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Radiological approach to non-compressive myelopathies

M. Sarthak Swarup, Stuti Chandola, Radhika Batra*, Anjali Prakash and Anju Garg

Abstract

Background: Myelopathy, a pathological condition related to the spinal cord can broadly be categorized into compressive and non-compressive aetiologies. Magnetic resonance imaging remains the modality of choice when suspecting non-compressive myelopathy as it helps to localize the affected segment and exclude compression as the cause of myelopathy. This review deals with the imaging approach for non-compressive myelopathies.

Main body: Demyelinating disorders are the most common cause of non-compressive myelopathy and often show confounding features. Other causes include inflammatory, ischemic, metabolic, and neoplastic disorders. Non-compressive myelopathy can broadly be classified into acute and non-acute onset which can further be categorized according to the distribution of the signal abnormalities, including length of cord involvement, specific tract involvement, enhancement pattern, and the region of the spinal cord that is affected.

Conclusions: Imaging plays a critical role in the evaluation of clinically suspected cases of myelopathy and MR imaging (with or without contrast) remains the preferred modality. Compressive causes must be excluded as a cause of myelopathy. Despite a multitude of causes, the most common imaging appearance is a nonspecific T2 hyperintense signal in the spinal cord, and thus, a pragmatic diagnostic approach along with appropriate clinical and biochemical correlation is essential for arriving at an accurate diagnosis.

Keywords: Non-compressive myelopathy, Spinal cord, Magnetic resonance imaging, Demyelinating diseases, Infectious myelitis

Background

Myelopathy refers to a pathological condition with neurological deficits related to the spinal cord affection. It can broadly be categorized into compressive and non-compressive aetiologies. Magnetic resonance imaging remains the modality of choice, as it helps to localize the affected segment and exclude compression as the cause of myelopathy. After ruling out compressive aetiologies, the clinical history needs to be carefully analyzed for presentation and duration since the onset of symptoms. Myelopathy is considered acute in onset when the symptoms progress to their peak within 21 days [1]. Myelopathy that exhibits a more progressive time course can be considered subacute (weeks to months) or chronic (months to years) [2]. In addition to the pattern of clinical presentation, analysis of certain features on MRI may help to narrow down the differential diagnosis. These features include distribution and location of the abnormal signal, the longitudinal extent of cord involvement (short segment or long segment), the cross-sectional area involved, and the enhancement pattern [3]. In addition, the multiplicity of the lesion(s), presence of cord swelling, and the specific region of the spinal cord that is involved also need to be evaluated. Various causes of acute and nonacute presentations of non-compressive myelopathy have been listed in Tables 1 and 2, respectively, with the cross-sectional patterns of spinal cord involvement presented in Table 3.

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Table 1 Common causes of acute non-compressive myelopathy

Category	Pathologies
Demyelinating	Multiple sclerosis NMOSD ADEM
Systemic inflammatory	SLE Sjögren's syndrome Other connective tissue disorders Sarcoidosis
Infectious	Various viral, bacterial and fungal pathogens
Idiopathic	Idiopathic ATM
Vascular	Ischaemia, infarction
Others	Toxic, Drug-induced, Post-irradiation

NMOSD neuromyelitis optica spectrum disease, *ADEM* acute disseminated encephalomyelitis, *SLE* systemic lupus erythematosus, *ATM* acute transverse myelitis

Table 2 Common causes of subacute to chronic non-compressive myelopathy

Category	Pathologies
Metabolic	Vitamin B12 deficiency (Subacute combined degeneration) Vitamin E deficiency Chronic hepatic or renal disease
Vascular	Spinal Dural AVF/AVM Cavernoma
Neoplastic (intramedullary tumor)	Ependymoma, Astrocytoma, Hemangioblastoma Others – Metastases, Lymphoma
Inflammatory	SLE, Sjögren's syndrome, Sarcoidosis
Infectious	Chronic infection with HIV
Others	Post-irradiation Paraneoplastic/Autoimmune

AVF/AVM arteriovenous fistula/malformation, *SLE* systemic lupus erythematosus, *HIV* human immunodeficiency virus

Main body

Demyelinating disorders

Demyelinating disorders are a diverse group of diseases that show varying clinical and imaging features. They commonly present with features of myelopathy with altered spinal cord signal intensity on MR images. This group encompasses multiple sclerosis (MS), acute disseminated encephalomyelitis (ADEM), neuromyelitis optica spectrum disorder (NMOSD), and idiopathic transverse myelitis (TM) [2–4].

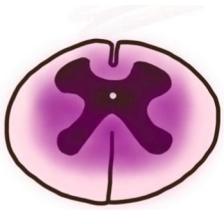
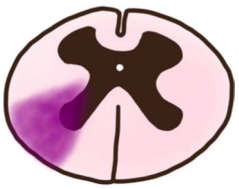
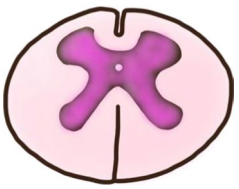
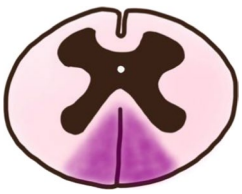
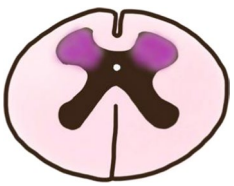
Multiple sclerosis (MS)

Multiple sclerosis is an immune-mediated demyelinating disease of the central nervous system which affects the brain and spinal cord. This is the most common demyelinating disease and affects females more commonly than males [3]. McDonald's criteria that assist in the diagnosis of this condition are based on demonstrating the multiplicity of the clinical attacks and the radiologic evidence of lesions being disseminated in space and time along with the presence of oligoclonal bands in CSF [5, 6]. Although more than 90% of patients with MS show spinal cord affection, isolated spinal involvement can be seen in up to 20–25% of patients, with the cervical segment of spinal cord being typically affected [7]. The lesions are usually multiple, short (spanning over one to two vertebral body segments), asymmetric, peripheral, wedge-shaped or round and affect less than 50% of the cross-sectional area of the cord on axial sections (usually in the lateral and posterior aspect of the spinal cord) (Fig. 1) [3, 4, 8]. Contrast enhancement may be seen in active or acute disease. In the chronic stage, cord atrophy may be seen [4, 7]. In the brain, the lesions are characteristically located at the calloso-septal interface and in periventricular, perivenular, intracortical, and infratentorial locations. When present, typical sites of cerebral involvement can aid in the diagnosis [8].

Acute disseminated encephalomyelitis (ADEM)

This is an acute inflammatory demyelinating disease that most commonly affects children with a history of viral infection or vaccination. With a propensity to involve both the brain and spinal cord, the lesions in ADEM are similar to MS but are more ill-defined, confluent, and often involve the deep gray nuclei (basal ganglia and thalamus). Approximately one-third of patients with ADEM show brainstem and spinal cord involvement [8]. In the spine, lesions are found more commonly in the thoracic cord, demonstrate ill-defined margins, show larger cross-sectional area and longer craniocaudal extent than MS (Fig. 2) [4, 9]. ADEM lesions usually do not show enhancement; however variable enhancement and cord swelling may be present in the acute stage [4, 8]. Other clinical features which suggest a diagnosis of ADEM include the monophasic course of the illness, signs of encephalopathy, and CSF analysis showing pleocytosis without oligoclonal bands [10].

