Spinal cord infection: myelitis and abscess formation.

K J Murphy, J A Brunberg, D J Quint and P H Kazanjian

AJNR Am J Neuroradiol 1998, 19 (2) 341-348
http://www.ajnr.org/content/19/2/341

This information is current as of July 22, 2023.
Spinal Cord Infection: Myelitis and Abscess Formation

Kieran J. Murphy, James A. Brunberg, Douglas J. Quint, and Powel H. Kazanjian

PURPOSE: Our purpose was to describe the MR findings and evolution of spinal cord abscess and to define those MR features that allow differentiation of cord infection from other intramedullary abnormalities.

METHODS: We retrospectively reviewed the MR studies of all patients in whom intramedullary spinal cord abscess was proved either by blood or cerebrospinal fluid culture or by serologic examination at our institution between January 1988 and January 1996. The study group included four adults and two children, 7 to 74 years old (mean age, 38 years).

RESULTS: Initial MR studies showed intramedullary high signal on T2-weighted sequences with poorly defined marginal enhancement on T1-weighted images. On follow-up contrast-enhanced T1-weighted studies, the lesions had well-defined enhancing margins with central low signal intensity. After the initiation of therapy, T2 signal abnormalities decreased markedly and contrast-enhanced studies showed ring enhancement. These T1 findings resolved with treatment over serial studies in four patients. The organisms identified were Streptococcus milleria, S pyogenes, atypical mycobacteria, Mycobacterium tuberculosis, and Schistosoma mansoni (both children).

CONCLUSION: A characteristic sequence of imaging findings aids in the differentiation of cord infection from other intramedullary lesions.

Intramedullary cord abscesses are uncommon; approximately 70 case have been reported in the literature, with most from the preantibiotic era (1–8). A case was reported in 1830 (2). Courville (3) found only one intramedullary abscess in 40 000 postmortem examinations. Forty percent of abscesses occur in the first two decades of life, with 27% occurring before the age of 10 years (4). Patients with symptoms of less than 4 days’ duration have a 90% mortality (5, 6), whereas patients whose symptoms last longer than 7 days have a 67% mortality rate (7). The purpose of this article is to describe the magnetic resonance (MR) imaging findings of spinal cord abscess in six patients, including the evolution of changes seen on follow-up imaging studies in five of them. We define those MR features that allow differentiation of cord infection from other intramedullary abnormalities.

Methods

We identified six patients (five male, one female) with proved intramedullary spinal cord abscess in whom MR studies were obtained at our institution from January 1988 to January 1996. Four patients were adults and two were children (age range, 7 to 74 years; mean age, 38 years). A total of 15 initial and follow-up studies were reviewed. All patients underwent an MR study consisting of a sagittal T1-weighted localizer image, sagittal proton density–weighted studies, T2-weighted fast spin-echo studies, and sagittal and axial T1-weighted spin-echo studies before and after administration of gadopentetate dimeglumine at a dose of 0.1 mmol/kg. Five patients had pretreatment studies. One patient had three follow-up MR studies, three patients had two follow-up studies, two patients had one follow-up study, and one patient had no follow-up studies. The time between the initial study and the first follow-up study ranged from 1 week to 1 month. Five patients had repeated imaging with clinical follow-up for 7 months, 9 months, 3 months, 2 months, and 5 months, respectively. One patient had no repeat imaging after the initial diagnostic studies but had clinical follow-up for 3 months. Clinical follow-up was by discussion with the referring physician. The location, the pattern of enhancement on sagittal and axial T1-weighted sequences, and the pattern of altered signal intensity on T2-weighted images were evaluated. Two patients also had serial brain imaging. All MR examinations were performed at 1.5 T.

No patient had a history of spinal trauma, spinal surgery, malignancy, or adjacent vertebral or epidural disease. One patient was immunocompromised as a result of steroid treat-
Case 1: 28-year-old woman with 10-year history of systemic lupus erythematosus and 10 days of fever, back pain, bilateral lower extremity paresthesia, and weakness. She had been on varying doses of prednisone for the preceding 9 months and was on prednisone (30 mg/day) at the time of presentation. M tuberculosis was isolated from cultures of lung tissue and from CSF. Residual gait disturbance was noted at 7-month clinical follow-up.

A–E, MR imaging at the time of presentation. T1-weighted sagittal and axial (T-11 level) images obtained before (A and C) and after (B and D) contrast administration show widened anteroposterior and transverse dimensions of the spinal cord with solid intramedullary contrast enhancement (arrow). T2-weighted image (E) shows central edema extending from the conus to the midthoracic levels (arrowheads).

F–H, Persistent cord expansion (arrows) is present 5 weeks after initiation of antituberculous treatment on an unenhanced T1-weighted image (F) with an area of enhancement (G) and increasing cord edema (H) on T2-weighted studies.

