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Magnetic resonance imaging in myelopathy: a pictorial review

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ABSTRACT

Myelopathies have multiple causes and broad differential diagnoses, including demyelinating, metabolic, vascular and neoplastic disorders, often with distinctive imaging manifestations.

Compressive myelopathy, especially of degenerative and neoplastic origin, is the most common cause of myelopathy, followed by inflammatory disorders such as multiple sclerosis, acute disseminating encephalomyelitis, neuromyelitis optica, and transverse myelitis of other etiologies.

An accurate and early diagnosis will guide the treatment and will provide information about the prognosis of the patient. The aim of this review is to illustrate the magnetic resonance imaging features of different etiologies of myelopathy.

1. Introduction

Myelopathy is a collective term referring to any pathologic condition or neurologic deficit related to the spinal cord [1,2]. Myelopathies are a frequent and potentially disabling neurologic emergency. Compressive myelopathy, especially of degenerative and neoplastic origin, is the most common cause of myelopathy, followed by inflammatory disorders such as multiple sclerosis (MS), acute disseminating encephalomyelitis, neuromyelitis optica, and transverse myelitis (TM) of other etiologies. TM is a common presentation of myelopathy as many pathologies present with this pattern, and is divided into idiopathic versus disease-associated forms [1,3,4]. Idiopathic TM is a diagnosis of exclusion, established after ruling out any known etiology, serologic or clinical evidence of connective tissue disease, central nervous system (CNS) manifestations of infections, brain abnormalities suggestive of MS, and history of clinically apparent optic neuritis [3]. Other exclusion criteria for both idiopathic and disease-associated TM are history of spine radiation, signs of vascular malformation and of spinal cord infarction [3,5]. TM is also divided into complete TM, usually with an imaging pattern of longitudinally extensive TM, involving at least three vertebral body segments in length and all or most of the cross-section of the cord, and partial TM, which has less than two segments in length and an eccentric or asymmetric appearance on the cross-section. Distinguishing between the two patterns is helpful in the differential of the underlying etiology and to assess the outcome [3,6]. Furthermore, vascular, infectious, neoplastic and metabolic entities may mimic a primary myelopathy [5,7].

Magnetic resonance imaging (MRI) is the standard imaging technique for evaluating myelopathies and their mimics. Other techniques could also be used for the initial evaluation. Computed tomography (CT) is very useful for revealing bone abnormalities such as fractures, spondylolysis, and for postsurgical evaluation to assess the instrumentation material, the fusion and the potential complications as hematomas and abscesses. CT is the first line technique for trauma patients. CT is also very good in the depiction of ligament ossifications, spinal canal and foraminal stenoses, and spinal cord compression. CT with intravenous contrast and CT myelography could both be useful when MRI is contraindicated or not available. CT myelography is superior for identifying spinal canal or intrathecal pathology when compared to unenhanced CT or to intravenous contrast-enhanced CT. CT spinal angiography and conventional angiography are techniques used in the evaluation of vascular malformations and spinal cord infarcts. The major limitation of all these techniques as compared with MRI is that they could not analyze the intrinsic abnormalities of the spinal cord [1,8].

Important MRI sequences for the evaluation of myelopathy are T1-weighted imaging (T1WI), T2WI, proton density (PD), short-tau inversion recovery (STIR), T2*, diffusion-WI (DWI) and gadolinium-

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enhanced T1WI. The majority of processes are iso- or hypointense on T1WI, but hyperintense lesions could be revealed especially for hemorrhagic components, fat or melanin. T2WI, PD and STIR usually show hyperintense signal in myelopathies. PD and STIR have higher sensitivity, and particularly in entities such as MS, they could detect significantly more lesions than T2WI. T2* sequences could reveal hemorrhagic components in trauma patients, in underlying tumors and vascular malformations. Myelographic sequences (volumic T2WI) could show cord and radicular compression. DWI could demonstrate diffusion restriction in spinal cord ischemia and abscesses (eg. epidural). As opposed to the brain, fluid attenuating inversion recovery (FLAIR) is not used in spinal cord imaging, being less sensitive due to cerebrospinal fluid pulsation artifact [1,9,10]. Gadolinium-enhanced T1WI could depict variable enhancement patterns (none, diffuse, patchy, peripheral) in inflammatory etiologies, and could give essential information in neoplasias, granulomatous diseases and vascular malformations [10–12]. MRI angiography (MRA), currently fast 3D MRA with contrast enhancement and 4D time-resolved MRA, could be useful in vascular malformations, to demonstrate abnormal vasculature and to guide spinal arteriography [13–15]. Advanced MRI techniques as diffusion tensor imaging, magnetization transfer, myelin water fraction, MRI spectroscopy and functional MRI, are usually research topics [16].

The aim of this review is to illustrate the MRI features of different etiologies and mimics of myelopathy.

2. Discussion

Table 1 enlists different broad categories and subcategories of myelopathy.

2.1. Multiple sclerosis

MS is the leading non-traumatic cause of neurological disability in young adults. The course is typically multiphasic. Various diagnostic criteria have been proposed, the latest version being 2017 McDonald's criteria [17]. These criteria give a central role to the MRI and require two conditions: dissemination in space and in time. Dissemination in space means at least 1 lesion in at least 2 locations of periventricular, cortical/juxtacortical, infratentorial, and spinal cord. Dissemination in time can be established either on a follow-up MRI, if new lesions occur, or on a single MRI, if there are both enhancing and nonenhancing lesions. Spinal cord lesions occur in 30%–40% of clinically isolated syndrome, and in up to 90% of definite MS. The cervical cord is most frequently involved. The lesions are usually focal, well defined, often peripheral in the posterior and lateral cord (Fig. 1) – where the white matter is located. Active lesions may enhance, but less frequently than brain lesions. Spinal cord swelling and a pattern of TM could be rarely observed [18].

2.2. Neuromyelitis optica (NMO) spectrum disorder (NMOSD)

Up to 90% of cases of NMOSD occur in women and have a multiphasic evolution. The classic triad consists of IgG-antibodies against Aquaporin-4 water-channels (AQP4), optic neuritis, and transverse myelitis (Fig. 2). The NMOSD-AQP4 subgroup includes patients with positive AQP4 and at least one clinical manifestation, while the NMOSD without AQP4 subgroup includes patients with negative/unknown AQP4 status fulfilling the dissemination in space criteria and presenting with a typical MRI pattern. Dissemination in space in NMOSD is defined as two or more different core clinical characteristics such as optic neuritis, acute myelitis, area postrema syndrome, acute brainstem syndrome, symptomatic narcolepsy or acute diencephalic clinical syndrome with NMOSD-typical diencephalic lesions on MRI, and symptomatic cerebral syndrome with NMOSD-typical brain lesions. Imaging features of NMOSD myelitis include longitudinally extensive TM, swelling and associated patchy central enhancement in the acute phase

Table 1
Etiologies of myelopathy [2,4,6].

Category	Subcategory
Compressive	Degenerative
	Neoplasias
	Collections (e.g. epidural abscess or hematoma)
	Rheumatoid arthritis
	Ankylosing spondylitis
Inflammatory	Idiopathic Transverse Myelitis
	Multiple Sclerosis
	Neuromyelitis Optica
	Acute disseminated encephalomyelitis
	Connective tissue diseases
	• Systemic lupus eythematosus
	• Sjogren's syndrome
	• Behcet's disease
	• Scleroderma/systemic sclerosis
	• Mixed connective tissue disease
	• Periarteritis nodosa
	• Primary biliary sclerosis
	Vasculitis
	Neurosarcoidosis
	Paraneoplastic Myelopathy
Infectious	Viral (Herpes, HIV, HTLV, CMV, Polio, …)
	Tuberculosis
	Syphilis
	Lyme
	Other
Toxic-metabolic	Cobalamin (B12 vitamin) or folate deficiency
	Copper deficiency
	Nitric oxide (N2O) toxicity
	Radiation myelopathy
	Other
Vascular	Cord ischemia
	Vascular malformations
	• Spinal dural arteriovenous fistula
	• Spinal arteriovenous malformation
	• Cavernoma
Congenital and developmental defects	Spina bifida
	Syringomyelia
Genetic diseases	Adrenoleukodystrophy
	Adrenomyeloneuropathy
	Other

