelopathies. Semin Neurol 2012; 32: 123–136

[95] van der Knaap MS, Bugiani M. Leukodystrophies: a proposed classification system based on pathological changes and pathogenetic mechanisms. Acta Neuropathol 2017; 134: 351–382

[96] Schulte EC, Hauer L, Kunz AB et al. Systematic review of cases of acute myelitis in individuals with COVID-19. Eur J Neurol 2021; 28: 3230–3244

[97] Huang HY, Shah LM, McNally JS et al. COVID-19-Associated Myelitis involving the Dorsal and Lateral White Matter Tracts: A Case Series and Review of the Literature. AJNR Am J Neuroradiol 2021; 42: 1912–1917

[98] Krings T. Vascular malformations of the spine and spinal cord. Clin Neuroradiol 2010; 20: 5–24

[99] Atkinson JL, Miller GM, Krauss WE et al. Clinical and radiographic features of dural arteriovenous fistula, a treatable cause of myelopathy. Mayo Clin Proc 2001; 76: 1120–1130

[100] Sechi E, Flanagan EP. Spinal arteriovenous fistula's often misdiagnosed as myelitis; can we stem the flow? J Neurol Sci 2020; 413: 116868

[101] Takai K, Endo T, Seki T et al. Congestive myelopathy due to craniocervical junction arteriovenous fistulas mimicking transverse myelitis: a multicenter study on 27 cases. J Neurol 2022. doi:10.1007/s00415-022-11536-7

[102] Whittam D, Huda S, Gibbons E et al. A case series of intracranial dural arterio-venous fistulae mimicking cervical myelitis: a diagnosis not to be missed. J Neurol 2021; 268: 4680–4686

[103] Nedeltchev K, Loher TJ, Stepper F et al. Long-term outcome of acute spinal cord ischemia syndrome. Stroke 2004; 35: 560–565