American College of Radiology
ACR Appropriateness Criteria®
Myelopathy

Variant 1: **Acute onset myelopathy. Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI spine area of interest without and with IV contrast	Usually Appropriate	O
MRI spine area of interest without IV contrast	Usually Appropriate	O
CT myelography spine area of interest	May Be Appropriate	Varies
CT spine area of interest with IV contrast	May Be Appropriate	Varies
CT spine area of interest without IV contrast	May Be Appropriate	Varies
Arteriography spine area of interest	Usually Not Appropriate	Varies
Radiography spine area of interest	Usually Not Appropriate	Varies
MRA spine area of interest with IV contrast	Usually Not Appropriate	O
MRA spine area of interest without and with IV contrast	Usually Not Appropriate	O
MRA spine area of interest without IV contrast	Usually Not Appropriate	O
MRI spine area of interest with IV contrast	Usually Not Appropriate	O
CT spine area of interest without and with IV contrast	Usually Not Appropriate	Varies
CTA spine area of interest with IV contrast	Usually Not Appropriate	Varies

Variant 2: **Chronic or progressive myelopathy. Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI spine area of interest without and with IV contrast	Usually Appropriate	O
MRI spine area of interest without IV contrast	Usually Appropriate	O
CT myelography spine area of interest	May Be Appropriate	Varies
CT spine area of interest with IV contrast	May Be Appropriate	Varies
CT spine area of interest without IV contrast	May Be Appropriate	Varies
Arteriography spine area of interest	Usually Not Appropriate	Varies
Radiography spine area of interest	Usually Not Appropriate	Varies
MRA spine area of interest with IV contrast	Usually Not Appropriate	O
MRA spine area of interest without and with IV contrast	Usually Not Appropriate	O
MRA spine area of interest without IV contrast	Usually Not Appropriate	O
MRI spine area of interest with IV contrast	Usually Not Appropriate	O
CT spine area of interest without and with IV contrast	Usually Not Appropriate	Varies
CTA spine area of interest with IV contrast	Usually Not Appropriate	Varies

MYELOPATHY

Expert Panel on Neurological Imaging: Vikas Agarwal, MD[a]; Lubdha M. Shah, MD[b]; Matthew S. Parsons, MD[c]; Daniel J. Boulter, MD[d]; R. Carter Cassidy, MD[e]; Troy A. Hutchins, MD[f]; Jamlik-Omari Johnson, MD[g]; A. Tuba Kendi, MD[h]; Majid A. Khan, MBBS, MD[i]; David S. Liebeskind, MD[j]; Toshio Moritani, MD, PhD[k]; A. Orlando Ortiz, MD, MBA[l]; Charles Reitman, MD[m]; Vinil N. Shah, MD[n]; Laura A. Snyder, MD[o]; Vincent M. Timpone, MD[p]; Amanda S. Corey, MD.[q]

Summary of Literature Review

Introduction/Background

Myelopathy refers to any pathologic process affecting the spinal cord. It is a clinical diagnosis based on signs and symptoms of spinal cord dysfunction [1]. Myelopathy can be due to primary intrinsic disorders of the spinal cord and include neoplastic, infectious, inflammatory, neurodegenerative, vascular, nutritional, and idiopathic disorders [2]. More commonly, however, myelopathy is due to secondary conditions, which result in extrinsic compression of the spinal cord. The most frequently encountered cause of extrinsic compression of the spinal cord in adults is degenerative disease of the cervical and thoracic spine [3]. Other causes of myelopathy from external spinal cord compression include bone metastases and blunt or penetrating trauma. A variety of cysts and benign neoplasms can also compress the cord; they tend to arise within the intradural compartment. The most common of these are nerve sheath tumors, meningiomas, and arachnoid adhesions/cysts [4-10].

Clinically, the diagnosis of myelopathy depends on the localization of the neurological finding to the spinal cord, rather than the brain or peripheral nervous system, and then to a particular segment of the spinal cord [11]. Although the causes of myelopathy may be many, the acuity of presentation and symptom onset provides the clinical team with a practical approach to the differential diagnosis [1,12]. Myelopathy is considered acute if symptoms begin abruptly or have an onset of days to weeks. Myelopathy with a time course of months to years is considered chronic or progressive.

Imaging plays a crucial role in refining the differential diagnosis. Historically, radiological evaluation of myelopathic patients consisted of positive contrast myelography. Later, this evaluation was supplemented by CT and CT myelography. MRI is now the mainstay in the evaluation of myelopathy because of its superb contrast resolution of the spinal cord [10,13-15].

For the purposes of this discussion, myelopathy secondary to trauma is excluded (see the ACR Appropriateness Criteria® topic on "Suspected Spine Trauma" [16]).

Special Imaging Considerations

Although history and physical examination can help localize the myelopathic level, it may be beneficial to study the entire spine, even in the setting of a localized myelopathic level. In certain cases, brain MRI may be a useful adjunct diagnostic test [17]. Newer imaging techniques, such as spinal cord diffusion tensor imaging, appear promising to further interrogate spinal cord injury at a microstructural level [18-21].

CT myelography is performed in conjunction with fluoroscopic myelography. For this document, the procedure term "CT myelography" is used to guide the referral to the radiologist. The ultimate judgment regarding the propriety of any specific procedure, lumbar versus cervical puncture route, amount of contrast, and the extent and modality of imaging coverage must be made by the radiologist, with appropriate documentation and coding [22].

[a]University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania. [b]Panel Chair, University of Utah, Salt Lake City, Utah. [c]Panel Vice-Chair, Mallinckrodt Institute of Radiology, Saint Louis, Missouri. [d]The Ohio State University Wexner Medical Center, Columbus, Ohio. [e]UK Healthcare Spine and Total Joint Service, Lexington, Kentucky; American Academy of Orthopaedic Surgeons. [f]University of Utah Health, Salt Lake City, Utah. [g]Emory University, Atlanta, Georgia. [h]Mayo Clinic, Rochester, Minnesota. [i]Johns Hopkins Hospital, Baltimore, Maryland. [j]University of California Los Angeles, Los Angeles, California; American Academy of Neurology. [k]University of Michigan, Ann Arbor, Michigan. [l]Jacobi Medical Center, Bronx, New York. [m]Medical University of South Carolina, Charleston, South Carolina; North American Spine Society. [n]University of California San Francisco, San Francisco, California. [o]Barrow Neurological Institute, Phoenix, Arizona; Neurosurgery expert. [p]University of Colorado School of Medicine, Anschutz Medical Campus, Aurora, Colorado. [q]Specialty Chair, Atlanta VA Health Care System and Emory University, Atlanta, Georgia.