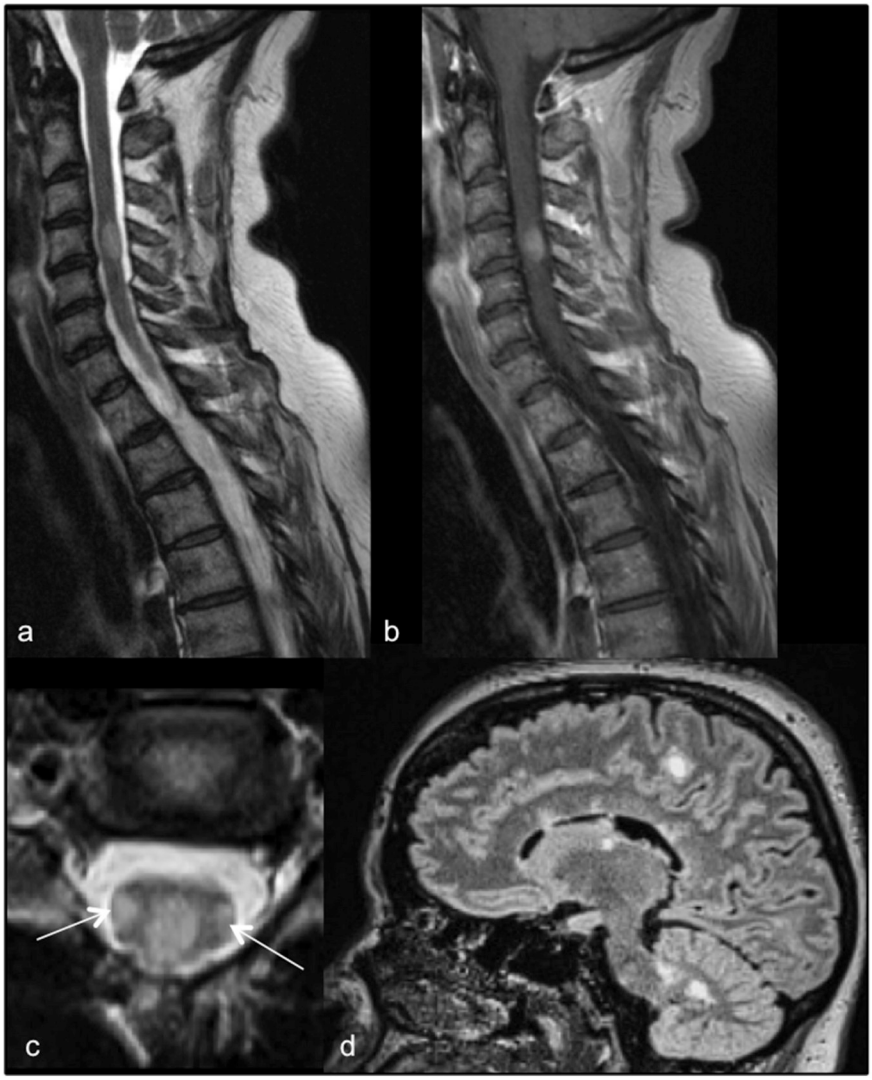

Fig. 1. Multiple sclerosis. Sagittal T2-weighted imaging (WI) and contrast-enhanced T1WI (a,b) show a short-segment T2 hyperintense lesion (a) of posterior distribution at the C4 level, with contrast enhancement (b). Smaller peripheral lesions are observed on axial T2WI (c, arrows). Sagittal FLAIR of the brain shows multiple hyperintense lesions involving the corpus callosum, the periventricular white matter and the infratentorial structures (d).

[19,20]. Some patients with NMOSD have antibodies to MOG (myelin oligodendrocyte glycoprotein). MOG positivity has a better prognosis, fewer recurrences, better response to therapy, and less long-term disability [21].

2.3. Acute disseminating encephalomyelitis (ADEM)

ADEM is an immune-mediated inflammatory disorder occurring in children and young adults in the setting of immune stimulation by a viral infection or vaccination. Imaging manifestations include large, bilateral and asymmetric lesions; involvement of basal ganglia is frequent, and represents a key-differentiating feature from MS. TM has been reported in up to one third of patients (Fig. 3). The course is classically monophasic (90% of cases), therefore MRI follow-up helps support the diagnosis, usually showing remission in ADEM, unlike MS or NMOSD [22].

2.4. Lupus myelitis

Myelitis represents a rare but severe complication of systemic lupus erythematosus (SLE). It occurs in 1–5% of SLE patients - typically in women of 30 to 40-year-old (90%) - and tends to occur as an early

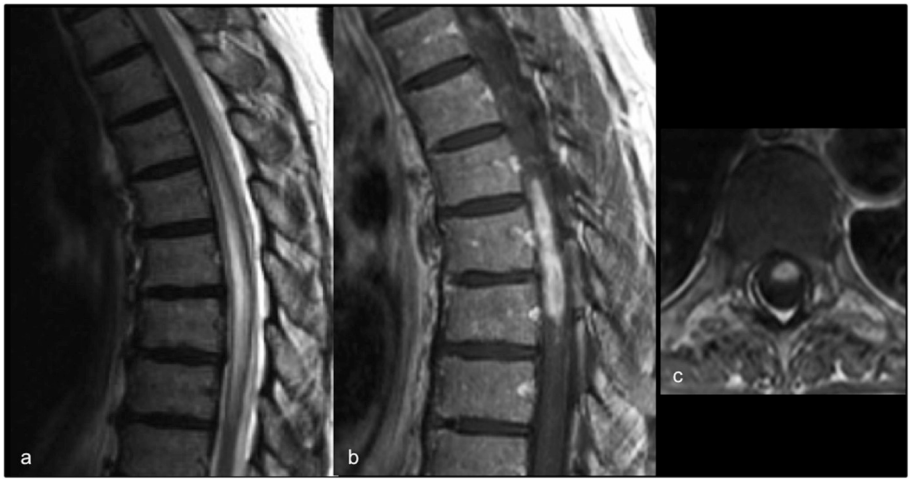

Fig. 2. Neuromyelitis optica (NMO). Sagittal T2WI (a) in a patient with known poor-controled NMO demonstrate longitudinally extensive transverse myelitis with enlargement of the upper dorsal spinal cord, and strong enhancement after contrast administration on sagittal and axial T1WI (b,c).

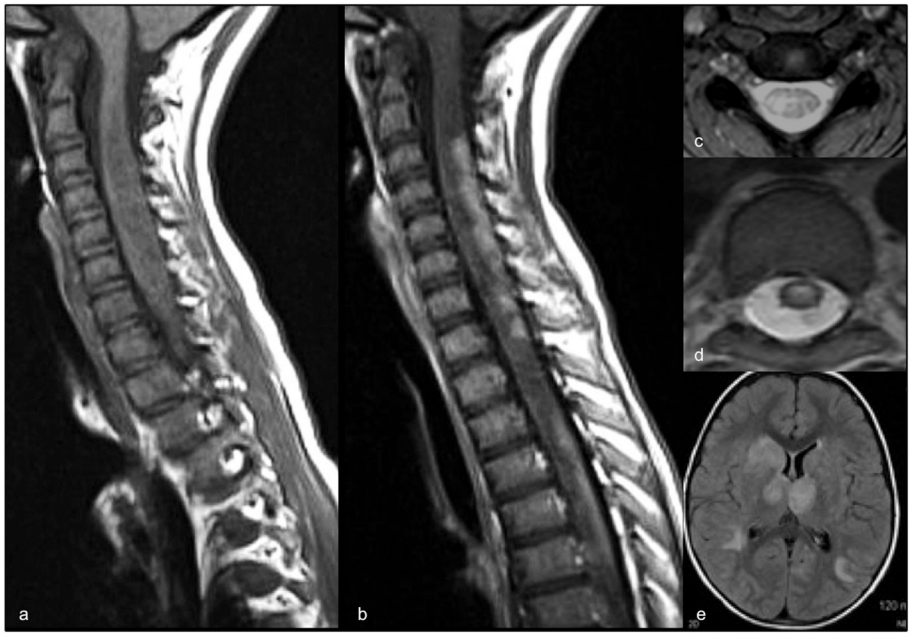

Fig. 3. Acute disseminated encephalomyelitis (ADEM). Sagittal pre- and post-contrast T1WI (a,b) in a 5-years-old boy 3 weeks after an infection show enlarged cervical spinal cord with marked enhancement after Gadolinium administration, and increased signal on axial T2 sequences (c,d). Multiple lesions involving basal ganglia, periventricular and subcortical white matter are shown on the FLAIR sequence (e). *Courtesy of Dr. S. Goldstein.*

manifestation of SLE. The most common MRI pattern is TM (Fig. 4). Enhancement is usually absent or minimal/patchy in active presentations. The outcome is variable, ranging from complete recovery to severe residual disability [23–25].