Table 3 Cross-sectional patterns of spinal cord involvement in various causes of non-compressive myelopathy

Pattern of involvement	Disease entity	Images
Central cord	NMOSD, ADEM, Systemic inflammatory disease (SLE and Sjogren's syndrome), Idiopathic transverse myelitis, Infectious (viral, bacterial, fungal), Radiation	
Peripheral, wedge-shaped, or oval	Multiple sclerosis	
Central gray matter	Infarction, d AVF, AVM, compressive myelopathy	
Dorsal column	Vitamin B12 and copper deficiency, Zinc and Nitrous oxide toxicity, Autoimmune	
Anterior horn	Infarction (watershed), Infections (polio, enterovirus), Post-vaccination	

NMOSD neuromyelitis optica spectrum disease, *ADEM* acute disseminated encephalomyelitis, *SLE* systemic lupus erythematosus, *d AVF/AVM* dural arteriovenous fistula/malformation

Neuromyelitis optica spectrum disease (NMOSD)

Neuromyelitis Optica spectrum disease, also known as Devic's disease, is an autoimmune astrocytopathy that is more commonly seen in females. This is characterized by optic neuritis, myelitis, and the presence of autoantibody against the water channel protein aquaporin-4, also known as an anti-NMO-IgG antibody. Imaging shows a long segment T2 hyperintense signal of the cord traversing at least three vertebral body levels with predominant involvement of the central gray matter [11]. It frequently extends upward into the medulla [12]. NMOSD lesions tend to show more heterogeneity with poor definition of margins. About 50% of cases may show "bright spotty lesions" which are defined as focal internal areas of signal alteration showing hyperintense signal similar to that of CSF on T2 weighted images with corresponding low signal on T1, and this is highly specific to NMOSD [13]. In addition, the lesions can be

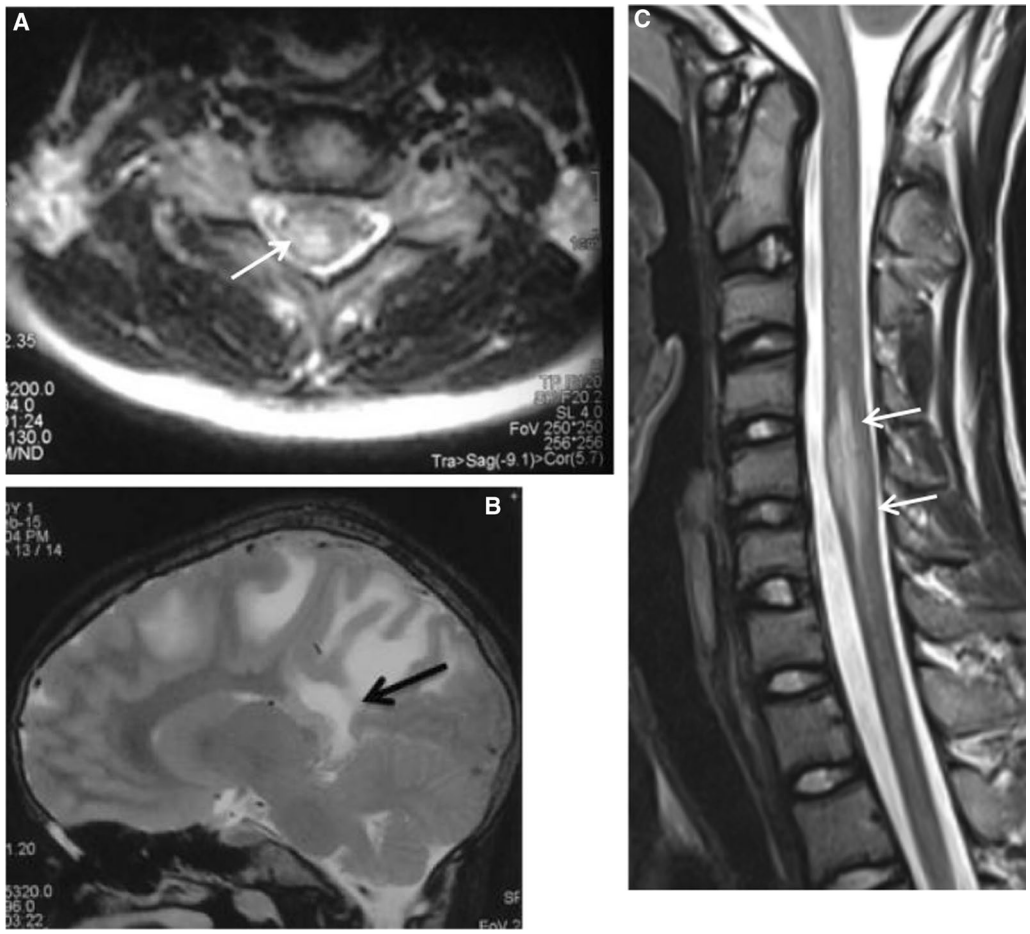


Fig. 1 Multiple sclerosis in a 13-year-old female who presented with paraparesis. T2W axial MR image of the upper cervical spine (**A**) shows a peripherally located wedge shaped hyperintense lesion (white arrow) which involves less than half of the cross-sectional area of the spinal cord. Sagittal T2W image of the brain (**B**) of the same patient demonstrates patchy areas of T2 hyperintensity in pericallosal and periventricular distribution (black arrow). Sagittal T2W MR image of the cervical and upper thoracic spine (**C**) in another case of diagnosed MS shows a short segment hyperintense area within the cervical spinal cord (white arrows)

expansile and often show enhancement on post-contrast images in acute settings [8]. Particularly, a 'lens-shaped' pattern of enhancement is observed on sagittal images in this disease [14].

Idiopathic acute transverse myelitis (ATM)

Idiopathic ATM is the most common acute inflammatory myelitis with the absence of a specific identifiable cause (such as MS, NMO, ADEM, connective tissue disease, infection, etc.) [15–17]. This is essentially a diagnosis of exclusion, which presents clinically with rapidly progressive, bilateral sensory and motor dysfunction with a distinct cord level. Pleocytosis and occasionally an elevated IgG index may be seen [15, 16]. It is more common in younger patients. On imaging, the lesions are typically located in the cervicothoracic spinal cord occupying at least two-thirds of the cross-sectional area of the cord with central cord predominance, with or without mild cord expansion (Fig. 3) [4, 15]. There is a variable length of cord involvement, with long segment involvement (longitudinally extensive TM) being more common than short segment involvement (acute partial TM) [17]. Enhancement is variable and is most commonly seen in the subacute stages. Enhancement patterns may be diffuse, poorly defined, heterogeneous, nodular, or peripheral [4].