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Initial Imaging Definition

Initial imaging is defined as imaging at the beginning of the care episode for the medical condition defined by the variant. More than one procedure can be considered usually appropriate in the initial imaging evaluation when:

- There are procedures that are equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient's care)

OR

- There are complementary procedures (ie, more than one procedure is ordered as a set or simultaneously in which each procedure provides unique clinical information to effectively manage the patient's care).

Discussion of Procedures by Variant

Variant 1: Acute onset myelopathy. Initial imaging.

The body regions covered in this clinical scenario are cervical, thoracic, and lumbar spine. These body regions might be evaluated separately or in combination as guided by physical examination findings, patient history, and other available information, including prior imaging.

Acute myelopathy can be subdivided into noninflammatory and inflammatory causes. Noninflammatory conditions include extrinsic compression of the spinal cord, vascular pathologies, and trauma. Inflammatory conditions include demyelinating diseases (ie, multiple sclerosis), systemic inflammatory diseases, and infection.

For the purposes of this discussion, myelopathy secondary to trauma is excluded (see the ACR Appropriateness Criteria® topic on "Suspected Spine Trauma" [16]). Vertebral fracture in the setting of weakened bone (eg, osteoporotic or pathologic fracture) with retropulsion can lead to myelopathy, even in minor trauma or no obvious history of trauma (see the ACR Appropriateness Criteria® topic on "Management of Vertebral Compression Fractures" [23]).

All patients with acute onset myelopathy require evaluation for extrinsic compression of the spinal cord [3]. In the acute setting, extrinsic compression of the spinal cord is most commonly caused by degenerative disease (spondylotic myelopathy) and is more prevalent in the cervical spine. Factors contributing to spondylotic myelopathy include spinal degenerative changes, disc herniations, and malalignment. These findings may be accentuated in the presence of congenitally short pedicles. Other causes of extrinsic compression of the spinal cord include pathology involving the epidural compartment (abscess or hematoma) [24]. In patients who have undergone spinal surgery, extrinsic compression of the spinal cord can develop throughout the postoperative course and may be secondary to seromas, pseudomeningoceles, hematomas, and/or epidural abscesses [25]. Primary or metastatic tumors of the extradural and intradural extramedullary spaces encroaching upon the spinal canal can cause extrinsic compression of the spinal cord, resulting in not only acute but also progressive myelopathy (see Variant 2). Please see the ACR Appropriateness Criteria® topic on "Follow-up of Malignant or Aggressive Musculoskeletal Tumors" [26] for further details on extradural tumors.

Although infrequent, spinal cord ischemia can result in acute onset myelopathy and in adults is most commonly the result of atheromatous disease or as a complication of aortic surgery [3]. Other pathologies that may predispose patients to developing spinal cord ischemia include systemic hypotension, thoracoabdominal aneurysms or dissection, sickle cell disease, and spinal arteriovenous malformations (AVMs) [27,28]. Very rarely, patients may develop hematomyelia and subsequently acute myelopathy because of an intrameduallary AVM or spinal artery aneurysm rupture [29,30]. Acute ischemic myelopathy can also develop in the setting of fibrocartilaginous embolic disease [31]. Depending on the level(s) of the spinal cord involved, patients will typically develop acute paraparesis or quadriparesis. Inflammatory conditions that can result in acute myelopathy include demyelinating diseases such as multiple sclerosis (MS), neuromyelitis optica (NMO), and acute disseminated encephalomyelitis (ADEM); systemic inflammatory conditions such as systemic lupus erythematous, Sjogren syndrome, mixed connective tissue disorder, Behcet disease, and sarcoidosis; and infectious diseases [8].

MRI Spine

MRI is useful for evaluation of the spinal cord when investigating the etiology of acute myelopathy [32]. MRI has superior soft-tissue resolution and multiplanar capability, making it ideal for evaluation of the spinal canal and its contents as well as the surrounding osseous and soft-tissue structures [13-15,33-36].

Intramedullary cord signal changes on MRI in patients with spondylotic myelopathy represent prognostic factors for neurosurgical outcome [21,37-41]. Intravenous (IV) contrast is typically not required for the diagnosis of spondylotic myelopathy, but characteristic patterns of enhancement can be seen immediately at and below a level of stenosis [42,43].

In patients who have undergone spinal surgery, complications in the early postoperative setting (eg, hematoma) can result in extrinsic compression of the spinal cord and are best evaluated using MRI without and with IV contrast [44].

In cases in which spinal cord ischemia is suspected as the cause for acute myelopathy, MRI without and with IV contrast is useful in cases where spinal cord ischemic is suspected as the cause for acute myelopathy [27,28,45-47]. Contrast enhancement is typically not seen in the early phase of acute ischemia and, if present, may suggest an alternative inflammatory or infectious etiology [48]. Diffusion-weighted imaging can show signal alteration in the spinal cord earlier after patient symptom onset compared with T2-weighted images [49,50]. As such, diffusion-weighted imaging should be included anytime there is concern for spinal cord ischemia [51].

When considering inflammatory or infections etiologies of myelopathy, visualization of the osseous spinal column as well as the spinal cord is useful and best accomplished noninvasively by MRI [4,7,45,52-60].