I–K, Seven months later, the cord is normal in anteroposterior dimension on unenhanced T1-weighted image (I), with a smaller persisting region of contrast enhancement (arrow, J). A T2-weighted study (K) shows only diffuse, mildly increased central signal intensity.
ment of systemic lupus erythematosus. None was human immunodeficiency virus (HIV) positive.

Tissue and microbiological (serology and polymerase chain reaction) confirmation of the specific infectious agent was available in four cases (mycobacteria in two cases, Schistosoma organisms in one case, and streptococcus in one case). Two patients, one infected by atypical Mycobacterium fortuitum and the other by S mansoni, underwent laminectomy and decompression with subtotal resection of the conus. One patient had a bronchoscopic biopsy confirming cerebrospinal fluid cultures of M tuberculosis. Another patient had a stereotactic brain biopsy confirming cerebrospinal fluid cultures of M tuberculosis. A third patient had a spinal biopsy confirming cerebrospinal fluid cultures of M tuberculosis. The fourth patient had a brain biopsy confirming cerebrospinal fluid cultures of M tuberculosis. All four patients had similar imaging changes in the cord. The initial imaging studies (see Table) showed areas of intramedullary high signal on proton density- and T2-weighted sequences (Fig 4C). The unenhanced T1-weighted sequences showed an increase in cord diameter (Fig 4B). After administration of contrast material, poorly defined marginal enhancement was visible on T1-weighted images (Fig 4E).

Results

The organisms responsible for these intramedullary infections were M tuberculosis (Fig 1), atypical M fortuitum (Fig 2), S pyogenes group A (Fig 3), S milleria (Fig 4), and, in two cases, S mansoni (Fig 5). All caused similar imaging changes in the cord. The initial imaging studies (see Table) showed areas of intramedullary high signal on proton density- and T2-weighted sequences (Fig 4C). The unenhanced T1-weighted sequences showed an increase in cord diameter (Fig 4B). After administration of contrast material, poorly defined marginal enhancement was visible on T1-weighted images (Fig 4E). This abnor-

Fig. 2. Case 2: M fortuitum intramedullary abscess in a 31-year-old man with a 10-day history of increasing lower back pain with pain and sensory changes in both lower extremities. He underwent laminectomy at T12-L1 at another institution. M fortuitum was cultured from the spinal cord tissue obtained during surgery. Decreased sensation in the left lower extremity was noted at the 9-month clinical follow-up.

A–E, T1-weighted sagittal and axial (T-12 level) images before (A and C) and after (B and D) contrast administration show widened anteroposterior and transverse dimensions of the distal cord and conus (long arrows, A) with intramedullary patchy and solid contrast enhancement (arrows, B and D) with central hypointensity (short arrow, A), which is most prominent posteriorly. T2-weighted image (E) shows edema extending from the conus to the T-12 level.

F–H, Four months later, sagittal T1-weighted images without (F) and with (G) contrast show a persisting cyst and punctate regions of enhancement (arrow, G) at the margins of the cyst. On the T2-weighted image (H) there is less edema and a decrease in the anteroposterior diameter of the terminal portion of the spinal cord.

A, Sagittal contrast-enhanced T1-weighted image shows widening of the anteroposterior diameter of the cord and low signal intensity in the conus. These is minimal enhancement (arrows) around the region of low signal intensity.

B, T2-weighted image shows high signal intensity in the central portion of the spinal cord (arrowheads).

C–F, One week later, T1-weighted sagittal (C) and axial (E) images at the T-12 level before (C and E) and after (D and F) contrast administration show widened anteroposterior and transverse dimensions of the spinal cord with ring enhancement extending to the surface of the spinal cord (arrows, D and F).

G, T2-weighted image shows intramedullary high signal at and above the conus (arrowheads).

H–I, Three months later, sagittal contrast-enhanced T1-weighted image shows subtle abnormal intramedullary enhancement (arrows, H) in the conus. The T2-weighted image shows resolution of edema with a central region of low signal intensity, possibly representing hemosiderin deposition (arrows, I).
mal enhancement was more localized than the diffuse and more extensive T2 high signal abnormality, which generally extended for several segments above and below the enhancing lesion (Fig 1G and H). After the initiation of therapy, the T2 signal abnormalities markedly decreased in size and in prominence (Fig 4C and F). Additionally, the regions where subtle, poorly defined marginal enhancement had been seen on pretreatment studies showed well-defined ring-enhancing margins with central low signal on post-contrast T1-weighted images, consistent with abscess formation (Fig 3A and D). These areas of enhancement resolved with treatment, as seen on serial imaging studies (Figs 1G and J, and 2B and G). An anatomic site of preferential involvement in the distal thoracic cord and conus was identified in all patients. Two patients also had multiple intracranial abscesses at the gray/white matter junction (Fig 4A). The brain lesions responded over the same time course as did the cord lesions. None of the patients had MR evidence of arachnoiditis. At clinical follow-up, one patient had normal findings, two had gait disturbance (one mild, one moderate), two had abnormal sensation in the lower extremities (one bilateral, one unilateral), and one had bowel, bladder, and gait disturbance.

