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MR Imaging of Intradural Inflammatory Diseases of the Spine

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Twenty-eight patients with intradural inflammatory disease of the spine were studied in order to characterize the MR imaging findings of infectious and inflammatory conditions. Patients were categorized according to the spinal compartment involved. Among the 12 patients in the intradural extramedullary group, unenhanced scans were either normal or nonspecific while contrast-enhanced scans were helpful in visualizing and localizing the lesion. Nevertheless, contrast-enhanced MR studies were unable to differentiate infection and inflammation from tumor in this compartment. Among 16 patients with intramedullary lesions, four had granulomatous disease and 12 had nongranulomatous disease. The granulomatous lesions resembled tumors and displayed MR characteristics of a focal lesion with large nodular enhancement. The patients with nongranulomatous intramedullary lesions exhibited two subsets of MR findings. In the first subset of nine patients, diffuse cord swelling and high signal were seen on long TR images, combined with either no enhancement or peripheral, diffuse, or speckled enhancement of the spinal cord on contrast-enhanced short TR images. In the second subset of three patients, minimal or no spinal cord swelling was displayed despite the visualization of high signal on long TR scans and nodular enhancement with contrast administration on short TR scans. Both subsets were sufficiently unique that nongranulomatous myelitis could usually be differentiated from spinal cord tumors.

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Intradural inflammatory diseases of the spine include meningitis and myelitis. There have been no previous reports of MR imaging in cases of spinal meningitis. Previous reports of MR imaging of myelitis have described numerous findings, including fusiform swelling of the spinal cord [1–7], high signal in the cord on T2-weighted images [1–3, 5, 6, 8, 9], high signal in the cord with no swelling or very slight cord swelling [8–10], or normal signal in the spinal cord on T2-weighted images despite spinal cord swelling [4]. Myelitis has been reported to enhance with contrast administration [1, 5, 7], but enhancement patterns have not been characterized. The purpose of this study is to see if MR imaging shows any abnormality in patients with intradural inflammatory disease and, if so, to then characterize these lesions.

Materials and Methods

The MR images of 28 patients with intradural inflammatory disease of the spine were reviewed. The patients were divided into two groups, those with intradural extramedullary lesions (12 patients) and those with intramedullary lesions (16 patients). The patients with intramedullary lesions were further divided into two groups, those with granulomatous disease and those with nongranulomatous disease. The diagnoses were confirmed by a variety of methods, including CSF analysis (organism isolation, antigen specific titers) (five patients: four intradural extramedullary, one intramedullary), open biopsy (six patients: three intradural extramedullary, three intramedullary), and, in patients with tuberculosis, dramatic response to medical therapy on follow-up images (six patients: three intradural extramedullary, three

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intramedullary). The diagnosis was strongly presumed when the disease manifested first in an extraspinal location followed by direct time-related causal involvement of the spine (five patients, all intramedullary) and was considered immune-mediated when the proper clinical history and findings were present (one patient, intradural extramedullary, presumed antigen-antibody reaction to the administration of gamma globulin) or idiopathic when exhaustive CSF and clinical analyses were unable to identify a specific origin (five patients: one intradural extramedullary, four intramedullary). Patients with known multiple sclerosis were excluded.

The patients were imaged on four different MR imaging systems. Eighteen patients were imaged on a 1.5-T magnet, eight on a 0.5-T system, and one each on a 1.0-T and a 2.0-T system. Both short TR (450–650) and long TR (1200–2500) spin-echo sequences were used. Gradient-moment nulling and cardiac gating were used where appropriate. In five patients, all with intradural extramedullary inflammatory disease, long TR sequences were not obtained.

All patients were imaged in the sagittal plane. Axial images were obtained in 23 cases and coronal images were obtained in four cases. A single patient was imaged in both the axial and coronal planes. In the majority of cases the slice thickness varied between 3 and 5 mm, although 7 mm was employed in some instances in the lower-field magnet. Eleven patients were imaged more than once.

Twenty-one of the 28 patients received IV gadopentetate dimeglumine (0.1 mmol/kg), and postcontrast short TR sagittal scans were acquired. The images were reviewed and the results tabulated.

Results

The number of patients in each diagnostic group are tabulated in Tables 1 and 2. The MR findings and associated diagnoses are tabulated in Tables 3 and 4.