2.5. Spinal cord infarct

Anterior spinal artery infarction is the most frequent subtype, accounting for 5–8% of acute myelopathies, and sudden onset is the main clinical feature. Common contexts are aortic pathology/surgery, or systemic hypotension in elderly patients with cardiovascular risk factors [26,27].

Bilateral injury to the gray matter of the anterior horns, corticospinal and spinothalamic tracts, frequently involving a long segment, while sparing the dorsal columns, are the most common imaging manifestations. Usually, the lower spinal cord and the conus medullaris are involved [26,28]. Despite technical challenges, restricted diffusion (cytotoxic oedema) could be demonstrated [27,29] (Fig. 5).

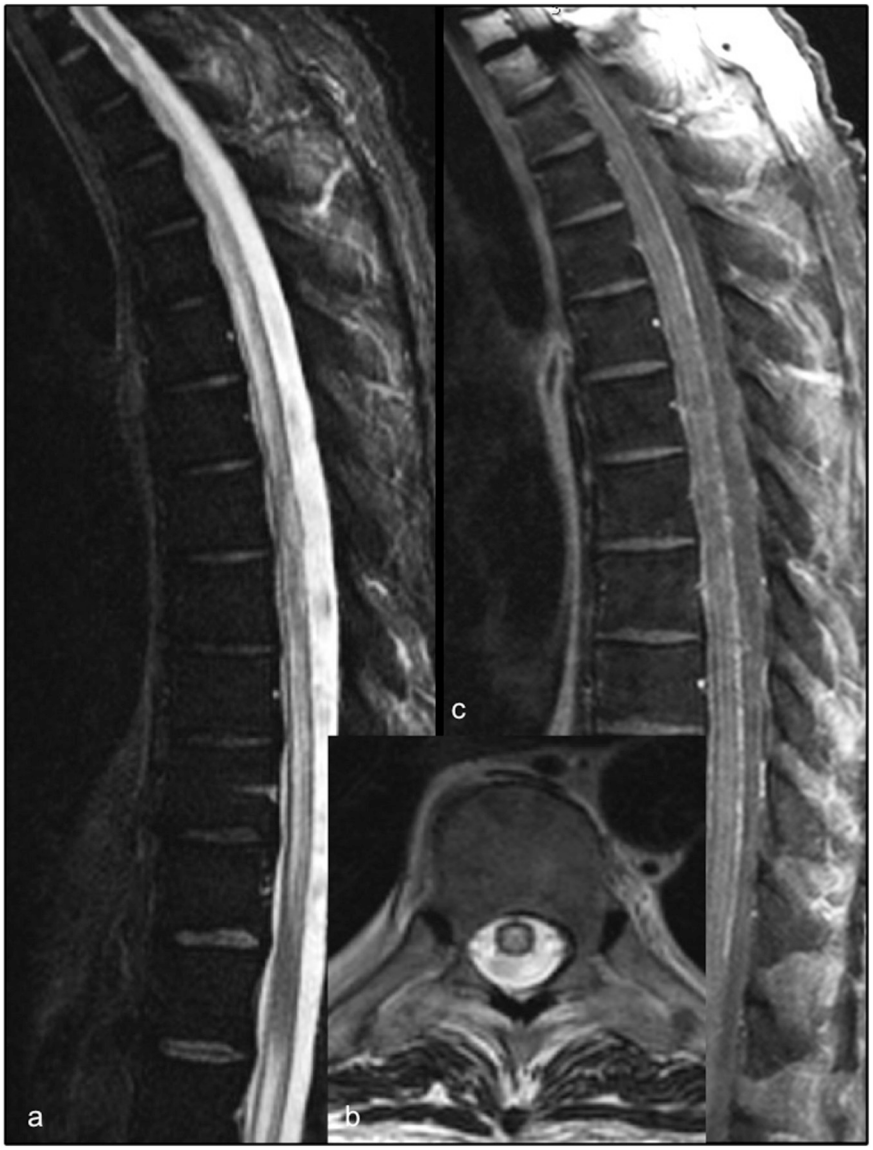

Fig. 4. Lupus myelitis. 27 years-old woman previously diagnosed with neuropsychiatric lupus who develops acute paraparesis and sphincter dysfunction. Longitudinally extensive transverse myelitis on sagittal STIR (a) and axial T2WI (b), with leptomeningeal contrast-enhancement (c).

2.6. Spinal cord arteriovenous shunts

From genetic perspectives, spinal cord vascular malformations were classified into three groups: genetic hereditary lesions, caused by disorders of vascular germinal cells, such as hereditary hemorrhagic telangiectasia; genetic nonhereditary lesions including spinal metameric syndromes such as Cobb, Klippel-Trenaunay and Parkes-Weber syndromes; and isolated sporadic arterio-venous shunts which is the largest group [30].

From the vascular anatomy point of view, spinal cord arteriovenous shunts were classified as dural and pial. The dural lesions are the dural arteriovenous fistulas (DAVFs), presumably acquired, accounting for 70% of all arteriovenous shunts of the spine, while the pial lesions are the spinal arterio-venous malformation (AVMs), considered inborn lesions. DAVFs are fed by radiculo-meningeal arteries, while AVMs by spinal cord feeding arteries (radiculo-medullary or radiculopial). AVMs are further classified as nidus-type (nidiform, glomerular or plexiform), with an intervening network of vessels, and fistulous, with direct

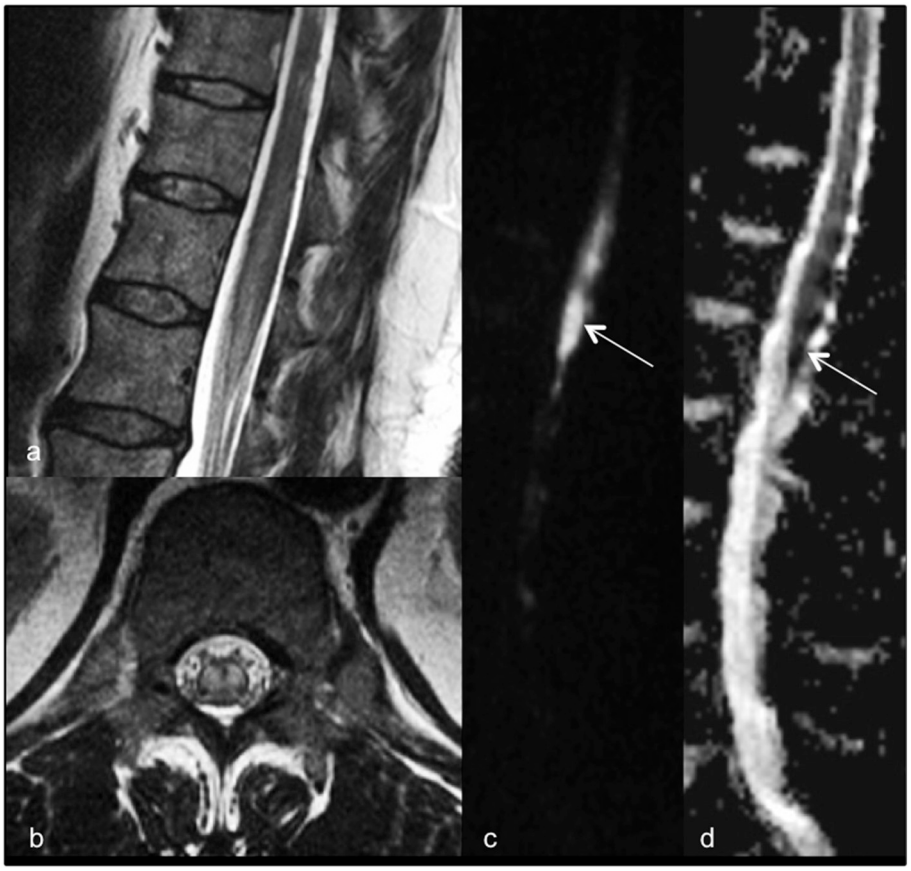

Fig. 5. Spinal cord infarct. Sudden paraparesis in a patient after aortic intervention. Sagittal and axial T2WI (a,b), sagittal DWI (c) and ADC map (d) show a slightly hyperintense area (a,b) with diffusion-restriction (arrows), involving the anterior columns of the conus medullaris, consistent with spinal cord ischemia.