Demyelinating diseases such as MS and NMO can present as acute myelopathy. MS is the classic demyelinating disease and is characterized by lesions affecting the spinal cord (and brain) and clinical defects disseminated in space and time [61]. Spinal cord involvement is seen in 80% to 90% of patients with MS, most commonly affecting the cervical cord [62]. Patients with primary progressive MS tend to have more spinal cord involvement than patients with relapsing-remitting MS [12]. When myelopathy due to MS is suspected, MRI detection of spinal cord lesion(s) fulfills part of the 2016 Magnetic Resonance Imaging in MS (MAGNIMS) criteria [63]. NMO is a demyelinating condition characterized by optic neuritis and spinal cord lesions. Brain lesions are not as commonly encountered in NMO as in MS, so, when present, tend to predominate in regions around the third and fourth ventricles [64-66]. ADEM is a demyelinating condition that typically manifests as encephalopathy. Spinal cord involvement is present in approximately 25% of cases of ADEM. MRI of the spine is generally considered the reference standard for imaging of the spinal cord in cases of suspected demyelinating disease [63,67,68] in addition to excluding alternative etiologies. Contrast-enhanced imaging is recommended for initial diagnostic evaluation [69,70].

MRA Spine

There is no relevant literature regarding the use of MR angiography (MRA) in the initial imaging evaluation of acute onset myelopathy. In cases of spinal cord ischemia, MRA can be used to identify the artery of Adamkiewicz or vertebral artery dissection/occlusion and should be considered as a follow-up to MRI [12]. Although MRA can be performed for suspected spinal vascular malformations in patients with hematomyelia, conventional angiography remains necessary for complete lesion characterization [71].

CT Myelography Spine

CT myelography may be useful in this clinical setting to answer specific questions before surgical intervention [72,73]. In spondylotic myelopathy, conventional myelography can be used to diagnose severe canal stenosis [74]. MRI, however, is best for evaluation of the marrow and the spinal canal/spinal cord [13-15].

CT Spine

CT can depict bony encroachment on the spinal canal in cases of disc-osteophyte complexes as well as subluxation and compression of neural structures by herniated disc material with better resolution than with radiographs. For inflammatory or infectious processes, CT can be beneficial to evaluate the osseous structures and adjacent soft-tissue involvement [75]. Although CT demonstrates osseous integrity with excellent assessment of bone destruction, MRI provides better visualization of the marrow and the spinal cord [13-15]. CT of the spine is not useful in the initial evaluation of spinal cord ischemia [76].

CTA Spine

There is no relevant literature regarding the use of CT angiography (CTA) in the initial imaging evaluation of acute onset myelopathy. In cases of spinal cord ischemia, CTA can be used to identify the artery of Adamkiewicz or vertebral artery dissection/occlusion and should be considered as a follow-up to MRI [12].

Radiography Spine
There is no relevant literature supporting the use of radiographs as the initial imaging evaluation of acute onset myelopathy. Although radiographs may demonstrate bone destruction, CT provides better visualization of the osseous spine [13-15]. In spondylotic myelopathy, radiographs may depict osteophytic narrowing of the spinal canal, whereas conventional myelography can be used to diagnose severe canal stenosis [74]. MRI, however, is best for evaluation of the marrow and the spinal canal/spinal cord [13-15]. Lateral radiographs can be obtained as an adjunct to cross-sectional imaging to help assess alignment parameters and dynamic instability [77].

Arteriography
There is no relevant literature regarding the use of conventional arteriography in the initial imaging evaluation of acute onset myelopathy. Even in cases of spinal cord ischemia and suspected spinal vascular malformations, conventional arteriography of the spine is not useful for initial evaluation [71,76].

Variant 2: Chronic or progressive myelopathy. Initial imaging.

The body regions covered in this clinical scenario are cervical, thoracic, and lumbar spine. These body regions might be evaluated separately or in combination as guided by physical examination findings, patient history, and other available information, including prior imaging.

As with acute myelopathy, all patients with chronic or progressive myelopathy require evaluation for extrinsic compression of the spinal cord [3]. In the chronic or progressive setting, extrinsic compression of the spinal cord is most commonly due to degenerative disease (spondylotic myelopathy) and is more prevalent in the cervical spine. Factors contributing to spondylotic myelopathy include spinal degenerative changes, disc herniations, epidural lipomatosis, and malalignment. These findings may be accentuated in the presence of congenitally short pedicles. In patients who have undergone spinal surgery, extrinsic compression of the spinal cord can develop throughout the postoperative course and may be secondary to seromas, pseudomeningoceles, hematomas, and/or epidural abscesses [25]. Primary or metastatic tumors of the extradural and intradural extramedullary spaces encroaching upon the spinal canal can cause extrinsic compression of the spinal cord, resulting in progressive myelopathy as well as acute myelopathy (see Variant 1). Please see the ACR Appropriateness Criteria® topic on "Follow-up of Malignant or Aggressive Musculoskeletal Tumors" [26] for further details on extradural tumors. Infrequently, other rare conditions such as Hirayama disease (cervical flexion myelopathy), dorsal arachnoid webs, and ventral cord herniation can result in progressive myelopathy [78-80].

Once extrinsic compression of the spinal cord has been excluded, chronic or progressive myelopathy can be subdivided into non-neoplastic and neoplastic causes. Non-neoplastic causes include demyelinating diseases such as MS, NMO, and ADEM; metabolic derangements such as Vitamin B12 (cobalamin) deficiency, copper deficiency, and nitrous oxide inhalation; chronic infections including human T cell lymphotropic virus myelitis, tuberculosis, schistosomiasis, human immunodeficiency virus vacuolar myelopathy, and tertiary syphilis; prior radiation treatment; autoimmune causes including paraneoplastic myelopathy; and vascular abnormalities such as spinal dural AVM/fistulas. Neoplastic causes include primary and metastatic tumors of the spinal cord.

MRI Spine
MRI is useful for evaluation of the spinal cord when investigating the etiology of chronic or progressive myelopathy [32]. MRI has superior soft-tissue resolution and multiplanar capability, making it ideal for evaluation of the spinal canal and its contents as well as the surrounding osseous and soft-tissue structures [13-15,33-36].

The imaging changes in the spinal cord due to myelomalacia and gliosis are best discerned by MRI [81,82]. Intramedullary cord signal changes on MRI in patients with spondylotic myelopathy represent prognostic factors for neurosurgical outcome [21,37-41]. IV contrast is typically not required for the diagnosis of spondylotic myelopathy, but characteristic patterns of enhancement can be seen immediately at and below a level of stenosis [42,43].

