A 22 years old male patient presented with quadriplegia with a high cervical sensory level of acute onset. The neurological disability occurred 10 days following an attack of flu.

Figure 1. A case of transverse myelitis. A, precontrast MRI T1 image and B,C,D postcontrast MRI T1 images, showing mild dilatation of C2,C3,C4,C5 cervical spinal segments with central intramedullary, multisegmental hypointensity and with peripheral contrast enhancement.
Figure 2. A case of transverse myelitis. MRI T2 images showing multisegmental (about five spinal segments from C2-C6) intramedullary hyperintensity with mild cord dilatation.

Figure 3. A case of transverse myelitis. Cross-sectional MRI T2 images showing mild dilatation of the spinal cord with central hyperintensities occupying more than 2/3 of the cross-sectional area of the spinal cord with absence of the central dot sign.
Figure 4. MRI T1 precontrast (A,B,C,D) and postcontrast (E,F,G) and MRI T2 image (H) showing a case of acute idiopathic transverse myelitis, notice cord swelling in the cervico dorsal region with patchy irregular and peripheral contrast enhancement. Also notice the central T2 hyperintensity. Peripheral contrast enhancement is outside and peripheral to the central T2 hyperintensity.

Figure 5. MRI T2 showing a case of acute idiopathic transverse myelitis. Notice cord swelling and the multisegmental, central increased cord signal intensity at the cervicodorsal region

Table 1. The MRI picture characteristic of idiopathic transverse myelitis

1. A centrally located multisegmental (3 to 8 spinal segments) MRI T2 hyperintensity that occupies more than two thirds of the cross-sectional area of the cord is characteristic of transverse myelitis. The MRI T2 hyperintensity commonly shows a slow regression with clinical improvement. The central spinal cord MRI T2 hyperintensity represents evenly distributed central cord edema. MRI T1 Hypointensity might be present in the same spinal segments that show T2 hyperintensity although to a lesser extent. The MRI T2 hyperintensity is central, bilateral, more or less symmetrical and multisegmental.

2. MRI T2 central isointensity, or dot (within and in the core of the MRI T2 hyperintensity) might be present and is believed to represent central gray matter squeezed by the uniform, evenly distributed edematous changes of the cord. (central dot sign). It might not be of any clinical significance.

3. Contrast enhancement is commonly focal or peripheral and maximal at or near the segmental MRI T2 hyperintensity. In idiopathic transverse myelitis enhancement is peripheral to the centrally located area of high T2 signal intensity rather than in the very same area. The prevalence of cord enhancement is significantly higher in patients with cord expansion.
4. Spinal cord expansion might or might not be present and when present is usually multisegmental and better appreciated on the sagittal MRI T1 images. Spinal cord expansion tapers smoothly to the normal cord, and is of lesser extent than the high T2 signal abnormality.

5. Multiple sclerosis plaques (and subsequent T2 hyperintensity) are located peripherally, are less than 2 vertebral segments in length, and occupies less than half the cross-sectional area of the cord. In contrast to transverse myelitis, enhancement in MS occurs in the same location of high-signal-intensity lesions seen on T2-weighted images.

Table 2. Differences between idiopathic transverse myelitis and spinal multiple sclerosis

<table>
<thead>
<tr>
<th>Disease entity</th>
<th>T2 hyperintensity</th>
<th>Number of segments involved</th>
<th>Contrast element</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic transverse myelitis</td>
<td>Central, multisegmental</td>
<td>4-8</td>
<td>In transverse myelitis enhancement is peripheral to the centrally located area of high T2 signal intensity rather than in the very same area.</td>
<td>Nonspecific necrosis that affects gray and white matter indiscriminately and destroys axons and cell bodies as well as myelin.</td>
</tr>
<tr>
<td>Spinal multiple sclerosis</td>
<td>Peripheral</td>
<td>1-2</td>
<td>In contrast to transverse myelitis, enhancement in MS occurs in the same location of high-signal-intensity lesions seen on T2-weighted images.</td>
<td>White matter demyelination only.</td>
</tr>
</tbody>
</table>

**DISCUSSION:**

The first cases of acute transverse myelitis (ATM) were described in 1882 by Bastian [1]. In 1922 and 1923, 200 cases of so-called “post-vaccination encephalomyelitis” were reported in Holland and England. It was in 1948 that the term ATM was used in reporting a case of severe myelopathy after pneumonia [2].

Transverse myelitis is a clinical syndrome characterized by bilateral motor, sensory, and autonomic disturbances [3]. About 50% of patients have paraparesis; 80% to 94% have numbness, paresthesias, and band-like dysesthesias; and all have bladder dysfunction [3]. The histopathologic features of TM include perivascular monocytic and lymphocytic infiltration, demyelination, and axonal injury [4]. TM may exist as part of a multifocal CNS disease; as a multisystemic disease; or as an isolated, idiopathic entity. The immunopathogenesis of disease-associated TM is varied and includes vasculitis neurosarcoidosis, MS, and lupus. Several reports of TM after vaccination have been published [5,6]. Recently, the term “parainfectious TM” has been introduced for TM cases with antecedent respiratory, gastrointestinal, or systemic illness [4]. A variety of immune stimuli (eg, molecular mimicry, superantigen-mediated immune activation) may trigger the immune system to injure the nervous system [4]. In a retrospective study of 288 patients who had TM, 45 (15.6%) met the criteria for idiopathic TM [7]. According to the published series, approximately one third of patients recover with little or no sequelae, one third are left with a moderate degree of permanent disability, and one third develop severe disability [8]. In 2002, the Transverse Myelitis Consortium Working Group proposed criteria for idiopathic ATM, with incorporation of CSF testing and MR imaging findings [8].