communication. Nidus-type AVMs are more common and usually intramedullary, whereas fistulous AVMs are located superficially on the spinal cord (also called perimedullary fistula-type AVMs or intradural AVFs). Fistulous AVMs are further subclassified, depending on the size of the feeding and draining vessels, and the shunt volume, into macro- and microfistulas, the later ones accounting for the majority of the fistulous AVMs [31,32].

According to the location, spinal cord arteriovenous shunts were also classified as intramedullary, pial, dural and epidural [33].

Spinal cord cavernomas represent non-shunting spinal vascular malformation [5,31].

MRI in DAVFs (Fig. 6) and AVMs could show dilated serpentine perimedullary vessels and spinal cord edema with longitudinal involvement of central cord. In DAVFs there is a common involvement of the conus medullaris. In AVMs a medullary distribution of the vessels or a nidus could be seen (Fig. 7). Sometimes, hemorrhagic complications can be observed [31,34–36].

2.7. Posterior reversible encephalopathy (PRES)

PRES is a neurotoxic state related to dysfunction of cerebrovascular regulation, frequently in the setting of severe hypertension, eclampsia and exposure to cytotoxic drugs. Classical imaging findings include cortical and subcortical areas of vasogenic edema, typically reversible. Rarely, ischemia or hemorrhage can occur. The parieto-occipital lobes are the most frequently affected sites. The anterior regions, basal ganglia, brainstem and cerebellum can also be involved. Involvement of the spinal cord is exceedingly rare [22,37–39] (Fig. 8).

2.8. Subacute combined degeneration (SCD)

The term SCD refers to demyelination and vacuolation of the posterior and lateral columns of the spinal cord (Fig. 9), usually subsequent to copper, vitamin E, or, more frequently, vitamin B12 deficiency [2]. Risk factors include dietary deficiency, gastric surgery, autoimmune gastritis and pernicious anemia, toxic exposures (nitrous oxide – N2O or

Fig. 6. Spinal dural arterio-venous fistula. Sagittal T2WI (a,b) show an extensive spinal cord involvement with T2 hyperintensity, sparing the outmost peripheral regions. Serpiginous dilated vessels with perimedullary distribution are noticed (arrows).

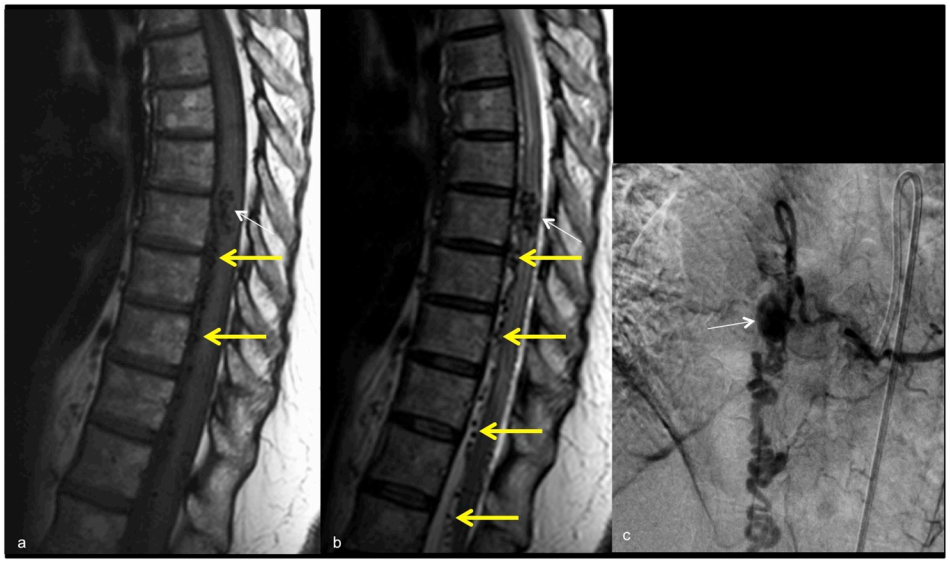

Fig. 7. Spinal arterio-venous malformation (AVM). Sagittal T1WI (a) and T2WI (b) show severely dilated perimedullary vessels (yellow arrows) and an intramedullary nidus (white arrows). There is also a hyperintensity of the spinal cord (b). Catheter angiography demonstrates the nidus (c, arrow) and the arterialized venous drainage, confirming the AVM. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

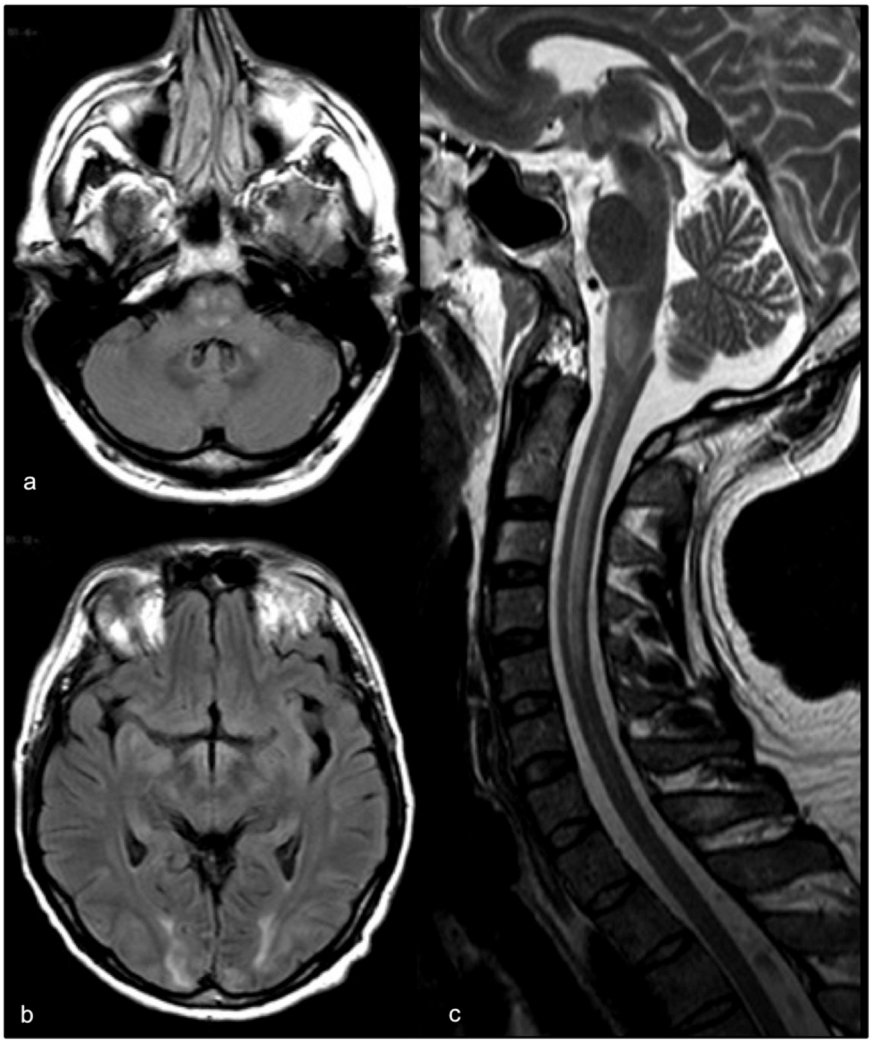

Fig. 8. Posterior reversible encephalopathy (PRES). 46-year-old male with headache, visual disturbances and vomiting, with systolic blood pressure of 210 mmHg, who progressed to coma. Axial brain FLAIR sequences (a,b) show hyperintense areas in the brainstem (a) and the occipital lobes (b), which corresponded to vasogenic oedema. Sagittal T2WI (c) shows longitudinally extensive hyperintensity and mild expansion of the upper cervical cord. Follow-up MRIs (not shown) several days after blood pressure normalization showed resolution of the abnormalities. *Courtesy of Prof. D. Balériaux.*

"laughing gas"), and genetic disorders such as Menkes disease [2,40]. Involvement of the posterior columns of the spinal cord is also typical in syphilitic myelitis ("tabes dorsalis") as well [41].