In patients who have undergone spinal surgery, late complications (eg, adjacent level degenerative disease with spinal stenosis, recurrent disc herniation) can result in extrinsic compression of the spinal cord and are best evaluated using MRI without and with IV contrast [44].

Demyelinating diseases such as MS can present as subacute/chronic myelopathy. MS is the classic demyelinating disease and is characterized by lesions affecting the spinal cord (and brain) and clinical defects disseminated in space and time [61]. Spinal cord involvement is seen in 80% to 90% of patients with MS, most commonly affecting the cervical cord [62]. Patients with primary progressive MS tend to have more spinal cord involvement than

patients with relapsing-remitting MS [12]. When myelopathy due to MS is suspected, MRI detection of spinal cord lesion(s) fulfills part of the 2016 MAGNIMS criteria [63]. Other demyelinating processes such as NMO and ADEM can present as chronic myelopathy less commonly. In patients with chronic or progressive myelopathy, MRI of the spinal cord can identify spinal cord lesions suggestive of demyelinating disease in addition to excluding alternative etiologies. Contrast-enhanced imaging is recommended for initial diagnostic evaluation [69,70].

Metabolic causes of chronic or progressive myelopathy result in changes in the spinal cord known as subacute combined degeneration and are best evaluated with MRI [11,83,84]. Chronic infections can have a similar appearance [12].

Radiation-induced myelopathy is a rare dose-dependent complication that anatomically localizes to a prior radiation port [85]. Autoimmune myelitis includes paraneoplastic myelopathy [86,87]. MRI without and with IV contrast is useful to evaluate the spinal cord in these instances.

Vascular malformations can likewise present with chronic and slowly progressive myelopathy [88]. MRI without and with IV contrast is useful to demonstrate spinal cord edema caused by venous hypertension and enlarged veins along the dorsal surface of the spinal cord. There may be patchy intramedullary enhancement due to breakdown of the blood-cord barrier. In some cases, abnormal vasculature may be identified that may be useful to guide spinal arteriography and intervention [5,89-91].

Primary and metastatic tumors of the spinal cord very rarely cause myelopathy and are best evaluated on contrast-enhanced MRI of the spine [92-96]. The distinction of syrinx from tumor, location of small tumor nodules, extent of cyst, and distinction of nodule and cyst from edema are crucial in treatment planning for intramedullary disease and best delineated with MRI [9,97].

In cases in which MRI shows findings suspicious for arachnoid cyst/arachnoid web or ventral cord herniation, CT myelography can be performed for further evaluation [79,80]. Likewise, in cases in which there is clinical concern for positional myelopathy, MRI with flexion/extension can be performed as a follow-up [78].

MRA Spine
There is no relevant literature regarding the use of MRA in the initial imaging evaluation of chronic or progressive myelopathy. If MRI demonstrates findings concerning for an underlying vascular malformations, MRA can be performed as a follow-up to demonstrate abnormal vasculature that may be useful to guide spinal arteriography and intervention [5,89-91].

CT Myelography Spine
CT myelography may be useful in this setting to answer specific questions before surgical intervention [72,73]. In cases in which MRI shows findings suspicious for arachnoid cyst/arachnoid web or ventral cord herniation, CT myelography can be performed for further evaluation [79,80]. Likewise, in cases in which there is clinical concern for positional myelopathy, extension/flexion positional CT myelography can be performed as a follow-up [98]. In spondylotic myelopathy, conventional myelography can be used to diagnose severe canal stenosis [74]. MRI, however, is best for evaluation of the marrow and the spinal canal/spinal cord [13-15].

CT Spine
CT can depict bony encroachment on the spinal canal in cases of disc-osteophyte complexes as well as subluxation and compression of neural structures by herniated disc material with better resolution than with radiographs. Although CT demonstrates osseous integrity with excellent assessment of bone destruction, MRI provides better visualization of the marrow and the spinal cord [13-15]. It is therefore not useful in the initial evaluation of noncompressive etiologies of chronic or progressive myelopathy.

CTA Spine
There is no relevant literature regarding the use of CTA in the initial imaging evaluation of chronic or progressive myelopathy. CTA continues to make progress as a preangiographic tool for localization of spinal vascular malformations [99-102].

Radiography Spine
There is no relevant literature supporting the use of radiographs as the initial imaging evaluation of chronic or progressive myelopathy. Although radiographs may demonstrate bone destruction, CT provides better visualization of the osseous spine [13-15]. In spondylotic myelopathy, radiographs may depict osteophytic narrowing of the spinal canal, whereas conventional myelography can be used to diagnose severe canal stenosis [74]. MRI, however,

is best for evaluation of the marrow and the spinal canal/spinal cord [13-15]. Lateral radiographs can be obtained as an adjunct to cross-sectional imaging to help assess alignment parameters and dynamic instability [77].

Arteriography

There is no relevant literature regarding the use of conventional arteriography in the initial imaging evaluation of chronic or progressive myelopathy.

Summary of Recommendations

- **Variant 1**: MRI spine area of interest without and with IV contrast or MRI spine area of interest without IV contrast is usually appropriate for the initial imaging of patients with acute onset myelopathy. These procedures are equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient's care).
- **Variant 2**: MRI spine area of interest without and with IV contrast or MRI spine area of interest without IV contrast is usually appropriate for the initial imaging of patients with chronic or progressive myelopathy. These procedures are equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient's care).

Supporting Documents

The evidence table, literature search, and appendix for this topic are available at https://acsearch.acr.org/list. The appendix includes the strength of evidence assessment and the final rating round tabulations for each recommendation.

For additional information on the Appropriateness Criteria methodology and other supporting documents go to www.acr.org/ac.