**Discussion**

The symptoms of spinal cord are indistinguishable from those of epidural abscess. Most are of hematogenous origin, and the primary source is usually the respiratory tract (Figs 1–3). Intramedullary abscess may also complicate congenital dermal sinuses or bacterial endocarditis. Because the disease process is so rare, there is relatively little accumulated experience in the management of intramedullary abscess (4). Treatment consists of a several-month course of antibiotic administration for bacterial processes and at least a year of therapy for mycobacterial condi-
tions. Medical therapy alone may be attempted, but laminectomy and surgical debridement are indicated if neurologic symptoms progress during medical therapy. However, because the illness is uncommon, a consensus on the indications for neurosurgical intervention has not been established (4).

In an attempt to understand the pathogenesis and imaging alterations associated with spinal cord abscesses, it has been suggested that the development of this process may be associated with focal venous infarcts that are complicated by bacterial colonization (9, 10). Sequential imaging findings in our series of patients suggest that the development of abscess within the spinal cord may be similar to the pathologic evolution of abscess in the brain, the serial imaging characteristics and histology of which have been described in detail in animals and humans (11, 12). The serial spinal cord images of our patient group, as well as of a patient with intramedullary spinal cord infection who was excluded from our study because of disk and epidural space infection, reveal the same imaging progression that has been documented in the brain (11). T2-weighted MR images of brain tissue obtained during the phase of early cerebritis initially show high signal, with poorly defined enhancement on postcontrast T1-weighted images (Fig 4B and D). Approximatley 1 week after the initiation of treatment, the region of myelitis became less diffusely hyperintense on T2-weighted sequences, with more clearly defined marginal enhancement on postcontrast T1-weighted images (Fig 4F). The surrounding edema continues to be more extensive than the margins of enhancement. We term this phase the early stage of infectious myelitis (Fig 4B and D). Approximately 1 week after the initiation of treatment, the region of myelitis became less diffusely hyperintense on T2-weighted sequences, with more clearly defined marginal enhancement on postcontrast T1-weighted images (Fig 4F). The surrounding edema continues to be more extensive than the margins of enhancement. We term this phase the early stage of infectious myelitis (Fig 4B and D).

Fig 5. Case 5: *S. mansoni* intramedullary abscess in an 11-year-old boy who had recently immigrated to the United States from Yemen. He presented with bilateral lower extremity weakness progressing over 7 days to paraplegia. Diagnosis was based on schistosomiasis antibody from serum and CSF (result, 6.87 IU; normal, <1.0 IU). At 5-month clinical follow-up, the patient had minimal residual lower extremity weakness.

A–E, T1-weighted sagittal and axial (T-12 level) images before (A and D) and after (B and E) contrast administration show widened anteroposterior and transverse dimensions of the spinal cord with low central signal intensity (arrows, D) and with intramedullary contrast enhancement (arrows, B and E), most prominent anteriorly. The initial T2-weighted image shows abnormal high signal in the distal cord and conus (arrowheads, C).

F–G, Five months later there is no persisting abnormal enhancement after contrast administration (F), and signal intensity on T2-weighted image is normal (G).
 imaging described as the late stage of cerebritis by Enzmann et al (11). The earliest that well-defined enhancement was seen in our series was on a study 7 days after presentation (Fig 3D and F). This finding is thought to represent the beginning of intramedullary cord abscess formation. The central cavitary portions of the intraaxial necrotic areas are seen on T1-weighted noncontrast images as areas of low signal intensity and as hyperintense foci on T2-weighted sequences. While regions of hyperintense signal on T2-weighted sequences were seen to subside over several weeks, the foci on T1-weighted postcontrast images slowly diminished in size for several months (Fig 1J and K). In all patients, the initial T2-weighted sequences showed more extensive cord abnormality than did the postcontrast T1-weighted studies (Fig 1E and B).