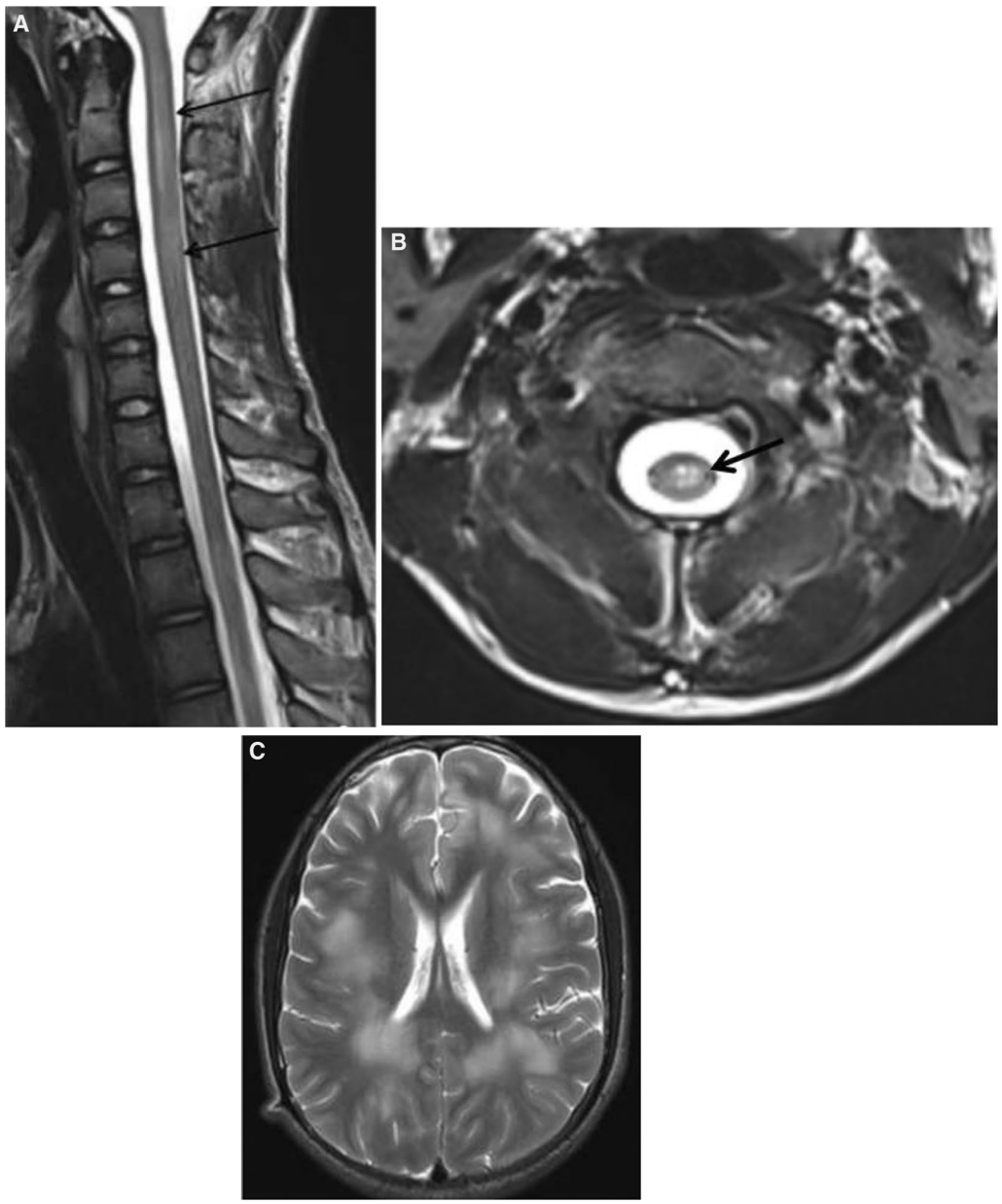


Fig. 2 Acute disseminated encephalomyelitis (ADEM) in a 16-year-old male with a recent history of short febrile illness followed by acute quadriparesis. Sagittal T2W image of the cervicothoracic spine (**A**) demonstrates multiple poorly defined short segment areas of hyperintensity involving the cervical and thoracic spinal cord (arrows). Axial T2W image of the cervical cord (**B**) shows that the lesion is involving less than two-thirds cross-section of the spinal cord (arrow). Axial T2W image of the brain (**C**) of the same patient reveals asymmetrical confluent areas of altered hyperintense signal in bilateral periventricular and subcortical white matter

Systemic inflammatory and immune-mediated disorders

Systemic lupus erythematosus (SLE), Sjögren syndrome, and sarcoidosis are the three common multisystemic inflammatory disorders affecting the spinal cord [3]. Central nervous system involvement including the spinal cord is uncommon in these disorders. Imaging morphology is nonspecific, and the diagnosis is established by considering the history and prior non-neurologic clinical manifestations. Imaging with MR shows a

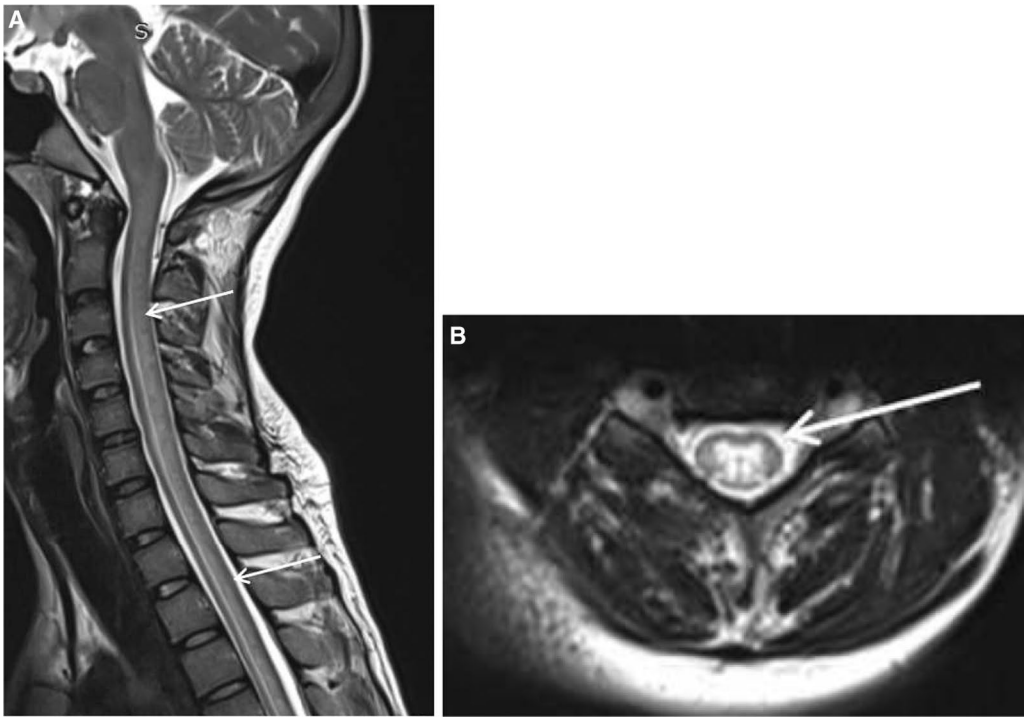


Fig. 3 Idiopathic long segment acute transverse myelitis in a 15-year-old patient with an acute history of backache and paraplegia. Sagittal T2W image of the cervicothoracic spine (**A**) demonstrates an extensive long segment area of hyperintensity involving the cervical and upper thoracic spinal cord (arrows) with associated cord swelling seen in the cervical region. Axial T2W image (**B**) shows that the altered hyperintense signal is centrally placed and appears to involve more than two-thirds cross-sectional area of the spinal cord (arrow)

long-segment intramedullary T2 hyperintense area with variable enhancement and without significant expansion [3]. The distinctive features favouring neurosarcoidosis include dorsal spinal cord predominance, leptomeningeal enhancement, and the trident sign – crescentic-shaped subpial enhancement of the posterior cord with additional subtle enhancement in the region of the central canal [18].