Appropriateness Category Names and Definitions

Appropriateness Category Name	Appropriateness Rating	Appropriateness Category Definition
Usually Appropriate	7, 8, or 9	The imaging procedure or treatment is indicated in the specified clinical scenarios at a favorable risk-benefit ratio for patients.
May Be Appropriate	4, 5, or 6	The imaging procedure or treatment may be indicated in the specified clinical scenarios as an alternative to imaging procedures or treatments with a more favorable risk-benefit ratio, or the risk-benefit ratio for patients is equivocal.
May Be Appropriate (Disagreement)	5	The individual ratings are too dispersed from the panel median. The different label provides transparency regarding the panel's recommendation. "May be appropriate" is the rating category and a rating of 5 is assigned.
Usually Not Appropriate	1, 2, or 3	The imaging procedure or treatment is unlikely to be indicated in the specified clinical scenarios, or the risk-benefit ratio for patients is likely to be unfavorable.

Relative Radiation Level Information

Potential adverse health effects associated with radiation exposure are an important factor to consider when selecting the appropriate imaging procedure. Because there is a wide range of radiation exposures associated with different diagnostic procedures, a relative radiation level (RRL) indication has been included for each imaging examination. The RRLs are based on effective dose, which is a radiation dose quantity that is used to estimate population total radiation risk associated with an imaging procedure. Patients in the pediatric age group are at inherently higher risk from exposure, because of both organ sensitivity and longer life expectancy (relevant to the long latency that appears to accompany radiation exposure). For these reasons, the RRL dose estimate ranges for

pediatric examinations are lower as compared with those specified for adults (see Table below). Additional information regarding radiation dose assessment for imaging examinations can be found in the ACR Appropriateness Criteria® Radiation Dose Assessment Introduction document [103].

Relative Radiation Level Designations		
Relative Radiation Level*	**Adult Effective Dose Estimate Range**	**Pediatric Effective Dose Estimate Range**
O	0 mSv	0 mSv
☢	<0.1 mSv	<0.03 mSv
☢☢	0.1-1 mSv	0.03-0.3 mSv
☢☢☢	1-10 mSv	0.3-3 mSv
☢☢☢☢	10-30 mSv	3-10 mSv
☢☢☢☢☢	30-100 mSv	10-30 mSv
*RRL assignments for some of the examinations cannot be made, because the actual patient doses in these procedures vary as a function of a number of factors (eg, region of the body exposed to ionizing radiation, the imaging guidance that is used). The RRLs for these examinations are designated as "Varies."		

References

1. Kranz PG, Amrhein TJ. Imaging Approach to Myelopathy: Acute, Subacute, and Chronic. Radiol Clin North Am 2019;57:257-79.
2. Tettenborn B, Hagele-Link S. Spinal Cord Disorders. Eur Neurol 2015;74:141-6.
3. Bhattacharyya S. Spinal Cord Disorders: Myelopathy. Am J Med 2018;131:1293-97.
4. Candy S, Chang G, Andronikou S. Acute myelopathy or cauda equina syndrome in HIV-positive adults in a tuberculosis endemic setting: MRI, clinical, and pathologic findings. AJNR Am J Neuroradiol 2014;35:1634-41.
5. El Mekabaty A, Pardo CA, Gailloud P. The yield of initial conventional MRI in 115 cases of angiographically confirmed spinal vascular malformations. J Neurol 2017;264:733-39.
6. Oh JK, Lee DY, Kim TY, et al. Thoracolumbar extradural arachnoid cysts: a study of 14 consecutive cases. Acta Neurochir (Wien) 2012;154:341-8; discussion 48.
7. Papadopoulos A, Gouliamos A, Trakadas S, et al. MRI in the investigation of patients with myelopathy thought to be due to multiple sclerosis. Neuroradiology 1995;37:384-7.
8. Pinto WB, de Souza PV, de Albuquerque MV, Dutra LA, Pedroso JL, Barsottini OG. Clinical and epidemiological profiles of non-traumatic myelopathies. Arq Neuropsiquiatr 2016;74:161-5.
9. Watts J, Box GA, Galvin A, Van Tonder F, Trost N, Sutherland T. Magnetic resonance imaging of intramedullary spinal cord lesions: a pictorial review. J Med Imaging Radiat Oncol 2014;58:569-81.
10. Young WB. The clinical diagnosis of myelopathy. Semin Ultrasound CT MR 1994;15:250-4.
11. Takenaka S, Kaito T, Hosono N, et al. Neurological manifestations of thoracic myelopathy. Arch Orthop Trauma Surg 2014;134:903-12.
12. Mariano R, Flanagan EP, Weinshenker BG, Palace J. A practical approach to the diagnosis of spinal cord lesions. Pract Neurol 2018;18:187-200.
13. Karnaze MG, Gado MH, Sartor KJ, Hodges FJ, 3rd. Comparison of MR and CT myelography in imaging the cervical and thoracic spine. AJR Am J Roentgenol 1988;150:397-403.
14. Song KJ, Choi BW, Kim GH, Kim JR. Clinical usefulness of CT-myelogram comparing with the MRI in degenerative cervical spinal disorders: is CTM still useful for primary diagnostic tool? J Spinal Disord Tech 2009;22:353-7.
15. Grams AE, Gempt J, Forschler A. Comparison of spinal anatomy between 3-Tesla MRI and CT-myelography under healthy and pathological conditions. Surg Radiol Anat 2010;32:581-5.
16. Beckmann NM, West OC, Nunez D, Jr., et al. ACR Appropriateness Criteria® Suspected Spine Trauma. J Am Coll Radiol 2019;16:S264-S85.
17. Tan Z, Zhou Y, Li X, et al. Brain magnetic resonance imaging, cerebrospinal fluid, and autoantibody profile in 118 patients with neuropsychiatric lupus. Clin Rheumatol 2018;37:227-33.