2.9. Neurosarcoidosis

Sarcoidosis affects the CNS in 5–10% of all cases. Spinal cord involvement is uncommon and has been reported in 6–8% of patients with CNS disease, most frequently in the cervical region. Lesions are often longitudinally extensive and sometimes may mimic those of an intramedullary mass. Cord enlargement and associated parenchymal and leptomeningeal enhancement are common (Fig. 10). Brain MRI frequently shows other signs of neurosarcoidosis [42,43].

2.10. Viral myelitis

Different classes of microorganisms can cause myelitis (viruses, bacteria, tuberculosis, spirochetes, fungi). They are uncommon but have to be considered in the differential diagnosis of acute myelopathies. MRI may reveal a swollen cord or focal lesions, and frequently contrast-enhancement of the cord surface, of the nerve roots, or even patchy intramedullary enhancement of peripheral distribution [44–46] (Fig. 11).

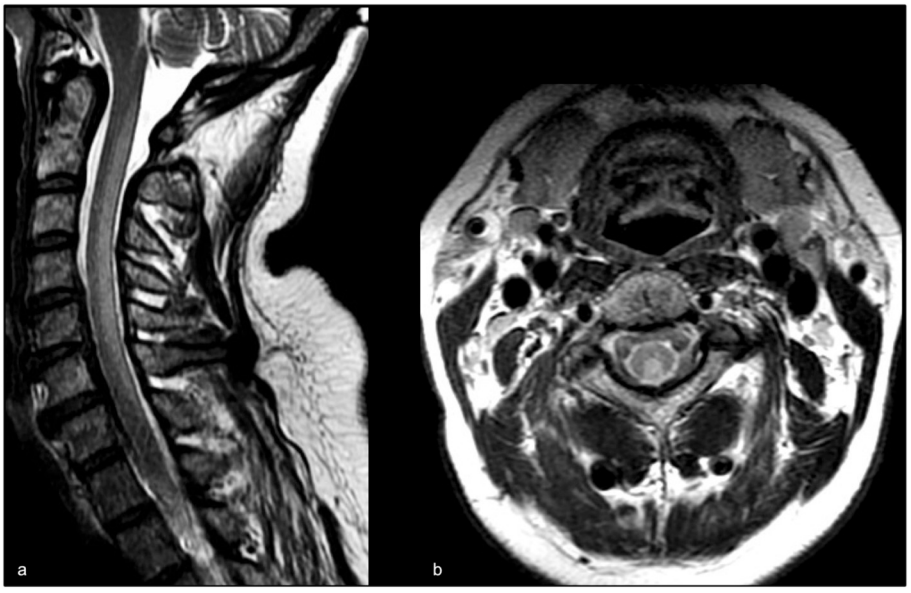

Fig. 9. Subacute combined degeneration. 24-years-old woman, strict vegetarian, with vitamin B12 deficiency, presenting with numbness, tingling and weakness. Sagittal (a) and axial (b) T2WI demonstrating a longitudinally extensive bilateral hyperintensity of posterior columns of the cervical spinal cord.

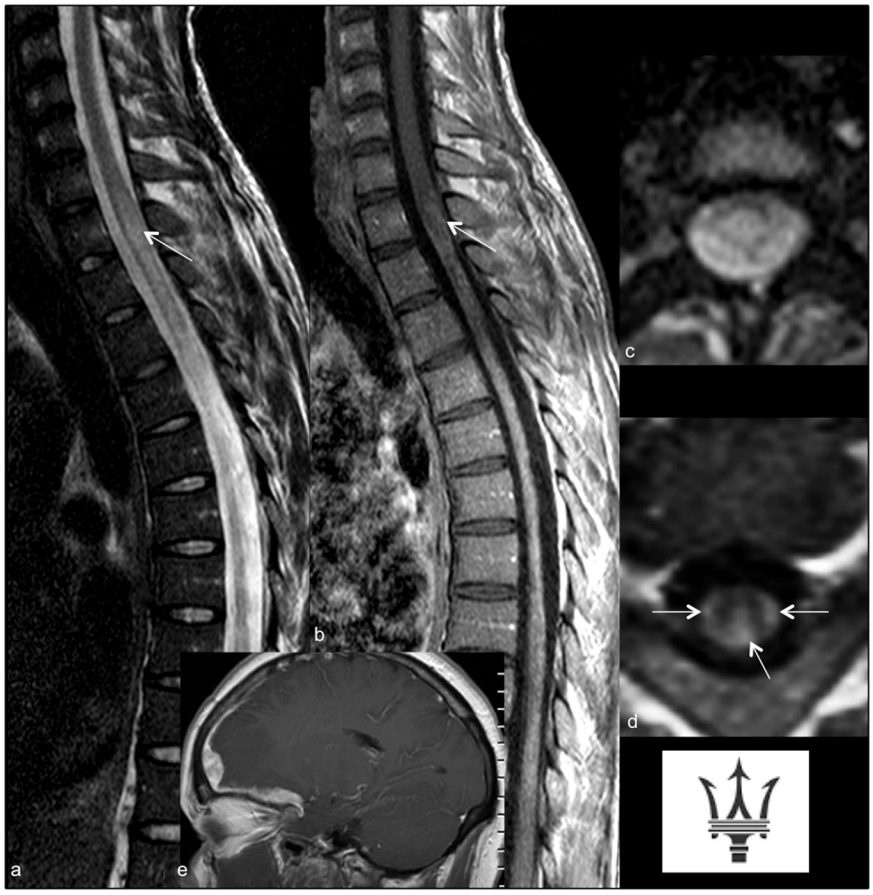

Fig. 10. Neurosarcoidosis. Sagittal T2WI (a) demonstrates longitudinally extensive hyperintensity and mildly expansion of the cervico-thoracic spinal cord (arrow), with slight peripheral enhancement after contrast administration on sagittal T1WI (b, arrow). Axial images (c,d) showing an important extension of the T2-hyperintensity on the axial plane (c) and a posterior and peripheral enhancement on contrast-enhanced T1WI (d, arrows) resembling a "trident" showed below. Contrast-enhanced T1WI of the brain (e) shows meningeal enhancement, predominantly in the anterior skull base and the basal cisterns.