Infectious myelitis

Infectious myelopathy can be caused by various viral, bacterial, and fungal pathogens. MRI shows nonspecific diffuse non-expansile T2 hyperintense signal [4, 8].

Tubercular myelitis

Compressive myelopathy due to tubercular spondylodiscitis is a common entity; however, isolated intramedullary involvement by tuberculosis is relatively rare. There are various manifestations of tubercular myelitis which include acute myelitis, longitudinally extensive transverse myelitis, radiculomyelitis, arachnoiditis, and tuberculomas (intramedullary or intradural-extramedullary) [19]. Both acute partial transverse myelitis and longitudinally extensive transverse myelitis have been described in patients with tuberculous meningitis (Fig. 4). The latter is indistinguishable from NMOSD through imaging, hence necessitating the need for relevant clinical and laboratory details. The most commonly affected areas include the thoracic and cervical segments of the cord [20]. Intradural extramedullary tubercular spinal granulomas without bony involvement and intramedullary spinal tuberculomas are rare and show morphological characteristics similar to intracranial tuberculomas i.e. iso-intense on T1W and iso to hypointense on T2W with ring or nodular enhancement on post-contrast images (Fig. 5). Intramedullary granulomas may show fusiform cord swelling with surrounding T2 hyperintense edema [4]. Spinal arachnoiditis is a frequent presentation in these infections, which shows exudates and clumping of cauda equina nerve roots (along with resultant empty thecal sac) which may show enhancement on post-contrast images [19].

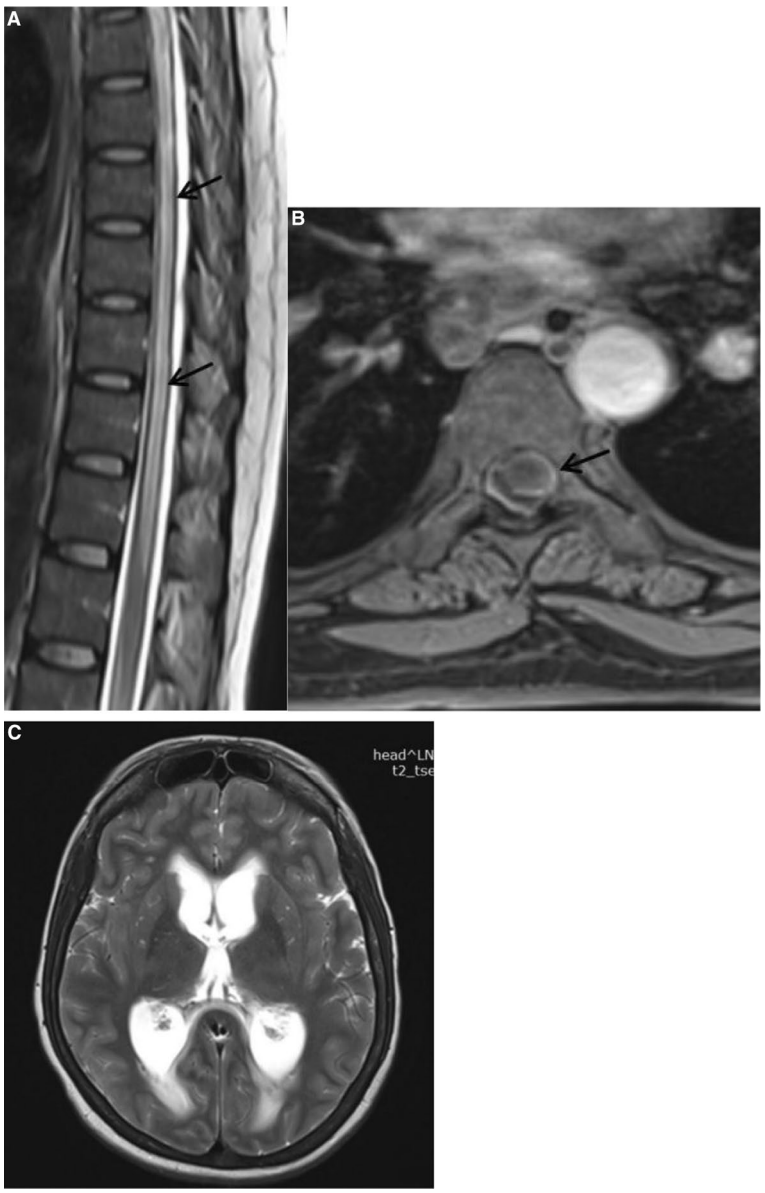


Fig. 4 Longitudinally extensive transverse myelitis due to tuberculosis in a 31-year-old female with a past history of treated tubercular lymphadenitis who presented to emergency with fever, altered sensorium, and paraparesis. CSF ADA levels were raised, measuring 24 IU/l (cut off: > 10 IU/l). Sagittal T2W image of dorsal spine (**A**) demonstrates a well-defined long segment area of hyperintensity (arrows) spanning the thoracic spinal cord. Axial post-contrast image (**B**) showing interrupted areas of subtle peripheral enhancement in the involved part of cord (arrow). Axial T2W image (**C**) of the brain shows obstructive hydrocephalus with dilated bilateral lateral and third ventricles and periventricular ooze due to tubercular meningitis

Human immunodeficiency virus (HIV) myelopathy

Myelopathy is a late-onset complication of HIV infection. MR can demonstrate varying appearances ranging from normal to diffuse T2 hyperintensity within the central spinal cord with accompanying cord atrophy. Additional findings include symmetrical non-enhancing T2 hyperintense areas in the dorsal columns of the spinal cord similar to subacute combined degeneration, although the signal abnormality is typically limited to the thoracic spine [20].