The criteria include (1) bilateral sensory, motor, or autonomic spinal cord dysfunction; (2) defined sensory level and bilateral signs and symptoms; (3) proof of inflammation within the spinal cord by MR imaging or CSF examination; (4) symptoms from onset to reach maximal deficit between few hours and 21 days; and (5) exclusion of extra-axial
compressive etiology [8]. The thoracic spine is most commonly involved, and middle-aged adults are usually affected. MR imaging findings include focal, centrally located increased signal on T2-weighted MR images, usually occupying more than two thirds of the cross-sectional area of the cord (Fig. 6) [9]. This was observed in 88% of patients in a series of 17 patients who had idiopathic TM [10]. Usually, the signal abnormality extends more than three to four vertebral segments in length. Cord expansion may or may not be present; it was found in 47% in published series [9]. Enhancement is usually absent; when enhancement was present, two patterns have been described: moderate patchy enhancement or diffuse abnormal enhancement (Fig. 7, Fig. 8) [7,10,11,12]. Enhancement was found in only 38% of cases of idiopathic TM in one series and in 47% and 53% in the two other series [7,9]. About 40% of TM cases display a normal MR imaging study [13]. MS is the most important differential diagnosis of TM. Signal abnormality located peripherally in the spinal cord that is less than two vertebral segments in length and occupying less than half the cross-sectional area of the cord favors a diagnosis of MS rather than TM [9].

Figure 6. ATM. (A) Sagittal T2-weighted MR image showing high-signal-intensity abnormality in the spinal cord lesion extending over several segments of the upper thoracic spine. (B) A focal, centrally located increased signal occupying more than two thirds of the cross-sectional area of the cord is demonstrated on the axial T2-weighted MR image. (C) On a sagittal, diffusion-weighted MR image performed using navigated interleaved multishot echo planar imaging (5-mm slice thickness, $b_{\text{max}} = 700$ s/mm²), high signal indicates increased diffusion in the area of increased signal on T2-WI. (D) High signal was observed on the apparent diffusion coefficient map, suggesting a T2 shine-through effect rather than restricted diffusion in spinal cord areas affected by myelitis.
There is growing evidence that the length of the lesion is likely important from a pathogenic and a prognostic standpoint. Patients who have acute partial transverse myelitis have signal abnormalities extending less than two segments on MR imaging, and patients who have complete longitudinally extensive transverse myelitis have abnormalities that extend to multiple segments (see Fig. 8). Patients in the first group are at higher risk for developing MS compared with those in the second group, where the risk is low [14].

DTI was recently used to characterize inflammatory processes of the spinal cord [15]. In cases of inflammatory myelitis, decreased FA values have been found in the region of a T2-weighted lesion and increased FA values in the lesion's boundaries. This pattern is different from that seen in invasive tumors, in which FA is low in peripheral regions of edema.
Novel biomarkers, such as cytokine interleukin-6 and collapsin response-mediator protein–5 are potentially useful prognostic indicators and markers of disease severity. The “idiopathic” form of ATM is rarely seen [16].

SUMMARY

Over the past decade, researchers and clinicians have gained new insights into the core of demyelinating diseases of the spinal cord, and much progress has been made in the management of these diseases. Although we are starting to uncover some of the structural and physiologic substrates of demyelination of the CNS, we are far from understanding what causes many of these demyelinating disorders and how to prevent their progression. With further development of new techniques, such as DTI and more potent MR units, spinal cord diseases may be distinguished from each other, and effective therapeutic strategies may be initiated before any cord damage occurs (Fig. 9).

In particular MRI is very helpful in differentiation between Spinal multiple sclerosis and transverse myelitis In the series reported by Choi et al, [18] the centrally located MRI T2 high signal intensity occupied more than two thirds of the cross-sectional area of the cord in transverse myelitis. In multiple sclerosis, plaques are usually located peripherally and occupy less than half the cross-sectional area of the cord. The central isointensity, or dot (commonly seen in transverse myelitis), represents central gray matter squeezed by the uniform, evenly distributed oedematous changes of the cord. Choi and colleagues [18] have demonstrated the role of contrast media in differentiating transverse myelitis from multiple sclerosis. In transverse myelitis, enhancement is in the periphery of a centrally located area of high T2 weighted images. In multiple sclerosis, the lesions show enhancement in the central zone of peripherally located high signal intensity on T2 weighted images. [14]

In conclusion, certain MRI characteristics help in differentiating acute transverse myelitis from spinal form of multiple sclerosis. These include: 1) centrally located high intensity signal extending over 3 to 4 segments and occupying more than two thirds of the cord cross-sectional area and 2) peripheral contrast enhancement of high intensity signal.
Figure 15. Differential diagnoses of intramedullary lesions based on their location at the cross-sectional area of the cord. (A) MS: Dorsally located wedge-shaped lesion involving less than two thirds of the cross-sectional area of the spinal cord seen on axial T2-Wi MR image. (B) Poliomyelitis: Bilateral enhancing anterior nerve roots demonstrated on postcontrast T1-Wi MR image. (C) Vacuolar myelopathy: Bilateral, symmetrical, high-signal-intensity abnormality located dorsally in the spinal cord in an HIV-positive patient. DD: Subacute combined degeneration. (D) ATM: On axial T2-Wi, a high-signal-intensity lesion involving more than two thirds of cross-sectional area of the spinal cord is observed. (E) Herpes-simplex-virus myelitis: Postcontrast T1-Wi axial MR image showing nodular enhancing lesion located in the lateral part of the cervical spinal cord. DD: active MS plaque. (F) Spinal cord infarction: Swelling of the anterior parts of the spinal cord is shown on axial T2-Wi MR images, indicating vulnerability of the anterior portions of the spinal cord to ischemia.

- **Addendum**
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REFERENCES

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