18. Ahmadli U, Ulrich NH, Yuqiang Y, Nanz D, Sarnthein J, Kollias SS. Early detection of cervical spondylotic myelopathy using diffusion tensor imaging: Experiences in 1.5-tesla magnetic resonance imaging. Neuroradiol J 2015;28:508-14.
19. Ellingson BM, Salamon N, Hardy AJ, Holly LT. Prediction of Neurological Impairment in Cervical Spondylotic Myelopathy using a Combination of Diffusion MRI and Proton MR Spectroscopy. PLoS One 2015;10:e0139451.
20. Guan X, Fan G, Wu X, et al. Diffusion tensor imaging studies of cervical spondylotic myelopathy: a systemic review and meta-analysis. PLoS One 2015;10:e0117707.
21. Shen C, Xu H, Xu B, et al. Value of conventional MRI and diffusion tensor imaging parameters in predicting surgical outcome in patients with degenerative cervical myelopathy. J Back Musculoskelet Rehabil 2018;31:525-32.
22. American College of Radiology. American College of Radiology. ACR– ASNR– SPR Practice Parameter for the Performance of Myelography and Cisternography. Available at: https://www.acr.org/-/media/ACR/Files/PracticeParameters/myelog-cisternog.pdf. Accessed September 30, 2020.
23. Shah LM, Jennings JW, Kirsch CFE, et al. ACR Appropriateness Criteria® Management of Vertebral Compression Fractures. J Am Coll Radiol 2018;15:S347-S64.
24. Domenicucci M, Mancarella C, Santoro G, et al. Spinal epidural hematomas: personal experience and literature review of more than 1000 cases. J Neurosurg Spine 2017;27:198-208.
25. Jain NK, Dao K, Ortiz AO. Radiologic evaluation and management of postoperative spine paraspinal fluid collections. Neuroimaging Clin N Am 2014;24:375-89.
26. American College of Radiology. ACR Appropriateness Criteria®: Follow-up of Malignant or Aggressive Musculoskeletal Tumors. Available at: https://acsearch.acr.org/docs/69428/Narrative/. Accessed September 30, 2020.
27. Vargas MI, Gariani J, Sztajzel R, et al. Spinal cord ischemia: practical imaging tips, pearls, and pitfalls. AJNR Am J Neuroradiol 2015;36:825-30.
28. Zalewski NL, Rabinstein AA, Krecke KN, et al. Spinal cord infarction: Clinical and imaging insights from the periprocedural setting. J Neurol Sci 2018;388:162-67.
29. Donauer E, Aguilar Perez M, Jangid N, Tomandl B, Ganslandt O, Henkes H. Spontaneous Cervical Intramedullary and Subarachnoid Hemorrhage due to a Sulco-Commissural Artery Aneurysm. Clin Neuroradiol 2019;29:777-81.
30. Matsui T, Taniguchi T, Saitoh T, et al. Hematomyelia caused by ruptured intramedullary spinal artery aneurysm associated with extramedullary spinal arteriovenous fistula--case report. Neurol Med Chir (Tokyo) 2007;47:233-6.
31. AbdelRazek MA, Mowla A, Farooq S, Silvestri N, Sawyer R, Wolfe G. Fibrocartilaginous embolism: a comprehensive review of an under-studied cause of spinal cord infarction and proposed diagnostic criteria. J Spinal Cord Med 2016;39:146-54.
32. Tessitore E, Broc N, Mekideche A, et al. A modern multidisciplinary approach to patients suffering from cervical spondylotic myelopathy. J Neurosurg Sci 2019;63:19-29.
33. Abdulhadi MA, Perno JR, Melhem ER, Nucifora PG. Characteristics of spondylotic myelopathy on 3D driven-equilibrium fast spin echo and 2D fast spin echo magnetic resonance imaging: a retrospective cross-sectional study. PLoS One 2014;9:e100964.
34. Ellingson BM, Salamon N, Holly LT. Advances in MR imaging for cervical spondylotic myelopathy. Eur Spine J 2015;24 Suppl 2:197-208.
35. Kovalova I, Kerkovsky M, Kadanka Z, et al. Prevalence and Imaging Characteristics of Nonmyelopathic and Myelopathic Spondylotic Cervical Cord Compression. Spine (Phila Pa 1976) 2016;41:1908-16.
36. Puzzilli F, Mastronardi L, Ruggeri A, Lunardi P. Intramedullary increased MR signal intensity and its relation to clinical features in cervical myelopathy. J Neurosurg Sci 1999;43:135-9; discussion 39.
37. Avadhani A, Rajasekaran S, Shetty AP. Comparison of prognostic value of different MRI classifications of signal intensity change in cervical spondylotic myelopathy. Spine J 2010;10:475-85.
38. Nouri A, Martin AR, Kato S, Reihani-Kermani H, Riehm LE, Fehlings MG. The Relationship Between MRI Signal Intensity Changes, Clinical Presentation, and Surgical Outcome in Degenerative Cervical Myelopathy: Analysis of a Global Cohort. Spine (Phila Pa 1976) 2017;42:1851-58.
39. Salem HM, Salem KM, Burget F, Bommireddy R, Klezl Z. Cervical spondylotic myelopathy: the prediction of outcome following surgical intervention in 93 patients using T1- and T2-weighted MRI scans. Eur Spine J 2015;24:2930-5.