Intradural Extramedullary Disease

Twelve patients were found to have intradural extramedullary disease. In these patients, unenhanced scans were usually unrevealing. In two cases no abnormality was identified; in 10 cases there was a questionable abnormality of a nonspecific nature. These nonspecific findings varied from linear strands of soft-tissue intensity in the spinal canal to a complete obliteration of the normal canal space. The location of the lesion was indeterminate and the relationship to the cord or nerve roots unclear. Long TR images were obtained in seven of the 12 patients with intradural extramedullary inflammatory disease. In three of the seven patients, the long TR images masked the intradural abnormality. The dura was considered thickened in four of the seven cases.

TABLE 1: Intradural Extramedullary Spinal Lesions

Diagnosis	No. of Patients
Enterococcal meningitis	1
Syphilitic meningitis	1
Coccidioidomycosis meningitis	1
Aseptic meningitis	2
Sarcoidosis	2
Tuberculous meningitis	5

TABLE 2: Intramedullary Granulomatous and Nongranulomatous Spinal Lesions

Diagnosis	No. of Patients
Sarcoidosis	1
AIDS myelitis	1
Tropical spastic paraparesis (HTLV-1)	1
Acute disseminated encephalomyelitis	1
Herpes zoster myelitis	3
Tuberculosis	3
Inflammatory myelitis of uncertain origin	6

Eleven of the 12 patients with intradural extramedullary disease received gadopentetate dimeglumine. The administration of contrast agent proved to be useful in visualizing the lesion in two patients in whom unenhanced scans were normal and in defining the lesion in the remaining nine patients (Fig. 1). After the administration of contrast material, the lesion could be accurately located in the intradural extramedullary compartment by visualization of the intact spinal cord or nerve roots surrounded by enhancing tissue, creating an impression of a filling defect.

Three patterns of enhancement of the intradural extramedullary lesions were noted. The first pattern was linear enhancement on the surface of the spinal cord and/or nerve roots and was present in nine patients (Fig. 1). The enhancement was delicate and smooth and outlined the underlying nerve roots and spinal cord.

The second mode of enhancement was nodular in nature. This was present in three patients (Fig. 1), and consisted of discrete foci of enhancement on the surface of the spinal cord and/or nerve roots. In two patients there was a combination of nodular and linear enhancement within the intradural compartment.

The third pattern, seen in two patients, consisted of diffuse, thick, intradural enhancement of the leptomeninges, completely surrounding the spinal cord and appearing as an intradural filling defect (Fig. 2). No correlation was found between the pattern of enhancement and the severity of the presenting neurologic symptoms.

Intramedullary Disease

The 16 patients with intramedullary disease were divided into two groups, those with granulomatous disease and those with nongranulomatous disease. In the group of patients with intramedullary granulomatous disease (four patients), each showed the same MR characteristics. Focal spinal cord swelling was identified on the short and long TR images (Figs. 3A and 3B). There was localized high signal intensity on the long TR images in the region of cord swelling. After contrast administration, nodular enhancement within the cord was seen at the site of the suspected abnormality (Fig. 3C). On follow-up MR after 6 months of antituberculous therapy in the patients with tuberculomas, the tuberculomas disappeared and the spinal cord returned to normal.

In the 12 patients with nongranulomatous intramedullary inflammatory disease, a spectrum of MR findings was noted. First, the amount of cord swelling varied from none to diffuse. In the cases in which the spinal cord was diffusely swollen, the swelling extended over numerous spinal segments. In two cases, there were skip areas of uninvolved cord (Fig. 4).

TABLE 3: MR Findings and Final Diagnoses of Patients with Intradural Extramedullary Lesions