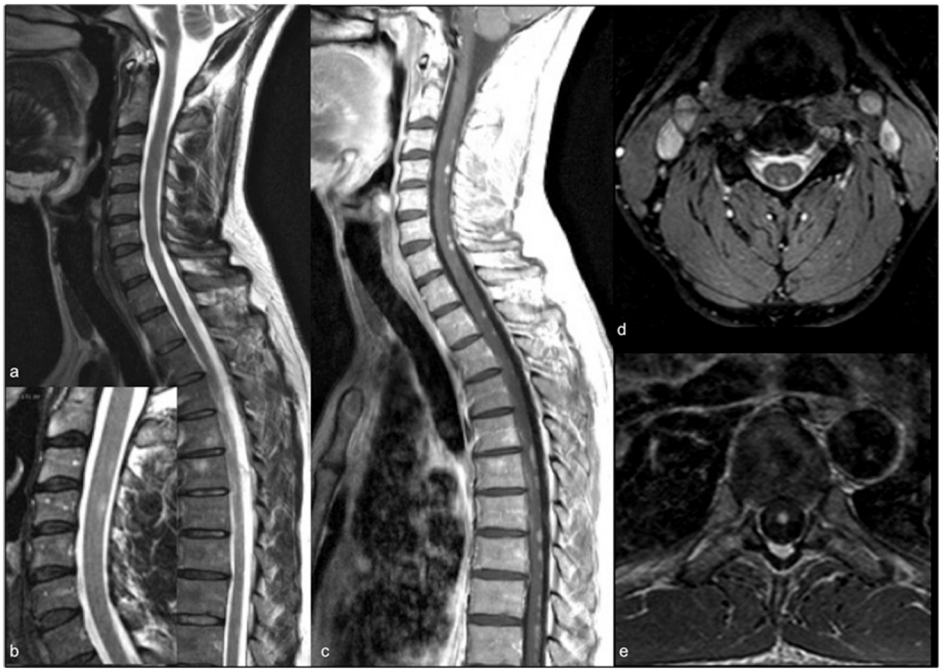

Fig. 11. Viral myelitis. 40-year-old male patient presenting with inferior limbs and perineal dysesthesia, in the setting of genital Herpes Simplex infection (with positive IgM anti-HSV antibodies). MRI revealed multiple, short segment T2 hyperintense lesions in the posterior midline cervical and thoracic spinal cord (a,b,d), with Gadolinium enhancement (c,e), consistent with areas of active demyelination. *Courtesy of Prof. D. Balériaux.*

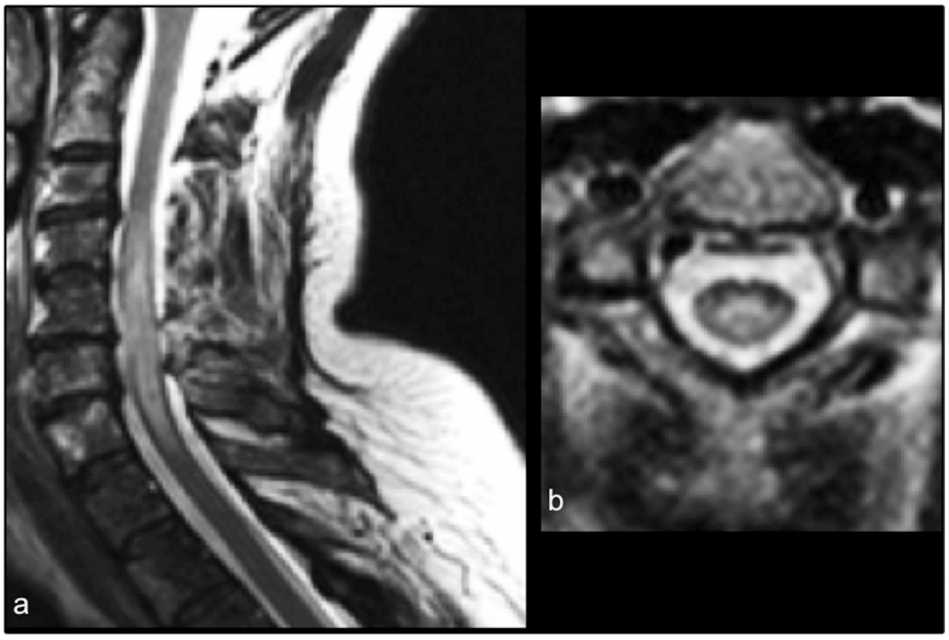

Fig. 12. Radiation myelitis. Patient with breast cancer, bone metastases and diffuse infiltration of the vertebral column, with recent radiotherapy in the cervical region. Sagittal and axial T2 (a,b) show enlargement of the cervical spinal cord with T2 hyperintensity, as well as heterogeneous aspect of the bone marrow.

2.11. Radiation myelitis

Spinal cord damage may occur secondary to radiation therapy for vertebral metastases or primary neck, thoracic, or abdominal cancers. Depending on the time from radiotherapy, radiation myelitis can be early and transient (first 6 month) or late (> 6 months). MRI usually shows extensive cord swelling corresponding to the location of the radiation (Fig. 12). Patchy contrast-enhancement is frequently observed [47].

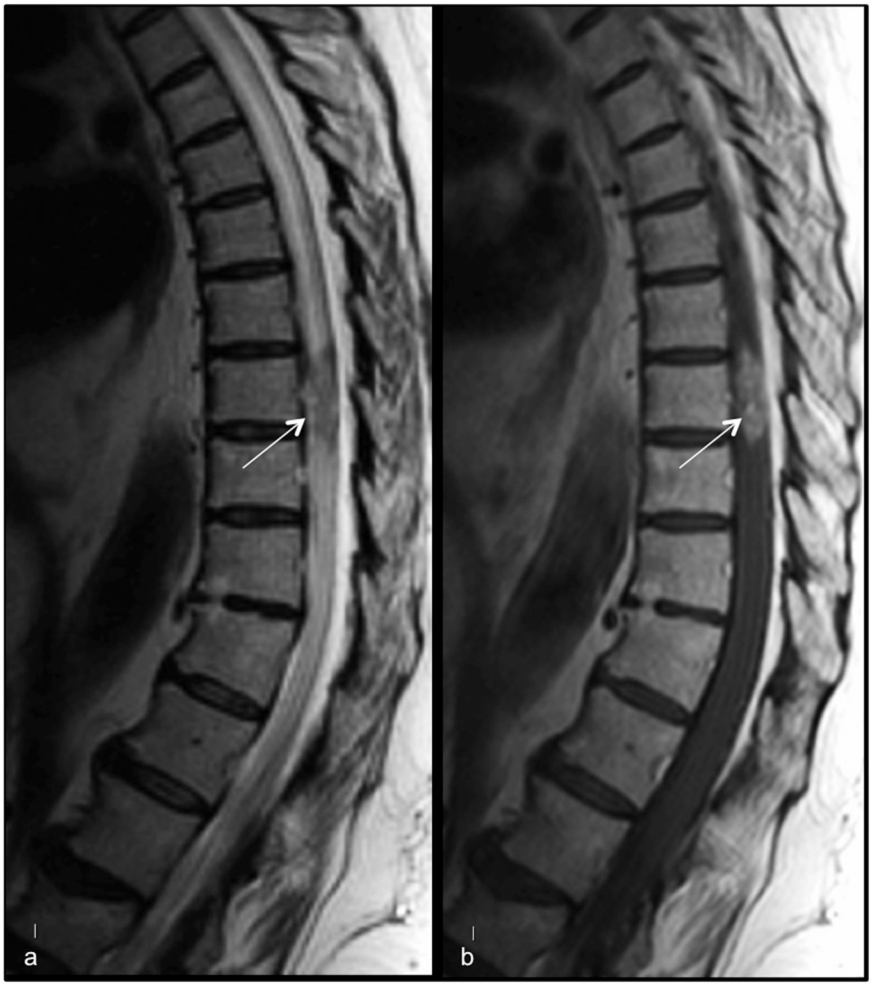

Fig. 13. Spinal cord metastasis. Sagittal T2WI (a) and post-contrast T1WI (b) in a patient with previous diagnosis of disseminated breast cancer, show an intramedullary lesion with contrast enhancement (arrows) and associated diffuse hyperintensity of the entire spinal cord (a) indicating a very extensive spinal cord oedema.

2.12. Spinal cord metastasis

Intramedullary metastases are rare and usually occur in the setting of extensive metastatic disease. They are most common in the cervical and thoracic cord and in posterolateral regions. MRI findings are intramedullary lesion, extensive edema and cord swelling. Contrast administration will demonstrate the lesion [48–50] (Fig. 13).

2.13. Compressive myelopathy

There are multiple causes of cord compression, usually of degenerative, traumatic, infectious (abscesses) or tumoral etiology. Imaging studies show displacement and compression of the spinal cord, canal stenosis, and the primary responsible condition (Fig. 14). Myelopathy is frequently localized to the site of compression [51,52].

A diagram of the most common imaging patterns of myelopathy is shown on the Fig. 15.