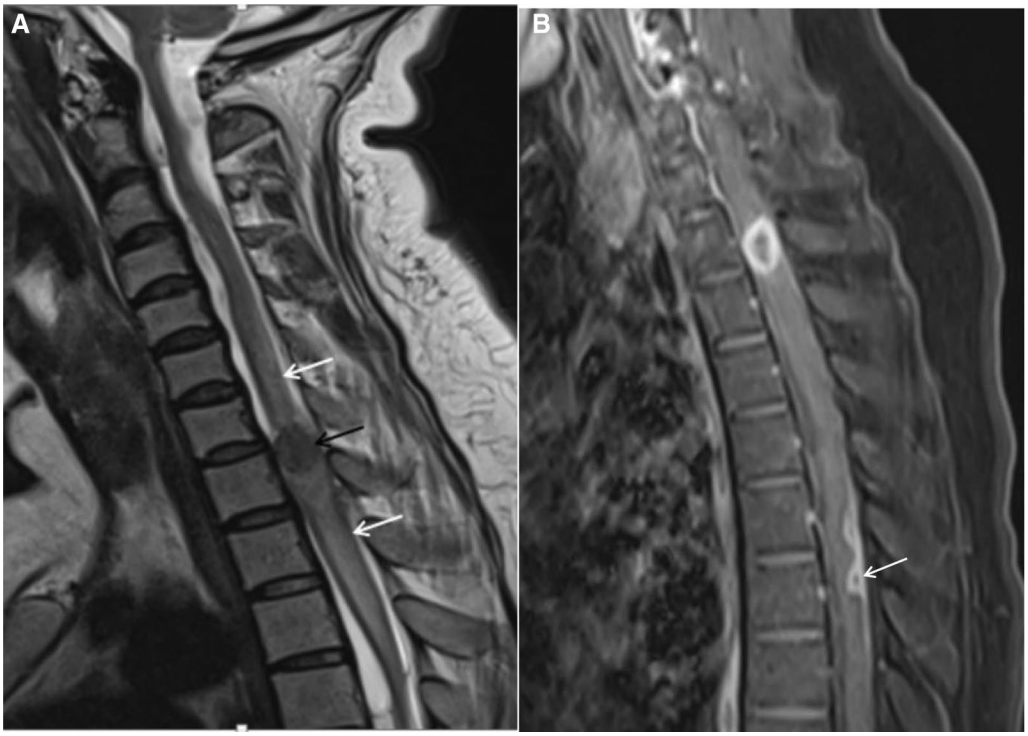


Fig. 5 Intramedullary tuberculoma in a 35-year-old female with pulmonary tuberculosis and complaints of progressive paraparesis. Sagittal T2W image of cervico-thoracic spine (**A**) demonstrates a well-defined round to oval hypointense focal lesion (black arrow) within the upper thoracic spinal cord with perilesional edema (white arrow) extending craniocaudal in the cord. Mild swelling of the cord is also seen. The lesion shows thick ring enhancement on post-contrast sagittal T1 fat saturated image (**B**). Similar smaller lesions are also seen caudally to the above-mentioned lesion (arrow in **B**)

Ischemic myelopathy

Spinal cord ischemia and infarction usually have an acute presentation, with symptoms onset and progression in less than 4 h [2]. It can be arterial or venous, with the former being more common. Multiple radiculomedullary arteries form the anterior and posterior spinal arteries perfusing the anterior two-thirds and posterior one-third of the spinal cord, respectively. Ischemia most commonly affects the territory of the anterior spinal artery and typically affects the lower thoracic cord [2]. Underlying aetiologies include severe atherosclerotic aortic disease, aortic dissection or aneurysm, and aortic surgery/stenting. On MR imaging, characteristic findings include long segment T2 hyperintense signal within the central gray matter, producing an "H-shaped" or "butterfly-shaped" pattern with corresponding restricted diffusion on diffusion-weighted images [2]. Watershed territory infarction may cause abnormalities in the ventral horns of the gray matter only, producing the typical "snake-eyes" or "owl's-eyes" appearance [21]. Imaging morphology of long segment involvement, central gray matter predominance, and absence of expansion, together with the hyperacute onset of symptoms, are highly suggestive of ischemia and help to discriminate it from other causes of acute myelopathy [2, 22].

Spinal vascular malformation

The most common vascular malformation is a dural arteriovenous fistula (d AVF), which represents approximately 80% of all spinal vascular malformations [23]. This

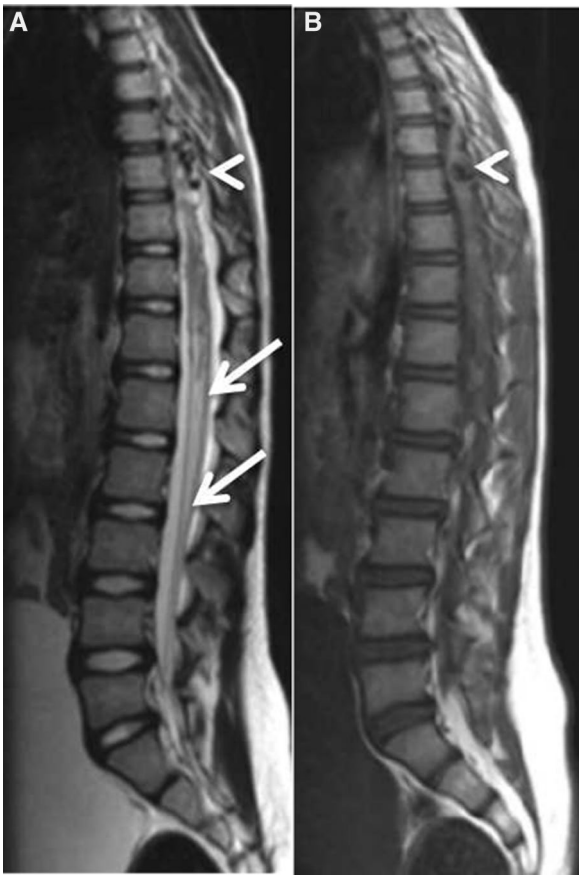


Fig. 6 Spinal dural arteriovenous malformation causing myelopathy. Sagittal T2W (**A**) and T1W (**B**) images showing long segment intramedullary altered signal intensity in the lower dorsal cord till conus medullaris appearing hyperintense on T2 W image (arrows in **A**) with serpentine flow voids at D8–D9 vertebral levels (arrowheads)

comprises an abnormal direct communication between the dural artery and vein of a nerve root sleeve. It is usually prevalent in the elderly male population and presents with nonspecific symptoms overlapping with spondylosis or polyneuropathy. A dural arteriovenous fistula is most commonly found at the mid and lower spinal levels, from T4 to L3 [2]. MRI characteristically shows numerous serpentine flow voids on the dorsal surface of the cord due to associated venous engorgement, which shows marked post-contrast enhancement. The spinal cord demonstrates long segment intramedullary T2 hyperintensity due to edema caused by venous hypertension often extending to the conus medullaris (Fig. 6). Ill-defined parenchymal contrast enhancement of the spinal cord can be seen. The gold standard study for localization of fistula and demonstration of arterial supply is digital subtraction angiography. MR angiography, however, can be useful in localizing the fistula site, allowing for a more focused catheter angiogram [2].

Intramedullary arteriovenous malformation (AVM) consists of an arteriovenous capillary nidus supplied by an enlarged feeding artery and drained via a tumid venous plexus. AVMs demonstrate intramedullary serpiginous flow voids and a short segment of signal change within the cord (representing the nidus) on T2W MRI, which shows enhancement on post-contrast images [2, 3]. If there is venous congestion, cord edema is seen as ill-defined intramedullary T2 hyperintensity with cord expansion. In contrast to AVF, these lesions more commonly show haemorrhage, which is a useful discriminating feature on MRI. Haemorrhage may be either intramedullary or subarachnoid in location and shows variable signal intensities depending upon the age of blood products [23].

Metabolic causes

Subacute combined degeneration of the spinal cord

This condition is the most common presentation of spinal cord involvement due to metabolic derangements and affects the dorsal columns, hence presenting clinically with gait ataxia and loss of vibration and proprioception in the extremities [8]. Most of the cases invariably occur due to Vitamin B12 deficiency [8]. Other aetiologies for this manifestation include copper deficiency and nitric oxide inhalation. MR imaging shows almost exclusive involvement of the dorsal columns in the cervical and thoracic spinal cord which show a non-expansile, long segment T2 hyperintensity producing a characteristic reversed V sign on axial images (Fig. 7) [24].

Neoplastic causes

Intramedullary neoplastic lesions can manifest as focal T2 hyperintensity within the spinal cord. Typically, expansion of the spinal cord on imaging and a subacute to chronic onset distinguish neoplastic from nonneoplastic conditions [25]. Astrocytoma, ependymoma, and hemangioblastoma are the common lesions in this group. Less commonly, lymphoma and metastases can also manifest as intramedullary lesions.