Case No.	Sagittal T1	Sagittal T2	Sagittal Postcontrast	Final Diagnosis
1	Possible posterior clumping of nerve roots	Not performed	Enhancing nerve roots, linear coating, nodular enhancement	Syphilitic meningitis: CSF VDRL positive, serum VDRL positive, improved on treatment
2	Loss of cord-CSF interface, high signal in subarachnoid space, featureless thecal sac	Not performed	Intense thick intradural enhancement coating cord and nerve roots	Enterococcal meningitis: enterococci grown from blood, gram positive cocci in CSF smear, improved on treatment
3	Soft tissue in subarachnoid space of similar intensity to cord	Not performed	Nodular and linear coating of cord	Coccidioidomycosis meningitis: coccidioidomycosis grown from CSF cultures
4	Irregular swollen cervical spinal cord	Not performed	Large nodules enhancing densely on cord surface and indenting and possibly invading cervical cord	Sarcoidosis: biopsy proved, improved on steroids
5	Nonspecific fine soft-tissue signal adjacent to distal thoracic cord	Normal thoracic cord and CSF space	Linear enhancement of nerve roots and cord	Sarcoidosis: lymph node biopsy
6	Normal thoracic cord and conus medullaris	Normal	Linear enhancement coating conus medullaris and cauda equina	Aseptic meningitis: spontaneous clinical improvement
7	Normal	Not performed	Linear enhancement coating nerve roots	Aseptic meningitis: presumed immune mediated, patient received gamma globulin prior to illness, clinical improvement on steroids
8	Featureless thecal sac	Thick dura on long TR images	Contrast agent not administered	Tuberculous meningitis: CSF isolation of tuberculous bacilli, endemic area, response to anti-TB treatment
9	Epidural mass, localized increased signal in thecal sac	Thick dura	Localized intradural enhancement, linear enhancement of nerve roots	Tuberculous meningitis: CSF analysis, response to anti-TB treatment, endemic area
10	Epidural mass, localized increased signal in thecal sac opposite epidural mass	High-signal epidural mass, dura not seen	Meningeal enhancement, linear enhancement coating cord	Tuberculous meningitis: CSF analysis, response to anti-TB treatment, endemic area
11	Epidural mass, increased signal in canal at this site	High-signal-intensity epidural mass, thickened dura	Thick dural enhancement, epidural abscess	Tuberculous meningitis: CSF analysis, response to anti-TB treatment, endemic area
12	Epidural mass, possibly thickened dura	High-signal-intensity epidural mass, thick dura	Spondylitis, enhancing dura very thick	Tuberculous meningitis: open biopsy, response to anti-TB treatment

Second, the signal characteristics within the cord on unenhanced scans varied. On unenhanced short TR scans, only once case showed diffuse low signal in the cord. In the other 11 cases, the cord was of normal signal intensity or slightly increased signal intensity on short TR images. The faintly increased signal intensity was only seen in those cases associated with cord swelling. In the two patients in whom there was no cord swelling, no signal abnormality could be identified on the short TR sequence. In all cases, high signal intensity was seen on long TR sequences in a focal or diffuse location (Figs. 4 and 5). There was normal signal intensity in the cord in the apparently uninvolved skip areas (Fig. 4).

Contrast-enhanced scans were available in eight patients with nongranulomatous inflammatory intramedullary lesions. Three patterns of enhancement were seen. More than one enhancement pattern was seen in two patients.

In three patients, diffuse intramedullary enhancement was present (Fig. 4). This diffuse enhancement pattern was associated with a diffusely swollen spinal cord demonstrating abnormal, increased signal on the long TR image. In those

TABLE 4: MR Findings and Final Diagnoses of Patients with Intramedullary Lesions

Case No.	Sagittal T1	Sagittal T2	Sagittal Postcontrast	Final Diagnosis
1	Fusiform swelling of upper thoracic cord	Diffuse high signal in region of cord swelling	Contrast agent not administered	Herpes zoster myelitis: CSF analysis, herpetic pustules in same dermatomal distributions, stable on acyclovir
2	Normal thoracic cord	Multiple focal high signal abnormalities, normal intervening areas	Peripheral cord enhancement	Herpes zoster myelitis: viral isolation, herpetic papule, concomitant myelopathy; CSF analysis; spontaneous slow recovery at rehabilitation center
3	Fusiform swelling of numerous contiguous segments of thoracic cord	Diffuse high signal in swollen region of cord	Multiple small nodular enhancing lesions	Herpes zoster myelitis: zoster skin lesions, some clinical improvement on acyclovir
4	Fusiform swelling of cervical cord, slightly increased signal in swollen cord	Diffuse high signal in swollen region of cord	Peripheral cord enhancement over involved segment	Tropical spastic paraparesis (HTLV-1 myelitis): CSF analysis, lost to follow-up
5	Fusiform swelling of cervical cord	Focal high signal abnormality at C2–C3	Contrast agent not administered	AIDS myelitis: CSF analysis, clinical course, lost to follow-up
6	Minimal fusiform swelling of cervical cord, slightly increased signal focally	Focal high signal	Nodular intramedullary enhancing lesion	ADEM: brain lesions typical of ADEM on MR, clinical history, stable with steroids then lost to follow-up
7	Fusiform swelling of thoracic cord over numerous segments, slightly increased signal in swollen cord	Diffuse high signal in cord over region of swelling	Contrast agent not administered	Inflammatory myelitis: nonspecific CSF analysis, clinical course, MR findings, no evidence of MS, normal MR of brain
8	Fusiform swelling of cervical cord over numerous segments, skip areas, slightly increased signal in swollen cord	Diffuse high signal in swollen regions of cord, normal signal in uninvolved skip areas	No enhancement on initial exam, diffuse enhancement of involved segments at follow-up	Inflammatory myelitis: nonspecific CSF analysis, clinical course, MR findings, no evidence of MS on 1-yr follow-up, spontaneous slow improvement
9	Fusiform swelling of thoracic cord over numerous segments, skip areas, slightly increased signal in swollen cord	Diffuse high signal in swollen region of cord, uninvolved skip areas	Diffuse enhancement of involved segments	Inflammatory myelitis: nonspecific CSF analysis, clinical course, MR findings, no evidence of MS, follow-up MR over 1 yr showed decreased enhancement, slow minimal clinical improvement
10	Fusiform swelling of cervical cord over a few segments	Fusiform high signal in cord corresponding to region of cord swelling	Minimal nodular enhancement within region of cord	Inflammatory myelitis: nonspecific CSF analysis, clinical course, MR findings, no evidence of MS, normal brain MR, clinically improved, returned to work
11	Diffuse fusiform swelling of cervical cord over numerous segments with central low signal, syrinx versus necrotic cord	Intense high signal in cervical cord corresponding to low signal on short TR in central cord	Peripheral cord enhancement followed by full thickness enhancement over a period of 45 min	Inflammatory myelitis: cord biopsy showed inflammatory changes, spontaneous slow recovery