Among the causes of spinal cord abscesses are important epidemiologic, social, and pathogenic differences that may relate to diagnosis, treatment, and outcome. Human schistosomiasis, for example, is not endemic in the United States. Approximately 200 million people are, however, infected with schistosome worms worldwide, and immigration and travel are increasingly bringing these patients to medical attention in this country (13). Tuberculosis is undergoing a national resurgence, particularly among HIV-infected persons but also in the immunocompetent population (15). Tuberculous meningitis is, in most cases, associated with tuberculous intracranial involvement of the meninges or brain parenchyma, or with tuberculous arachnoiditis of the spine (16). The imaging features may include irregular cord surface, clumped nerve roots, arachnoid cysts, and secondary syrinx formation within the cord

### Table: Six patients with intramedullary cord abscess

<table>
<thead>
<tr>
<th>Case</th>
<th>Organism</th>
<th>No. of Studies</th>
<th>Level of Abnormal T2 Signal</th>
<th>Expansion</th>
<th>Findings on Contrast-Enhanced MR Images</th>
<th>Findings at MR Follow-up</th>
<th>Clinical Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td><em>M tuberculosis</em></td>
<td>4</td>
<td>Distal cord, conus</td>
<td>Yes</td>
<td>Solid enhancement</td>
<td>At 5 wk, solid and ring enhanced</td>
<td>At 7 mo, gait disturbance</td>
</tr>
<tr>
<td>2</td>
<td><em>M fortuitum</em></td>
<td>2</td>
<td>Distal cord, conus</td>
<td>Yes</td>
<td>Solid enhancement, slight central hypointensity</td>
<td>At 4 mo, peripheral hypointensity</td>
<td>At 9 mo, decreased sensory disturbance</td>
</tr>
<tr>
<td>3</td>
<td>Group A <em>S pyogenes</em></td>
<td>3</td>
<td>Distal cord, conus</td>
<td>Yes</td>
<td>Peripheral, poorly defined enhancement</td>
<td>At 1 wk, ring-enhanced abscess</td>
<td>At 3 mo, resolution of symptoms</td>
</tr>
<tr>
<td>4</td>
<td>Group D <em>S milleria</em></td>
<td>3</td>
<td>T-1 to distal cord</td>
<td>No</td>
<td>Peripheral, poorly defined enhancement</td>
<td>At 10 d, ring-enhanced abscess</td>
<td>At 2 mo, lower limb sensory disturbance</td>
</tr>
<tr>
<td>5</td>
<td><em>S mansoni</em></td>
<td>2</td>
<td>Distal cord, conus</td>
<td>Yes</td>
<td>Solid with slight central hypointensity &quot;ricelike&quot; areas</td>
<td>At 5 mo, normal</td>
<td>At 5 mo, minimal residual gait disturbance</td>
</tr>
<tr>
<td>6</td>
<td><em>S mansoni</em></td>
<td>1</td>
<td>Distal cord, conus</td>
<td>Yes</td>
<td>Patchy central “ricelike” areas</td>
<td>...</td>
<td>At 3 mo, gait and bowel/bladder disturbance</td>
</tr>
</tbody>
</table>

Six patients with intramedullary cord abscess

- Solid enhancement
- Species not specified
- Solid enhancement, slight central hypointensity
- Peripheral, poorly defined enhancement
- Peripherally defined enhancement
- Peripheral, poorly defined enhancement
- Solid enhancement
- Peripheral, poorly defined enhancement
- Solid enhancement
- Solid enhancement
In the distal thoracic cord and conus medullaris was identified in all six patients studied. The diagnosis of septic myelitis needs to be considered in any patient with intramedullary high signal intensity and abnormal contrast enhancement on T2-weighted images.

References


Conclusion

Infection within the spinal cord initially appears as regional, increased signal intensity on T2-weighted images and as poorly defined enhancement on postcontrast T1-weighted images. In our series, ring enhancement was not seen during the myelitis phase at the time of clinical presentation; however, subsequent imaging, after at least 7 days, revealed ring enhancement and development of true intramedullary cord abscess. An anatomic site of preferential involvement in the case of meningial exudate (9). A similar potential mechanism did not appear to be operative in our patient population in that none of the six patients in our series had a history of preexisting cord ischemia or presenting symptoms suggestive of acute cord infarction, and all improved clinically after therapy. Additionally, there was no nerve root or meningeal enhancement to suggest meninges-related vascular inflammation as a cause of arterial or venous cord infarction. It is possible, however, that the cord expansion and edema seen in five of these six patients were associated with compromised cord perfusion and relative ischemia.

The differential diagnostic considerations of an intramedullary abscess includes cord ischemia, Guillain-Barré syndrome, acute disseminated myelitis, multiple sclerosis, hemangioblastoma, or other cavitating cord tumors. Serial imaging combined with microbiological and serologic evaluation of CSF, blood, and tissue can correctly identify protozoal or bacterial abscesses. We suggest that the radiologic management of this group of patients should focus on the clinically involved cord segment. Imaging of the entire spine or brain is not indicated unless the patient’s clinical status indicates its necessity.

Please see the Editorial on page 395 in this issue.