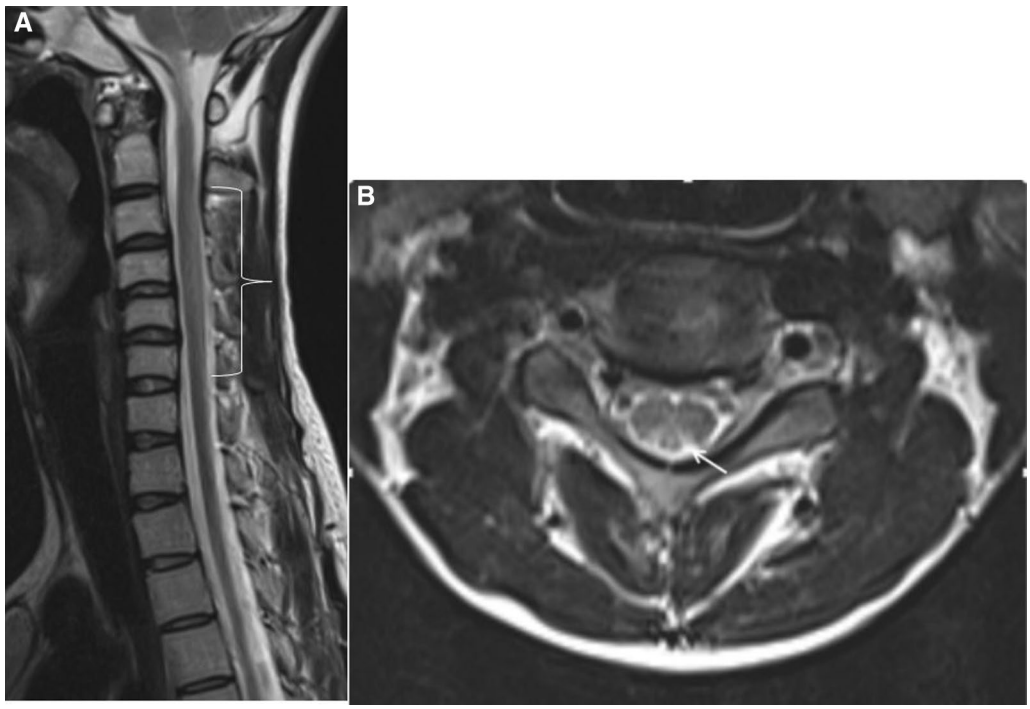


Fig. 7 Subacute combined degeneration in a 21-year-old male with a progressive history of sensory deficit and gait disturbance for 2 months who subsequently showed low serum vitamin B12 levels. Sagittal T2W image (**A**) demonstrates a long segment hyperintense signal within the posterior aspect of the cervical cord extending from C3 to C6 (bracket). Axial T2W image (**B**) shows hyperintensity involving the dorsal columns with the typical inverted V pattern (arrow)

On MR imaging, location along the cord, the length of involvement, and enhancement characteristics are valuable for determination of the plausible causes [8]. Astrocytomas and hemangioblastomas more commonly affect the thoracic cord, whereas ependymomas and metastasis commonly affect the cervical cord. Myxopapillary ependymoma is typically restricted to the filum terminale with rare involvement of the conus medullaris. Long segment involvement is seen in astrocytomas (4–7 vertebral bodies) and ependymomas (up to 4 vertebral bodies), while hemangioblastomas and metastases typically involve short segments of the cord [8]. Ependymomas typically show homogeneous enhancement as opposed to astrocytomas which exhibit more heterogeneous enhancement. Additional differentiating features of ependymoma include the presence of peritumoral cysts, hemorrhage, and identification of the hemosiderin "cap sign", consisting of a rim of T2 hypointensity at one or both poles, which occurs in about one-third of cases (Fig. 8) [25]. Peculiar findings of hemangioblastomas include the presence of a feeding vessel, detectable as a flow void on T2W images, and a focus of intense nodular enhancement on post-contrast images. Metastases merit consideration when dealing with multiple and short segment enhancing lesions [8].

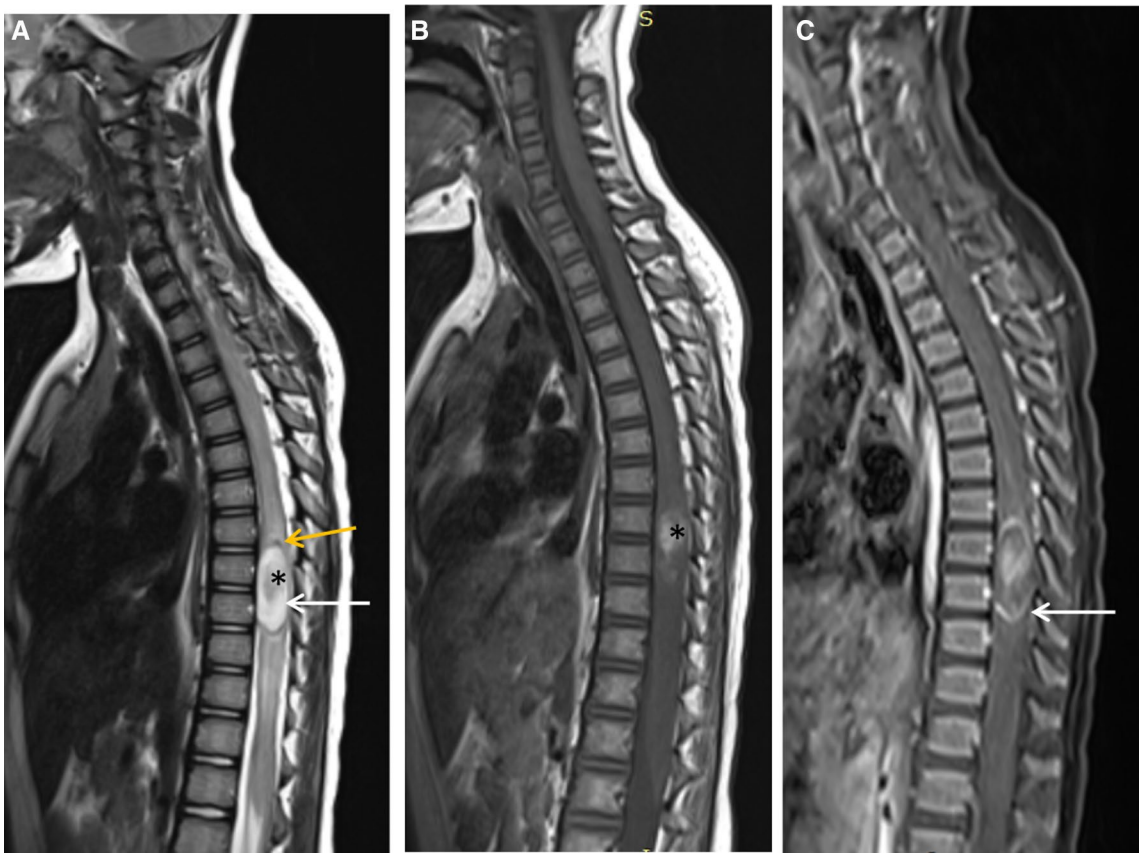


Fig. 8 Intramedullary ependymoma in a 6-year-old girl. Sagittal T2W (**A**) and T1W (**B**) images demonstrate a well-marginated oval-shaped lesion in the dorsal spinal cord (white arrow) extending from D8–D9 vertebral levels with associated cord expansion and perilesional edema. The lesion appears hyperintense on the T2W image with hypointense areas in the center (asterisk) which also shows a hyperintense signal on the T1W (**B**) image likely due to intratumoral hemorrhage. A T2 hypointense rim noted at superior and inferior poles of the lesion represents the hemosiderin cap (yellow arrow). Sagittal T1 fat saturated post-contrast image (**C**) demonstrates mild peripheral enhancement in the lesion (arrow)