TABLE 4: *Continued*

Case No.	Sagittal T1	Sagittal T2	Sagittal Postcontrast	Final Diagnosis
12	Normal thoracic cord	Diffuse high signal in upper thoracic cord	Multiple enhancing nodules of various size	Inflammatory myelitis: cord biopsy showed inflammatory changes, AIDS, lost to follow-up
13	Fusiform swelling over numerous segments with diffuse low signal	Focal high signal in swollen region of cord	Solid nodular enhancement over two segments	Sarcoidosis: biopsy, improved on steroids
14	Focal enlargement of cervicomedullary junction, low signal focally	Focal high signal with low intensity centrally	Contrast agent not administered	Tuberculoma: endemic area, marked response to anti-TB therapy, complete resolution on follow-up MR
15	Low-intensity signal focally at cervicomedullary junction	Focal high signal with low intensity centrally	Thick rim enhancement with central low signal	Tuberculoma: endemic area, marked response to anti-TB therapy, complete resolution on follow-up MR
16	Focal enlargement of conus, low-intensity signal focally	Focal high signal with low intensity centrally	Thick rim enhancement with central low signal	Tuberculoma: endemic area, marked response to anti-TB therapy, complete resolution on follow-up MR

patients with skip areas, the spinal cord enhanced over the entire length of the enlarged segments but failed to enhance in areas of normal intervening cord. In two cases, the initial contrast-enhanced MR study showed little or no spinal cord enhancement despite fusiform swelling and associated diffuse high signal over numerous segments on long TR scans. On subsequent follow-up MR scans several weeks later, the contrast-enhanced scans showed clear diffuse enhancement in the spinal cord in the abnormally enlarged areas.