40. Seki S, Kawaguchi Y, Nakano M, et al. Clinical significance of high intramedullary signal on T2-weighted cervical flexion-extension magnetic resonance imaging in cervical myelopathy. J Orthop Sci 2015;20:973-7.
41. Uchida K, Nakajima H, Takeura N, et al. Prognostic value of changes in spinal cord signal intensity on magnetic resonance imaging in patients with cervical compressive myelopathy. Spine J 2014;14:1601-10.
42. Flanagan EP, Krecke KN, Marsh RW, Giannini C, Keegan BM, Weinshenker BG. Specific pattern of gadolinium enhancement in spondylotic myelopathy. Ann Neurol 2014;76:54-65.
43. Ozawa H, Sato T, Hyodo H, et al. Clinical significance of intramedullary Gd-DTPA enhancement in cervical myelopathy. Spinal Cord 2010;48:415-22.
44. Hayashi D, Roemer FW, Mian A, Gharaibeh M, Muller B, Guermazi A. Imaging features of postoperative complications after spinal surgery and instrumentation. AJR Am J Roentgenol 2012;199:W123-9.
45. Kister I, Johnson E, Raz E, Babb J, Loh J, Shepherd TM. Specific MRI findings help distinguish acute transverse myelitis of Neuromyelitis Optica from spinal cord infarction. Mult Scler Relat Disord 2016;9:62-7.
46. Nogueira RG, Ferreira R, Grant PE, et al. Restricted diffusion in spinal cord infarction demonstrated by magnetic resonance line scan diffusion imaging. Stroke 2012;43:532-5.
47. Zecca C, Cereda C, Wetzel S, et al. Diffusion-weighted imaging in acute demyelinating myelopathy. Neuroradiology 2012;54:573-8.
48. Vuong SM, Jeong WJ, Morales H, Abruzzo TA. Vascular Diseases of the Spinal Cord: Infarction, Hemorrhage, and Venous Congestive Myelopathy. Semin Ultrasound CT MR 2016;37:466-81.
49. Gass A, Back T, Behrens S, Maras A. MRI of spinal cord infarction. Neurology 2000;54:2195.
50. Thurnher MM, Bammer R. Diffusion-weighted MR imaging (DWI) in spinal cord ischemia. Neuroradiology 2006;48:795-801.
51. Andre JB, Bammer R. Advanced diffusion-weighted magnetic resonance imaging techniques of the human spinal cord. Top Magn Reson Imaging 2010;21:367-78.
52. Chee CG, Park KS, Lee JW, et al. MRI Features of Aquaporin-4 Antibody-Positive Longitudinally Extensive Transverse Myelitis: Insights into the Diagnosis of Neuromyelitis Optica Spectrum Disorders. AJNR Am J Neuroradiol 2018;39:782-87.
53. Costallat BL, Ferreira DM, Costallat LT, Appenzeller S. Myelopathy in systemic lupus erythematosus: clinical, laboratory, radiological and progression findings in a cohort of 1,193 patients. Rev Bras Reumatol Engl Ed 2016;56:240-51.
54. Durel CA, Marignier R, Maucort-Boulch D, et al. Clinical features and prognostic factors of spinal cord sarcoidosis: a multicenter observational study of 20 BIOPSY-PROVEN patients. J Neurol 2016;263:981-90.
55. Flanagan EP, Kaufmann TJ, Krecke KN, et al. Discriminating long myelitis of neuromyelitis optica from sarcoidosis. Ann Neurol 2016;79:437-47.
56. Isoda H, Ramsey RG. MR imaging of acute transverse myelitis (myelopathy). Radiat Med 1998;16:179-86.
57. Mok CC, Lau CS, Chan EY, Wong RW. Acute transverse myelopathy in systemic lupus erythematosus: clinical presentation, treatment, and outcome. J Rheumatol 1998;25:467-73.
58. Quintanilla-Gonzalez L, Atisha-Fregoso Y, Llorente L, Fragoso-Loyo H. Myelitis in systemic lupus erythematosus: clinical characteristics and effect in accrual damage. A single-center experience. Lupus 2017;26:248-54.
59. Uygunoglu U, Zeydan B, Ozguler Y, et al. Myelopathy in Behcet's disease: The Bagel Sign. Ann Neurol 2017;82:288-98.
60. Wendebourg MJ, Nagy S, Derfuss T, Parmar K, Schlaeger R. Magnetic resonance imaging in immune-mediated myelopathies. J Neurol 2020;267:1233-44.
61. Dobson R, Giovannoni G. Multiple sclerosis - a review. Eur J Neurol 2019;26:27-40.
62. Kearney H, Miller DH, Ciccarelli O. Spinal cord MRI in multiple sclerosis--diagnostic, prognostic and clinical value. Nat Rev Neurol 2015;11:327-38.
63. Filippi M, Rocca MA, Ciccarelli O, et al. MRI criteria for the diagnosis of multiple sclerosis: MAGNIMS consensus guidelines. Lancet Neurol 2016;15:292-303.
64. Jurynczyk M, Craner M, Palace J. Overlapping CNS inflammatory diseases: differentiating features of NMO and MS. J Neurol Neurosurg Psychiatry 2015;86:20-5.