Miscellaneous causes

Guillain–Barré syndrome (GBS)

GBS is a post-infectious/inflammatory autoimmune disease typically involving multiple peripheral nerves, most commonly the ventral nerve roots arising at the cauda equina. Although, this is essentially radiculopathy, ascending myelopathic changes can occur in some cases. CSF findings typically reveal albumin-cytologic dissociation, referring to an elevation of CSF protein without pleocytosis [26]. The clinical history of ascending paralysis and areflexia characteristically occurring post-infection or a vaccination along with typical CSF findings and electrophysiological examination can aid in diagnosis. Characteristic findings on MR imaging include thickening and contrast enhancement of conus medullaris and cauda equina nerve roots (predominantly anterior) (Fig. 9) [26]. Sometimes the more cephalad ventral gray matter horns and nerve roots may show enhancement.

Motor neuron disease

This consists of an uncommon group of fatal progressive neurodegenerative diseases which includes primary

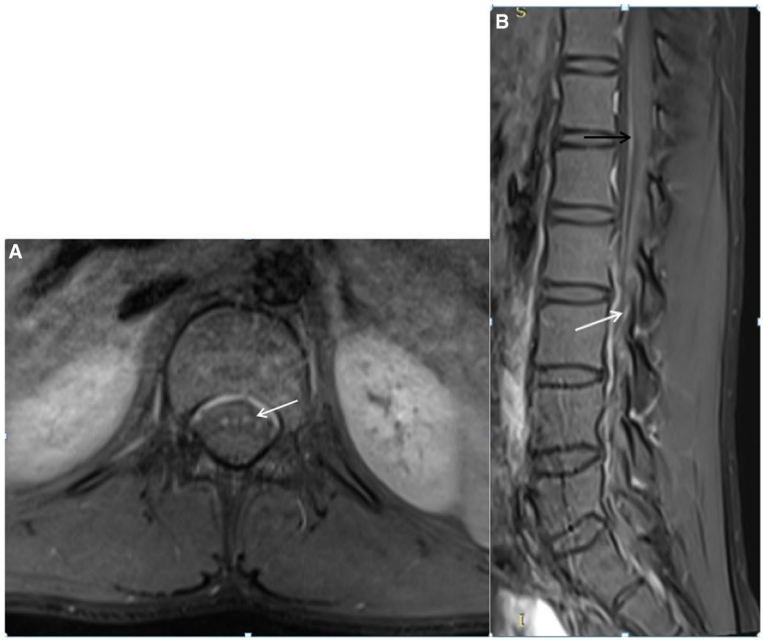


Fig. 9 Guillain–Barre's syndrome in a 16-year-old male who presented with acute ascending flaccid paralysis. Axial post-contrast image (**A**) at the level of conus medullaris demonstrates abnormal enhancement of the ventral nerve roots (arrow). Sagittal post-contrast image through the lower thoracic and lumbar spine (**B**) shows abnormal enhancement of nerve roots (white arrow) and ventral horns of the spinal cord (black arrow)

lateral sclerosis, spinocerebellar ataxia, iron neurodegeneration, Friedreich ataxia, and amyotrophic lateral sclerosis (ALS). ALS is the most common type of motor neuron disease. Typical findings comprise of long segment T2 hyperintensity involving the anterolateral columns with or without associated spinal cord atrophy. Associated intracranial extension of the abnormal signal along the corticospinal tracts can also be observed in some cases [27].

Radiation myelitis

Radiation myelitis presents as slowly progressive myelopathy with a variable latent period. Imaging characteristically demonstrates extensive long-segment T2 hyperintensity involving the irradiated field. A variable degree of enhancement may be appreciated in the acute stage. In the chronic stage, features of atrophy predominate with or without cystic necrosis [28]. Accompanying fatty bone marrow replacement in the adjacent vertebral bodies further increases the diagnostic confidence [28].

Myelopathy with normal MRI

Findings of normal spinal MRI in settings of clinically suspected acute myelopathy are not uncommon. There are several explanations for this: (1) the syndrome is not myelopathy rather an inflammatory radiculopathy; Guillain–Barre´s syndrome may be mistaken as myelitis, especially considering the abnormal CSF protein concentration and ascending symptoms that may mimic those seen in myelitis. (2) MRI performed during the convalescence period (if scanned after recovery from an acute attack of transverse myelitis and after resolution of spinal cord changes). (3) Many cases of acute myelitis/myelopathy may not demonstrate any altered intramedullary signal intensity. Newer MR techniques like tractography and fractional anisotropy have shown promising results in early detection of cord changes as compared to conventional MRI and may provide valuable information in such scenarios [15, 29]. History and clinical features should be reviewed carefully and correlated with CSF findings for arriving at a specific diagnosis. In some cases, MRI brain may be abnormal and that may help formulate the differential diagnosis [29].