In two patients peripheral enhancement within the cord was

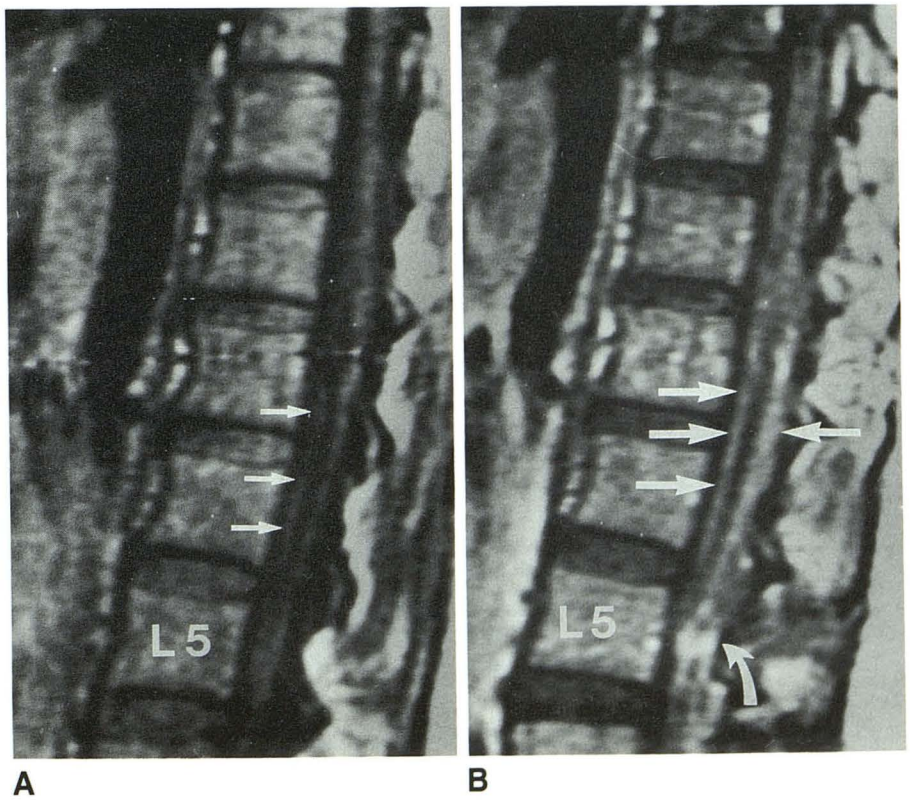


Fig. 1.—Case 1 (Table 3): 33-year-old woman, 3 months postpartum, who developed bilateral leg weakness and radiculopathy. Syphilitic meningitis proved by CSF VDRL.

***A* and *B*, Sagittal pre- (*A*) and post- (*B*) contrast T1-weighted images (600/20/4) of lumbar spine. Precontrast image (*A*) shows subtle clumping of nerve roots posteriorly (*arrows*). Postcontrast image (*B*) shows linear enhancement (*straight arrows*) of nerve roots and possibly the dura, as well as nodular enhancement distally (*curved arrow*).**

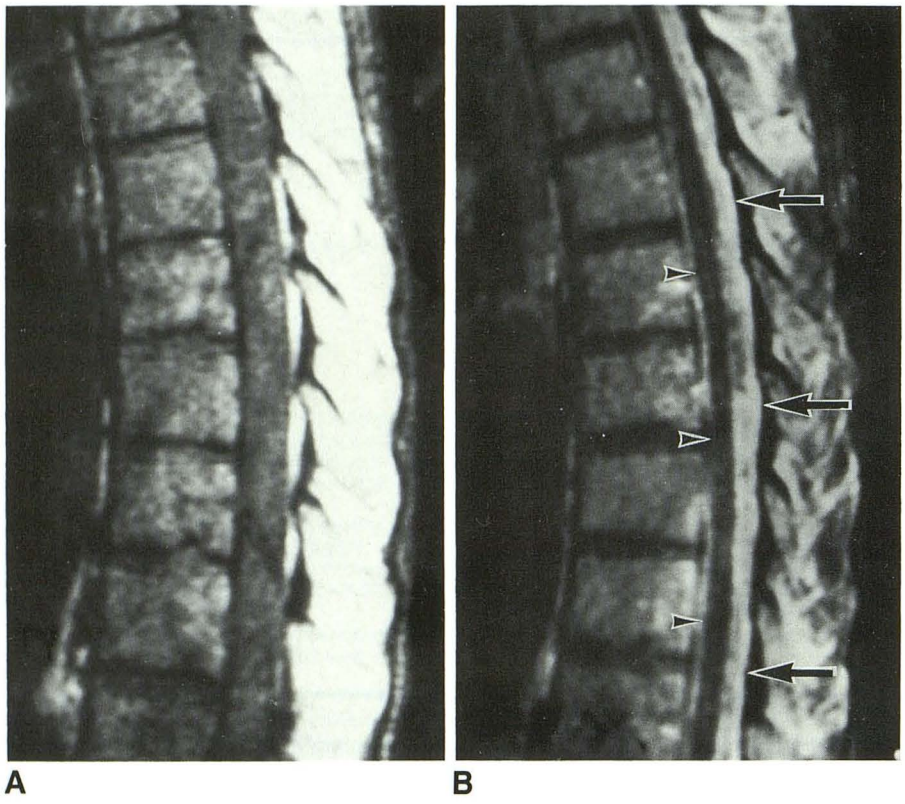


Fig. 2.—Case 2 (Table 3): 26-year-old man with fever who subsequently developed sepsis and quadriparesis. Gram positive cocci in CSF smear and enterococci grown from blood confirmed enterococcal meningitis.