65. Kim HJ, Paul F, Lana-Peixoto MA, et al. MRI characteristics of neuromyelitis optica spectrum disorder: an international update. Neurology 2015;84:1165-73.
66. Matthews L, Marasco R, Jenkinson M, et al. Distinction of seropositive NMO spectrum disorder and MS brain lesion distribution. Neurology 2013;80:1330-7.
67. Hemond CC, Bakshi R. Magnetic Resonance Imaging in Multiple Sclerosis. Cold Spring Harb Perspect Med 2018;8.
68. Thompson AJ, Banwell BL, Barkhof F, et al. Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria. Lancet Neurol 2018;17:162-73.
69. Arevalo O, Riascos R, Rabiei P, Kamali A, Nelson F. Standardizing Magnetic Resonance Imaging Protocols, Requisitions, and Reports in Multiple Sclerosis: An Update for Radiologist Based on 2017 Magnetic Resonance Imaging in Multiple Sclerosis and 2018 Consortium of Multiple Sclerosis Centers Consensus Guidelines. J Comput Assist Tomogr 2019;43:1-12.
70. Traboulsee A, Simon JH, Stone L, et al. Revised Recommendations of the Consortium of MS Centers Task Force for a Standardized MRI Protocol and Clinical Guidelines for the Diagnosis and Follow-Up of Multiple Sclerosis. AJNR Am J Neuroradiol 2016;37:394-401.
71. Pierce JL, Donahue JH, Nacey NC, et al. Spinal Hematomas: What a Radiologist Needs to Know. Radiographics 2018;38:1516-35.
72. Lee SY, Hur JW, Ryu KS, Kim JS, Chung HJ, Song MS. The Clinical Usefulness of Preoperative Imaging Studies to Select Pathologic Level in Cervical Spondylotic Myelopathy: Comparative Analysis of Three-Position MRI and Post-Myelographic CT. Turk Neurosurg 2019;29:127-33.
73. Penning L, Wilmink JT, van Woerden HH, Knol E. CT myelographic findings in degenerative disorders of the cervical spine: clinical significance. AJR Am J Roentgenol 1986;146:793-801.
74. Kitya D, Punchak M, Bajunirwe F. Role of Conventional Myelography in Diagnosis and Treatment of Degenerative Spine Disease in Low-Income Communities: Prospective Study. World Neurosurg 2017;104:161-66.
75. An HS, Seldomridge JA. Spinal infections: diagnostic tests and imaging studies. Clin Orthop Relat Res 2006;444:27-33.
76. Grelat M, Madkouri R, Tremlet J, Thouant P, Beaurain J, Mourier KL. Aim and indications of spinal angiography for spine and spinal cord surgery: Based on a retrospective series of 70 cases. Neurochirurgie 2016;62:38-45.
77. Hardacker JW, Shuford RF, Capicotto PN, Pryor PW. Radiographic standing cervical segmental alignment in adult volunteers without neck symptoms. Spine (Phila Pa 1976) 1997;22:1472-80; discussion 80.
78. Boruah DK, Prakash A, Gogoi BB, Yadav RR, Dhingani DD, Sarma B. The Importance of Flexion MRI in Hirayama Disease with Special Reference to Laminodural Space Measurements. AJNR Am J Neuroradiol 2018;39:974-80.
79. Reardon MA, Raghavan P, Carpenter-Bailey K, et al. Dorsal thoracic arachnoid web and the "scalpel sign": a distinct clinical-radiologic entity. AJNR Am J Neuroradiol 2013;34:1104-10.
80. Schultz R, Jr., Steven A, Wessell A, et al. Differentiation of idiopathic spinal cord herniation from dorsal arachnoid webs on MRI and CT myelography. J Neurosurg Spine 2017;26:754-59.
81. Chen H, Pan J, Nisar M, et al. The value of preoperative magnetic resonance imaging in predicting postoperative recovery in patients with cervical spondylosis myelopathy: a meta-analysis. Clinics (Sao Paulo) 2016;71:179-84.
82. Gbadamosi H, Mensah YB, Asiamah S. MRI features in the non-traumatic spinal cord injury patients presenting at the Korle Bu Teaching Hospital, Accra. Ghana Med J 2018;52:127-32.
83. Keddie S, Adams A, Kelso ARC, et al. No laughing matter: subacute degeneration of the spinal cord due to nitrous oxide inhalation. J Neurol 2018;265:1089-95.
84. Xiao CP, Ren CP, Cheng JL, et al. Conventional MRI for diagnosis of subacute combined degeneration (SCD) of the spinal cord due to vitamin B-12 deficiency. Asia Pac J Clin Nutr 2016;25:34-8.
85. Khan M, Ambady P, Kimbrough D, 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.
86. Dalmau J, Graus F. Antibody-Mediated Encephalitis. N Engl J Med 2018;378:840-51.
87. Flanagan EP, Keegan BM. Paraneoplastic myelopathy. Neurol Clin 2013;31:307-18.
88. Krings T, Geibprasert S. Spinal dural arteriovenous fistulas. AJNR Am J Neuroradiol 2009;30:639-48.

89. Kralik SF, Murph D, Mehta P, O'Neill DP. Diagnosis of spinal dural arteriovenous fistula using 3D T2-weighted imaging. Neuroradiology 2017;59:997-1002.
90. Suh DC, Song Y, Park D, et al. New grading system for the clinical evaluation of patients with spinal vascular lesions. Neuroradiology 2018;60:1035-41.
91. Zhou G, Li MH, Lu C, et al. Dynamic contrast-enhanced magnetic resonance angiography for the localization of spinal dural arteriovenous fistulas at 3T. J Neuroradiol 2017;44:17-23.
92. Abul-Kasim K, Thurnher MM, McKeever P, Sundgren PC. Intradural spinal tumors: current classification and MRI features. Neuroradiology 2008;50:301-14.
93. Arima H, Hasegawa T, Togawa D, et al. Feasibility of a novel diagnostic chart of intramedullary spinal cord tumors in magnetic resonance imaging. Spinal Cord 2014;52:769-73.
94. Kalayci M, Cagavi F, Gul S, Yenidunya S, Acikgoz B. Intramedullary spinal cord metastases: diagnosis and treatment - an illustrated review. Acta Neurochir (Wien) 2004;146:1347-54; discussion 54.
95. Payer S, Mende KC, Westphal M, Eicker SO. Intramedullary spinal cord metastases: an increasingly common diagnosis. Neurosurg Focus 2015;39:E15.
96. Samartzis D, Gillis CC, Shih P, O'Toole JE, Fessler RG. Intramedullary Spinal Cord Tumors: Part I-Epidemiology, Pathophysiology, and Diagnosis. Global Spine J 2015;5:425-35.
97. Graber JJ, Nolan CP. Myelopathies in patients with cancer. Arch Neurol 2010;67:298-304.
98. Fujiwara Y, Manabe H, Harada T, Izumi B, Adachi N. Extraordinary positional cervical spinal cord compression in extension position as a rare cause of postoperative progressive myelopathy after cervical posterior laminoplasty detected using the extension/flexion positional CT myelography: one case after laminectomy following failure of a single-door laminoplasty/one case after double-door laminoplasty without interlaminar spacers. Eur Spine J 2017;26:170-77.
99. Kiyosue H, Matsumaru Y, Niimi Y, et al. Angiographic and Clinical Characteristics of Thoracolumbar Spinal Epidural and Dural Arteriovenous Fistulas. Stroke 2017;48:3215-22.
100. Yamaguchi S, Takemoto K, Takeda M, et al. The Position and Role of Four-Dimensional Computed Tomography Angiography in the Diagnosis and Treatment of Spinal Arteriovenous Fistulas. World Neurosurg 2017;103:611-19.
101. Sakai Y, Matsuyama Y, Imagama S, Ito Z, Wakao N, Ishiguro N. Clinical utility of multidetector row computed tomography for diagnosing spinal dural arteriovenous fistulas undiagnosed by magnetic resonance imaging. Geriatr Gerontol Int 2010;10:255-63.
102. Yamaguchi S, Nagayama T, Eguchi K, Takeda M, Arita K, Kurisu K. Accuracy and pitfalls of multidetector-row computed tomography in detecting spinal dural arteriovenous fistulas. J Neurosurg Spine 2010;12:243-8.
103. American College of Radiology. ACR Appropriateness Criteria® Radiation Dose Assessment Introduction. Available at: https://www.acr.org/-/media/ACR/Files/Appropriateness-Criteria/RadiationDoseAssessmentIntro.pdf. Accessed September 30, 2020.