3. Conclusion

A systematic approach to myelopathies can help narrow the differential diagnosis and is therefore crucial for the therapeutic strategy. MRI represents a milestone in the diagnostic work-up of myelopathies and has also a prognostic role, offering valuable information about disease extension and potential complications.

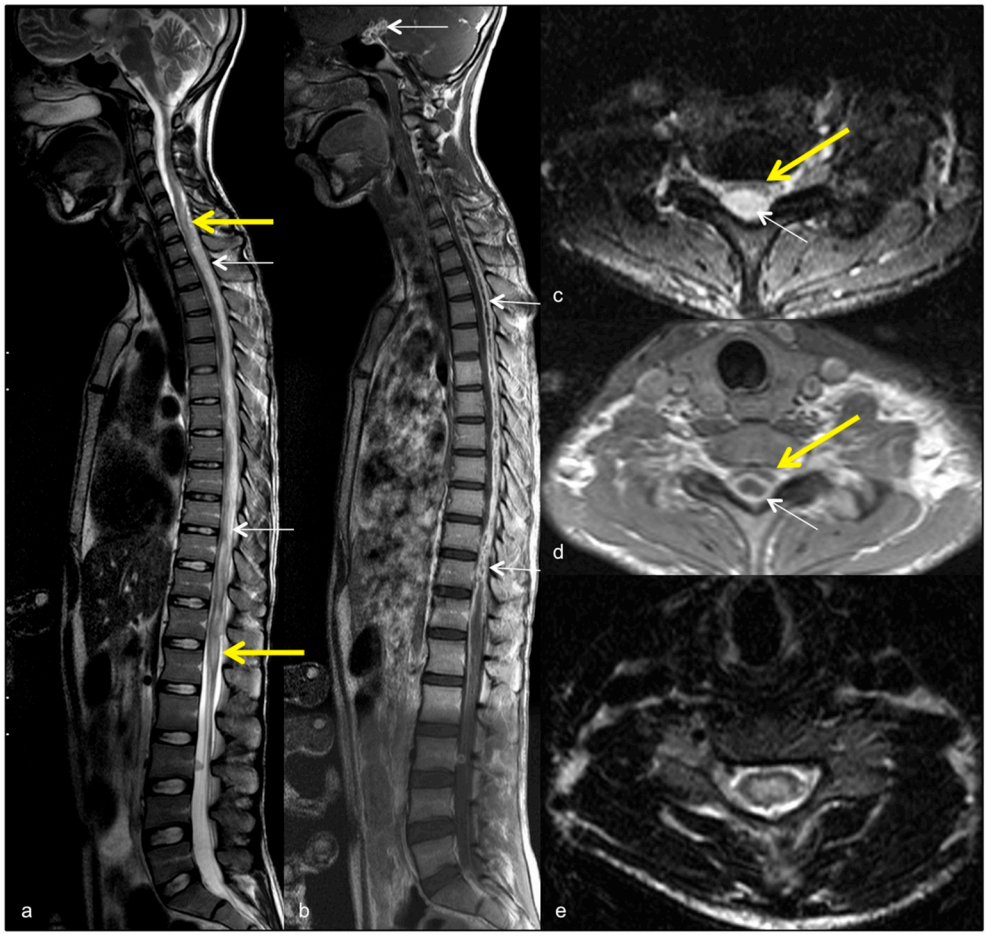

Fig. 14. Myelopathy in disseminated tuberculosis (TB) with compressive subdural collections. Sagittal T2WI (a) and contrast-enhanced T1WI (b) show very extensive subdural collections (white arrows) and hyperintensity of the cord at the ends of the collection (yellow arrows). Axial T2WI (c) and contrast-enhanced T1WI (d) at the cervico-dorsal junction demonstrate the subdural abscess posteriorly (white arrows) and the spinal cord severely displaced and compressed anteriorly (yellow arrows), while a level superior to the abscess the cord is expanded and hyperintense on T2WI (e). TB could be per se a cause of myelitis but the compressive mechanism seemed primordial in this case. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

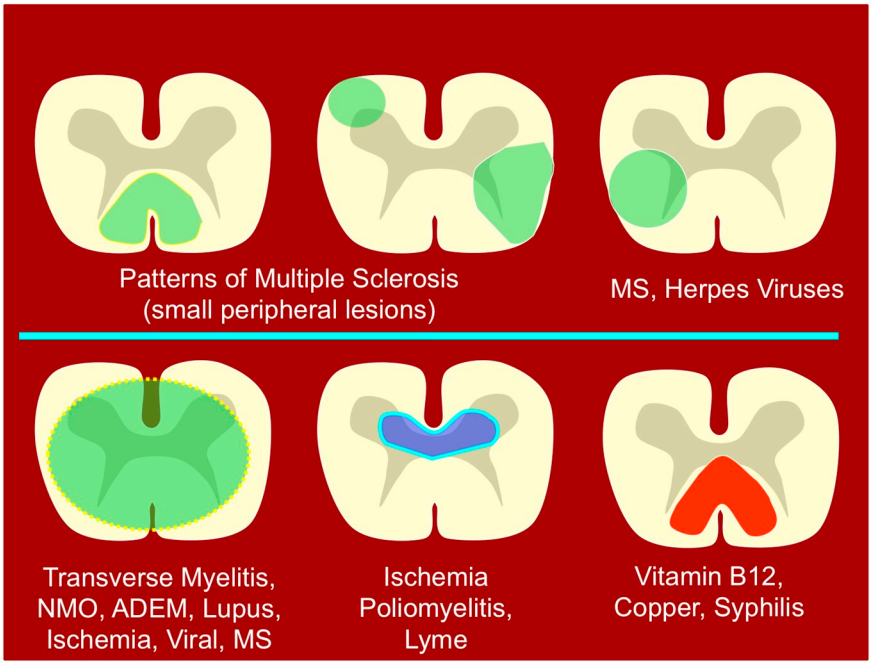

Fig. 15. Diagram showing most frequent patterns of involvement of the spinal cord and associated entities.

Declaration of Competing Interest

The authors have no competing interests to declare.