A and *B*, Sagittal pre- (*A*) and post- (*B*) contrast T1-weighted images (400/20/2) of thoracic spine. Precontrast image (*A*) shows loss of normal spinal cord-CSF interface and poor characterization of the type or location of the abnormality. On postcontrast image (*B*), diffuse, thick, intradural enhancement (*arrows*) outlining the spinal cord (*arrowheads*) locates the disease in the intradural extramedullary compartment.

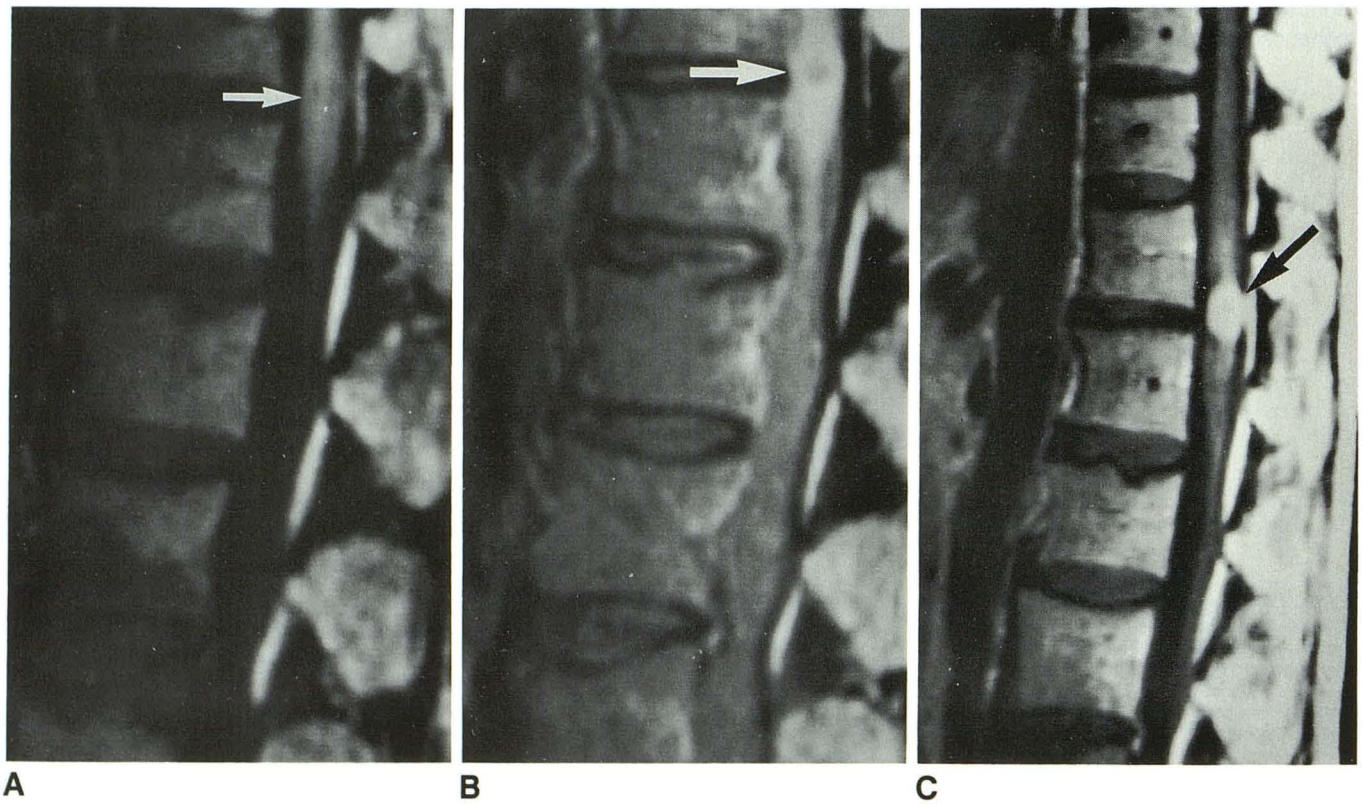


Fig. 3.—Case 16 (Table 4): Conus medullaris tuberculoma in male patient who presented with progressive urinary retention and leg weakness.

A, Sagittal precontrast T1-weighted image (450/30/6) shows focal enlargement of conus (*arrow*).

B, Precontrast T2-weighted image (1800/50/2) shows a nodular high-signal-intensity lesion in conus with surrounding poorly defined higher signal (*arrow*).