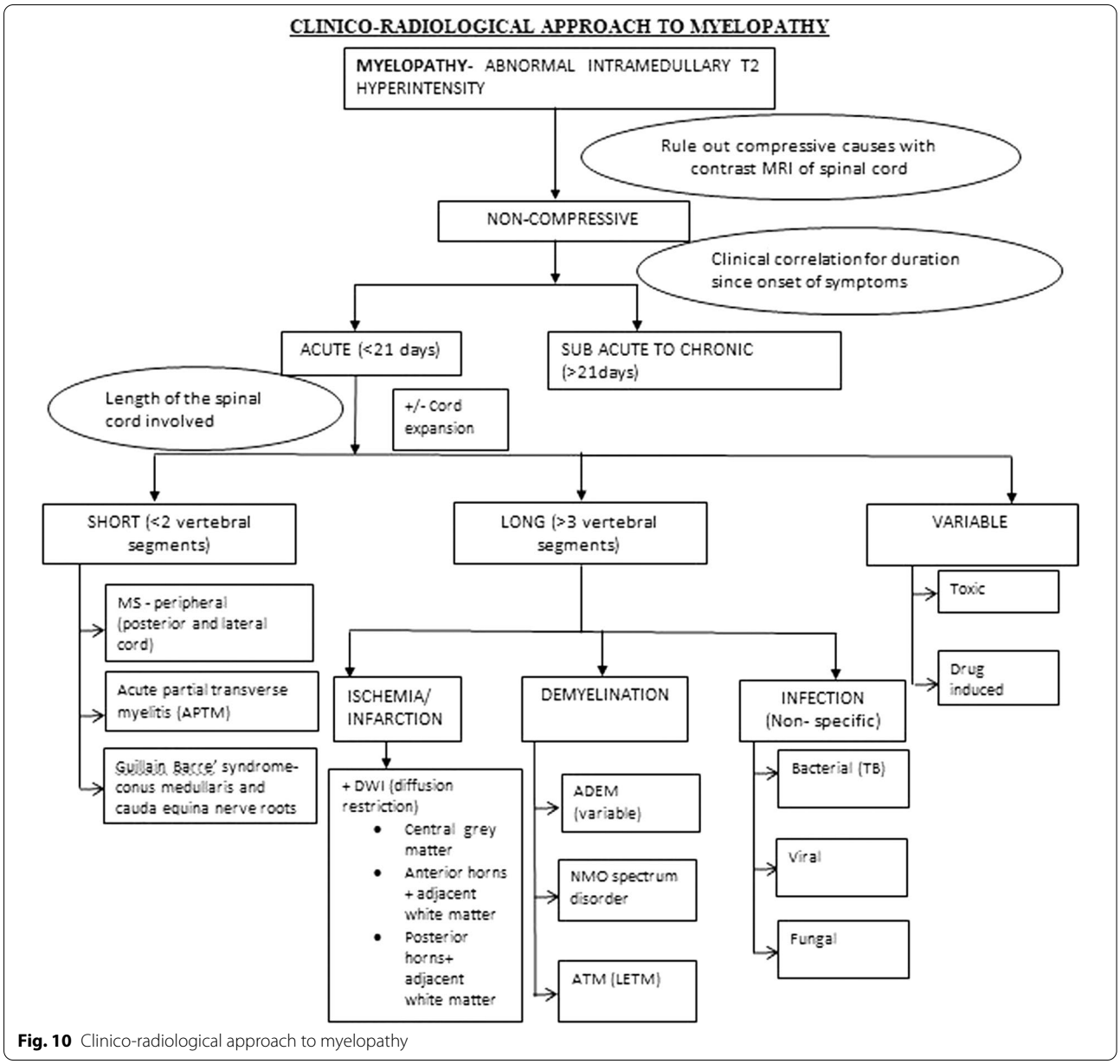


Fig. 10 Clinico-radiological approach to myelopathy

Conclusions

Imaging plays a critical role in the evaluation of clinically suspected cases of myelopathy, and MR imaging (with or without contrast) remains the preferred modality. Compressive causes must be excluded as a cause of myelopathy. As imaging morphology of non-compressive myelopathy is often nonspecific, careful attention to the patient's history, time course of presentation, and laboratory findings are necessary. Noncompressive myelopathy can broadly be classified into acute and non-acute onset and is further categorized by the distribution of the signal abnormalities, including length of cord involvement, specific tract involvement, enhancement pattern, and the region of the spinal cord that is affected. The algorithmic approach provided here (Fig. 10) is extremely useful in routine radiology practice. Knowledge of common and uncommon causes of myelopathy and identification of a few specific imaging features can help the radiologist in narrowing the differential diagnosis or establish a definitive diagnosis enabling appropriate clinical management.

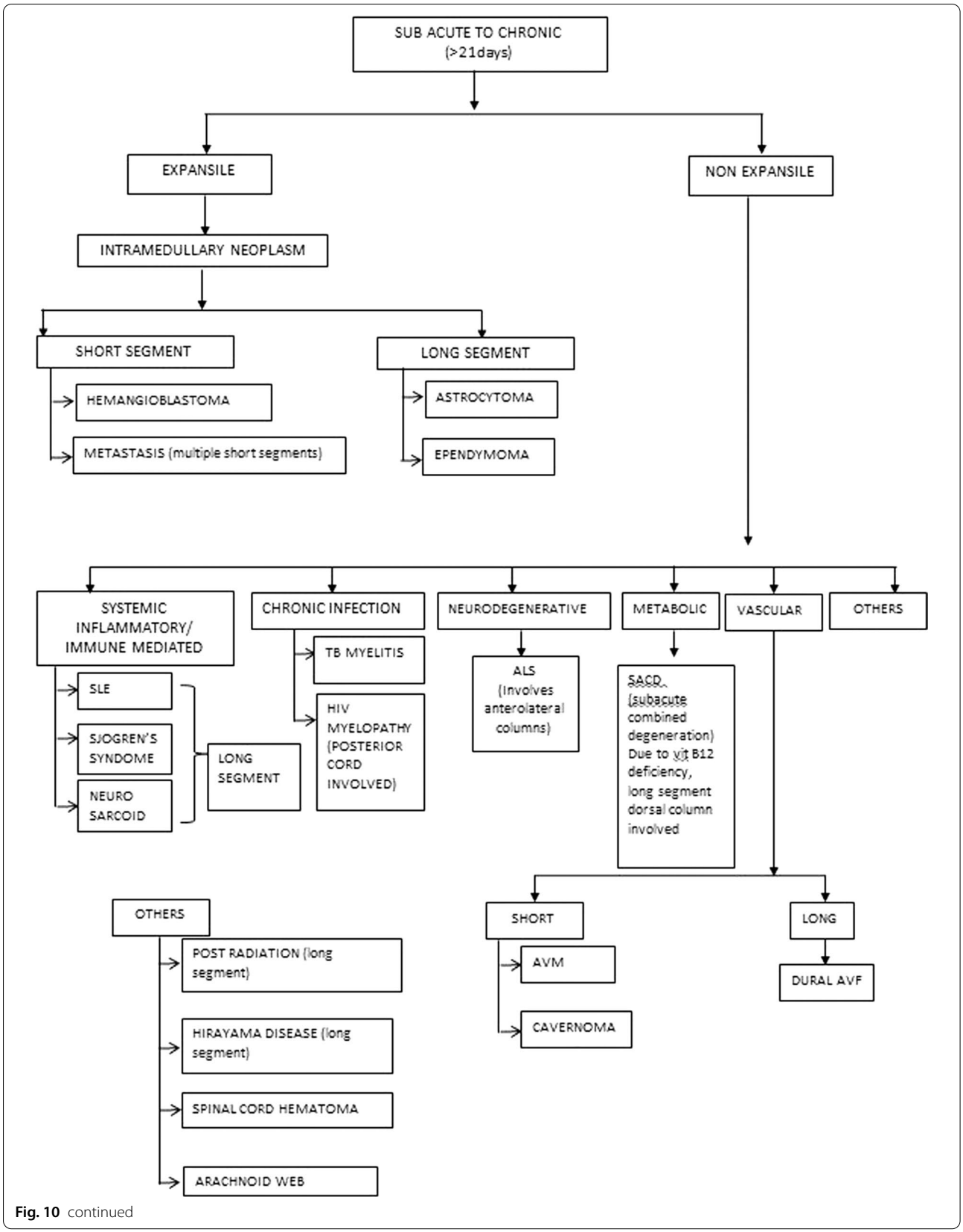


Fig. 10 continued

Abbreviations
MRI: Magnetic Resonance Imaging; MS: Multiple Sclerosis; ADEM: Acute Disseminated Encephalomyelitis; NMOSD: Neuromyelitis Optica Spectrum Disorder; TM: Transverse Myelitis; CSF: Cerebrospinal Fluid; HIV: Human Immunodeficiency Virus; GBS: Guillain-Barré Syndrome; ALS: Amyotrophic Lateral Sclerosis.

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Authors' contributions
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Availability of data and materials
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Declarations

Ethics approval and consent to participate
The need for approval was waived for this review by the local committee.

Consent for publication
A written informed consent for the publication of the images in this review was obtained from all patients.

Competing interests
The authors declare that they have no competing interests.

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