References

[1] Lavi ES, Pal A, Bleicher D, Kang K, Sidani C. MR imaging of the spine: urgent and emergent indications. Semin Ultrasound CT MRI 2018;39:551–69.
[2] Ramalho J, Nunes RH, da Rocha AJ, Castillo M. Toxic and metabolic myelopathies. Semin Ultrasound, CT MRI 2016;37:448–65.
[3] Transverse Myelitis Consortium Working Group. Proposed diagnostic criteria and nosology of acute transverse myelitis. Neurology 2002;59:499–505.
[4] Sá MJ. Acute transverse myelitis: a practical reappraisal. Autoimmun Rev 2009;9:128–31.
[5] Weidauer S, Wagner M, Nichtweiß M. Magnetic resonance imaging and clinical features in acute and subacute myelopathies. Clin Neuroradiol 2017;27:417–33.
[6] Goh C, Desmond PM, Phal PM. MRI in transverse myelitis. J Magn Reson Imaging 2014;40:1267–79.
[7] Nichtweiß M, Weidauer S. Differential diagnosis of acute myelopathies: an update. Clin Neuroradiol 2015;25:183–7.
[8] Cao J, Cui L, Jiang X, Gao S, Sun W, Xu K. Clinical application and diagnostic value of noninvasive spinal angiography in spinal vascular malformations. J Comput Assist Tomogr 2014;38:474–9.
[9] Morales H, Betts A. Abnormal spinal cord magnetic resonance signal: approach to the differential diagnosis. Semin Ultrasound, CT MRI 2016;37:372–83.
[10] Choi KH, Lee KS, Chung SO, Park JM, Kim YJ, Kim HS, et al. Idiopathic transverse myelitis: MR characteristics. AJNR Am J Neuroradiol 1996;17:1151–60.
[11] Sanders KA, Khandji AG, Mohr JP. Gadolinium-MRI in acute transverse myelopathy. Neurology 1990;40:1614–6.
[12] Ozawa H, Sato T, Hyodo H, Ishii Y, Morozumi N, Koizumi Y, et al. Clinical significance of intramedullary Gd-DTPA enhancement in cervical myelopathy. Spinal Cord 2010;48:415–22.
[13] Mathur S, Bharatha A, Huynh TJ, Aviv RI, Symons SP. Comparison of time-resolved and first-pass contrast-enhanced MR angiography in pretherapeutic evaluation of spinal dural arteriovenous fistulas. AJNR Am J Neuroradiol 2017;38:206–12.
[14] Condette-Auliac S, Boulin A, Roccatagliata L, Coskun O, Guieu S, Guedin P, et al. MRI and MRA of spinal cord arteriovenous shunts. J Magn Reson Imaging 2014;40:1253–66.
[15] Lindenholz A, TerBrugge KG, van Dijk JMC, Farb RI. The accuracy and utility of contrast-enhanced MR angiography for localization of spinal dural arteriovenous fistulas: the Toronto experience. Eur Radiol 2014;24:2885–94.
[16] Vargas MI, Delattre BMA, Boto J, Gariani J, Dhouib A, Fitsiori A, et al. Advanced magnetic resonance imaging (MRI) techniques of the spine and spinal cord in children and adults. Insights Imaging 2018;9:549–57.
[17] Thompson AJ, Banwell BL, Barkhof F, Carroll WM, Coetzee T, Comi G, et al. Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria. Lancet Neurol 2018;17:162–73.
[18] Zabad RK, Stewart R, Healey KM. Pattern recognition of the multiple sclerosis syndrome. Brain Sci 2017;7:138.
[19] Wingerchuk DM, Banwell B, Bennett JL, Cabre P, Carroll W, Chitnis T, et al. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. Neurology 2015;85:177–89.
[20] Wingerchuk DM, Lennon VA, Lucchinetti CF, Pittock SJ, Weinshenker BG. The spectrum of neuromyelitis optica. Lancet Neurol 2007;6:805–15.
[21] Weber MS, Derfuss T, Metz I, Brück W. Defining distinct features of anti-MOG antibody associated central nervous system demyelination. Ther Adv Neurol Disord 2018;11:1756286418762083.
[22] Sarbu N, Shih RY, Jones RV, Horkayne-Szakaly I, Oleaga L, Smirniotopoulos JG. White matter diseases with radiologic-pathologic correlation. Radiographics 2016;36:1426–47.
[23] Espinosa G, Mendizábal A, Mínguez S, Ramo-Tello C, Capellades J, Olivé A, et al. Transverse myelitis affecting more than 4 spinal segments associated with systemic lupus erythematosus: clinical, immunological, and radiological characteristics of 22 patients. Semin Arthritis Rheum 2010;39:246–56.
[24] Sarbu N, Sarbu MI, Bargallo N, Cervera R. Future perspectives in the diagnosis of neuropsychiatric lupus by advanced magnetic resonance imaging techniques. Curr Rheumatol Rev 2018;14:213–8.
[25] Roura E, Sarbu N, Oliver A, Valverde S, González-Villà S, Cervera R, et al. Automated detection of lupus white matter lesions in MRI. Front Neuroinform 2016;10:33.
[26] Yadav N, Pendharkar H, Kulkarni GB. Spinal cord infarction: clinical and radiological features. J Stroke Cerebrovasc Dis 2018;27:2810–21.
[27] Vargas MI, Gariani J, Sztajzel R, Barnaure-Nachbar I, Delattre BM, Lovblad KO, et al. Spinal cord ischemia: practical imaging tips, pearls, and pitfalls. AJNR Am J Neuroradiol 2015;36:825–30.
[28] Vuong SM, Jeong WJ, Morales H, Abruzzo TA. Vascular diseases of the spinal cord: infarction, hemorrhage, and venous congestive myelopathy. Semin Ultrasound, CT MRI 2016;37:466–81.
[29] Thurnher MM, Bammer R. Diffusion-weighted MR imaging (DWI) in spinal cord ischemia. Neuroradiology 2006;48:795–801.
[30] Rodesch G, Hurth M, Alvarez H, Tadié M, Lasjaunias P. Classification of spinal cord arteriovenous shunts: proposal for a reappraisal-the Bicêtre experience with 155 consecutive patients treated between 1981 and 1999. Neurosurgery 2002;51:374–9.
[31] Krings T. Vascular malformations of the spine and spinal cord. Clin Neuroradiol 2010;20:5–24.
[32] Krings T, Thron AK, Geibprasert S, Agid R, Hans FJ, Lasjaunias PL, et al. Endovascular management of spinal vascular malformations. Neurosurg Rev 2010;33:1–9.
[33] Takai K. Spinal arteriovenous shunts: angioarchitecture and historical changes in classification. Neurol Med Chir (Tokyo) 2017;57:356–65.
[34] Rubin MN, Rabinstein A. Vascular diseases of the spinal cord. Neurol Clin 2013;31:153–81.
[35] Da Costa L, Dehdashti AR, terBrugge KG. Spinal cord vascular shunts: spinal cord vascular malformations and dural arteriovenous fistulas. Neurosurg Focus 2009;26:E6.
[36] Morris JM. Imaging of dural arteriovenous fistula. Radiol Clin North Am 2012;50:823–39.
[37] Ou S, Xia L, Wang L, Xia L, Zhou Q, Pan S. Posterior reversible encephalopathy syndrome with isolated involving infratentorial structures. Front Neurol 2018;9:843.
[38] Chen T-H, Lin W-C, Kao W-T, Tseng C-M, Tseng Y-H. Posterior reversible encephalopathy syndrome with spinal cord involvement in children. J Child Neurol 2017;32:112–9.
[39] Liu L, Dai D, Cao F, Zhang L, Wang X. Posterior reversible encephalopathy syndrome with spinal cord involvement but without hemisphere lesions: a case report. Medicine (Baltimore) 2019;98:e13649.
[40] Schimana R, Ober H, Neuhaus O. Subacute combined degeneration of the spinal cord in vitamin B_{12} and copper deficiency. Case Reports 2017;2017:22–4. [2017:bcr-2017-222446].
[41] Tsui EYK, Ng SH, Chow L, Lai KF, Fong D, Chan JHM. Syphilitic myelitis with diffuse spinal cord abnormality on MR imaging. Eur Radiol 2002;12:2973–6.
[42] Soni N, Bathla G, Pillenahalli Maheshwarappa R. Imaging findings in spinal sarcoidosis: a report of 18 cases and review of the current literature. Neuroradiol J 2019;32:17–28.
[43] Rovira Á, Auger C, Rovira A. Other noninfectious inflammatory disorders. Handb Clin Neurol 2016;135:425–46.
[44] Yokota H, Yamada K. Viral infection of the spinal cord and roots. Neuroimaging Clin N Am 2015;25:247–58.
[45] Montalvo M, Cho TA. Infectious myelopathies. Neurol Clin 2018;36:789–808.
[46] DeSanto J, Ross JS. Spine infection/inflammation. Radiol Clin North Am 2011;49:105–27.
[47] Khan M, Ambady P, Kimbrough D, Shoemaker T, Terezakis S, Blakeley J, et al. Radiation-induced myelitis: initial and follow-up MRI and clinical features in patients at a single tertiary care institution during 20 years. AJNR Am J Neuroradiol 2018;39:1576–81.
[48] Kosmas C, Koumpou M, Nikolaou M, Katselis J, Soukouli G, Markoutsaki N, et al. Intramedullary spinal cord metastases in breast cancer: report of four cases and review of the literature. J Neurooncol 2005;71:67–72.
[49] Payer S, Mende KC, Westphal M, Eicker SO. Intramedullary spinal cord metastases: an increasingly common diagnosis. Neurosurg Focus 2015;39:E15.
[50] Kalayci M, Cağavi F, Gül S, Yenidünya S, Açikgöz B. Intramedullary spinal cord metastases: diagnosis and treatment - an illustrated review. Acta Neurochir 2004;146:1347–54.
[51] You JY, Lee JW, Lee E, Lee GY, Yeom JS, Kang HS. MR classification system based on axial images for cervical compressive myelopathy. Radiology 2015;276:553–61.
[52] Nouri A, Martin AR, Mikulis D, Fehlings MG. Magnetic resonance imaging assessment of degenerative cervical myelopathy: a review of structural changes and measurement techniques. Neurosurg Focus 2016;40(6):E5.