C, Sagittal postcontrast T1-weighted image shows nodular enhancement (*arrow*). In addition, faint enhancement of uncertain origin may be present inferiorly and superiorly to the lesion.

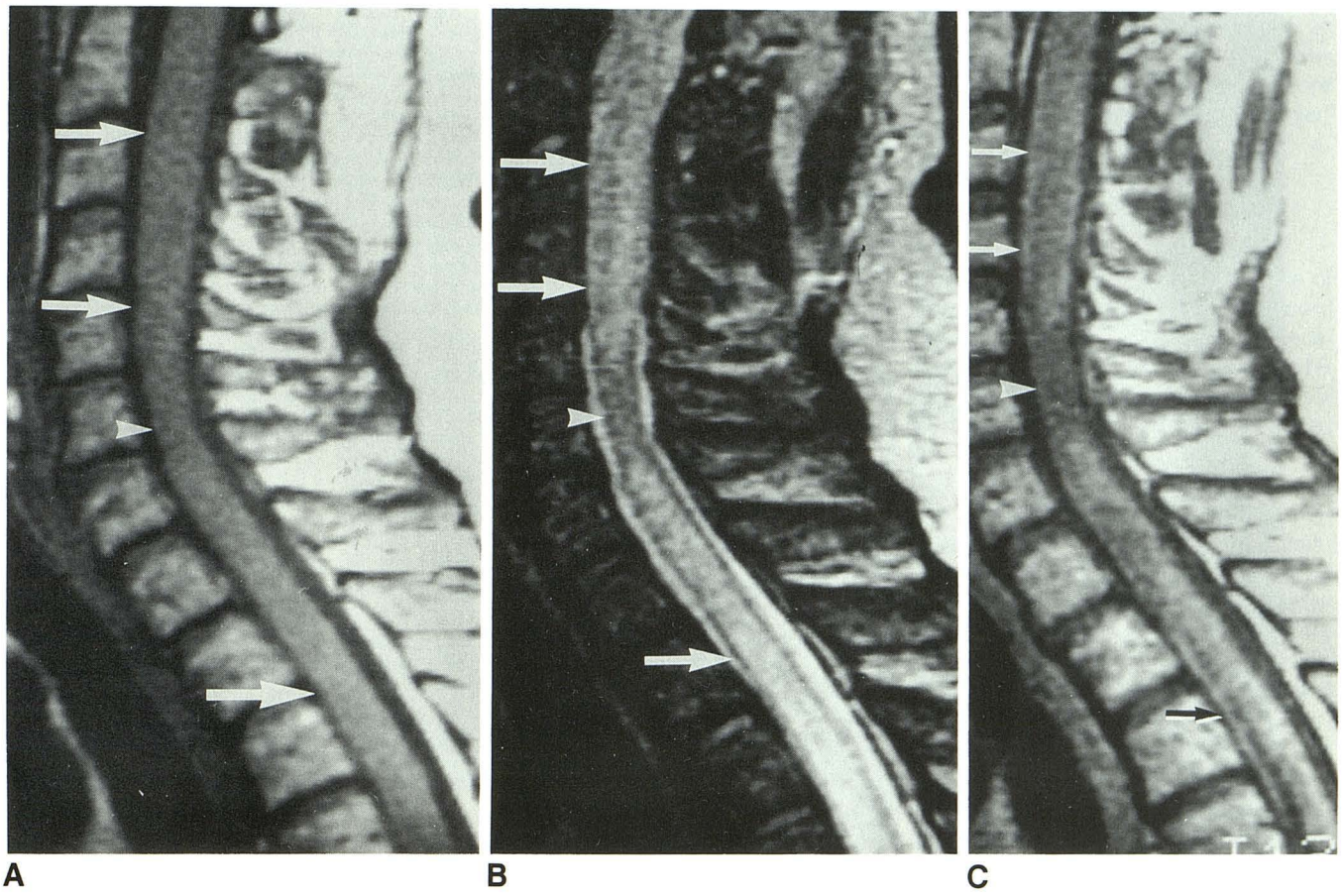


Fig. 4.—Case 8 (Table 4): Inflammatory myelitis in 45-year-old man who presented with chest and abdominal numbness.

