Imaging Approach to the Cord T2 Hyperintensity (Myelopathy)

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KEYWORDS

• Myelopathy • Acute transverse myelitis • MS • ADEM • Vascular malformation • MRI

KEY POINTS

- In clinically suspected case of myelopathy, MR imaging without and with gadolinium remains the modality of choice.
- The first and best imaging approach in the evaluation of myelopathy is to identify whether the cause of myelopathy is compressive or noncompressive.
- The commonest imaging finding in myelopathy is either focal or diffuse cord hyperintensity on the T2-weighted MR imaging images.
- Detailed clinical history, acuity of symptoms (acute vs insidious onset), distribution of the signal abnormalities, including length of cord involvement, specific tract involvement, and the region of the spinal cord that is affected, are very useful in making the diagnosis.

INTRODUCTION

Myelopathy is defined as a neurologic deficit related to a spinal cord pathologic abnormality. The commonest imaging finding in myelopathy is either focal or diffuse cord hyperintensity on the T2-weighted magnetic resonance (MR) images (T2 WI). Intramedullary T2 hyperintensity can pose a serious dilemma in spinal imaging because it has a myriad of causes, which can be difficult to differentiate. The clinical symptoms of myelopathy can be insidious in onset and vague in presentation. In addition, the numerous patterns of tract decussation can lead to confusing physical examination symptoms and difficulty in pinpointing a level of pathologic abnormality. The radiologic appearance of myelopathy is no less confusing, as many pathologies present with T2 hyperintensity. A systematic approach that incorporates history, physical examination, and radiologic findings is imperative to narrow the differential diagnosis. Contrast-enhanced MR imaging remains the primary investigation in the evaluation of a myelopathic patient. The first and best imaging approach is to identify whether the cause of myelopathy is compressive or noncompressive, because evaluation of compressive causes is often straightforward with a specific

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cause identified on MR. In contrast, negotiating the exact cause of noncompressive myelopathy may be very challenging. Detailed clinical history, acuity of symptoms (acute vs insidious onset), distribution of the signal abnormalities, including length of cord involvement, specific tract involvement, and the region of the spinal cord that is affected, are very useful in making the diagnosis.

Common causes of the myelopathy can be classified into compressive, infection, inflammatory, vascular, metabolic/toxic, and hereditary (**Table 1**).

COMPRESSIVE

Trauma

Trauma is one of the most common causes of compressive myelopathy, which may be secondary to osseous spinal canal compromise or epidural hematoma. This effect may be compounded in the setting of degenerative changes, especially in the cervical spine. Cord compression following trauma may be due to the presence of associated spinal fractures, subluxation, ligamentous injury, prevertebral swelling, and epidural hematoma. Following trauma, any neurologic symptom related to the cord is an indication for an MR imaging, especially when computed tomography (CT) is negative.[1] One of the common clinical presentations is that of the central cord syndrome, which consists of motor and sensory impairment. Central cord syndrome is most commonly seen in the cervical spine and, therefore, the symptoms are most pronounced in the upper extremities.[2]

In the setting of trauma, CT remains the primary modality because of its speed and sensitivity in the diagnosis of bony injury. Fracture fragment, subluxation, and extradural or subdural hematoma may cause severe canal compromise and cord compression (**Fig. 1**). MR imaging is more sensitive in identifying soft tissue, ligamentous, and cord injury. MR imaging may show mild to diffuse cord swelling, focal or diffuse edema, and in some cases, intra- or extramedullary hemorrhage. Cord edema is seen as focal or diffuse hyperintensity on T2 WI. Hemorrhagic contusion, defned as a discrete focus of hemorrhage within the spinal cord, is seen as foci of low signal intensities within cord edema, on T2, and on gradient echo sequence (GRE) images (**Fig. 2**). In the setting of trauma, the extent of cord edema and the presence of hemorrhagic contusion should be evaluated because edema spanning over 2 levels of vertebral segments is associated with a greater severity of neurologic deficits and cord edema alone has a more favorable prognosis than cord hemorrhage.[3]

It is not uncommon to see trauma to the cord following spine surgery or instrumentation; this may be a direct injury to the cord with the surgical instruments or due to vascular compromise during surgical manipulation. Clinical presentation is fairly acute with sensory or motor deficit immediately following the procedure. On imaging, most of these lesions are focal and at the site of surgery (**Fig. 3**).

Spondylosis

Degenerative changes may cause myelopathy in the absence of trauma, secondary to direct cord compression or vascular compromise (mainly venous). Both disc herniation and osteophyte formation may cause compressive myelopathy and the problem may be compounded by superimposed spinal canal stenosis. In fact, cervical spondylotic myelopathy is the most common cause of myelopathy in patients older than 55 years[4] with C5-C6 followed by C6-C7 being the most common levels. There is no uniform prognosis or anticipated progression of disease with some patients presenting with progressive symptoms, whereas others have long periods of relatively little symptoms.[5]

Imaging, especially MR imaging, plays a very important role in the diagnosis of myelopathy due to spondylosis. The role of plain radiographs is debatable; however, in clinical practice radiographs remain the first investigation performed. Spinal canal narrowing can be assessed on lateral plain radiographs of the cervical spine by measuring the distance from the most posterior aspect of one vertebral body to the closest spinolaminar line. Measurements less than 13 mm are associated with an increased risk for myelopathy[6] and absolute/severe stenosis is defined as measurement less than 10 mm. Cross-sectional imaging CT and MR imaging are complementary to each other in diagnosis of compressive myelopathy. Bony changes such as early osteophytes and un-covertebral hypertrophy are better seen on CT, whereas MR imaging, due to its superior ability to image soft tissue, provides additional information regarding cord, disc ligaments, and cysts. GRE on MR imaging is very sensitive in distinguishing osteophytes from disc herniation because osteophytes show low signal intensity relative to higher signal intensity disc material. Canal measurements and cord signal intensity are best assessed on T2 WI images. Cord changes on MR imaging will depend on the duration of the compression.[6] In acute to subacute stages where the changes are predominately due to cord edema, T2 WI will show bright signal intensity focal at either the level of disc herniation or the long segment depending on the extent of stenosis