***A*, Sagittal precontrast T1-weighted image (600/20/4) shows fusiform swelling of noncontiguous segments of cervical cord (*arrows*), which are separated by a short normal intervening skip segment (*arrowhead*) of cord.**

***B* and *C*, Swollen areas of spinal cord demonstrate diffuse high signal on precontrast T2-weighted image (2200/80/2) (*arrows* in *B*) and diffuse enhancement after contrast administration (*arrows* in *C*) with a nonenhancing skip area (*arrowhead*). This pattern is unlike that usually seen in tumor.**

seen (Fig. 6). The unenhanced short and long TR MR scans showed fusiform swelling of the spinal cord over numerous segments and diffuse high signal on the long TR images. The immediate postcontrast scans showed peripheral enhancement but in one of these cases a delayed scan was performed, which showed progressive enhancement of the spinal cord toward the center (Fig. 7).

Finally, a nodular enhancement pattern within the spinal cord was identified in four cases (Fig. 5). The amount of cord swelling varied from none to diffuse. In one case of acute disseminated encephalomyelitis, the unenhanced short TR scan was normal despite a focal nodular high-signal-intensity intramedullary lesion on the long TR scan. This focal area demonstrated intense enhancement after contrast administration. In the remaining three cases the enhancement consisted of multitudinous small foci of varying size, all smaller than 4 mm in diameter. No dominant nodule was seen. The enhancement occurred within the cord in the region of cord swelling or signal abnormality only. One patient in this group had three follow-up MR examinations over a 14-month period, all of which showed a decrease in the enhancement in the spinal cord (Fig. 5) associated with clinical improvement.

Discussion

Intradural inflammatory diseases of the spine include meningitis and myelitis of diverse origin. Patients who present with isolated acute or subacute neurologic symptoms referable to the spinal cord require prompt attention. Initial investigation is aimed at identifying surgical from nonsurgical lesions [11], and imaging studies, especially MR, assist in making this distinction. The administration of contrast material allows the definitive identification of lesions and subsequent characterization.

Intradural Extramedullary Disease

In patients with inflammatory intradural extramedullary lesions, unenhanced MR scans were either normal or unable to help localize the abnormality to one of the three spinal compartments. Contrast-enhanced MR imaging was essential in some cases (two patients) for visualizing the abnormality and in others for localizing the inflammation to the intradural extramedullary space and for defining the extent of the disease.

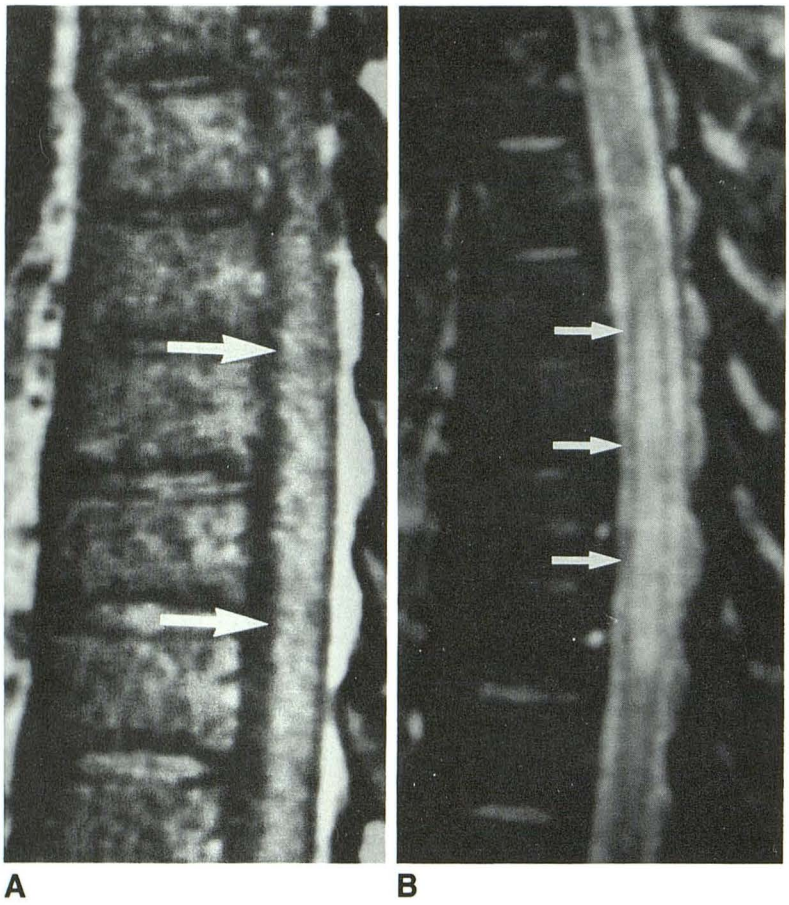
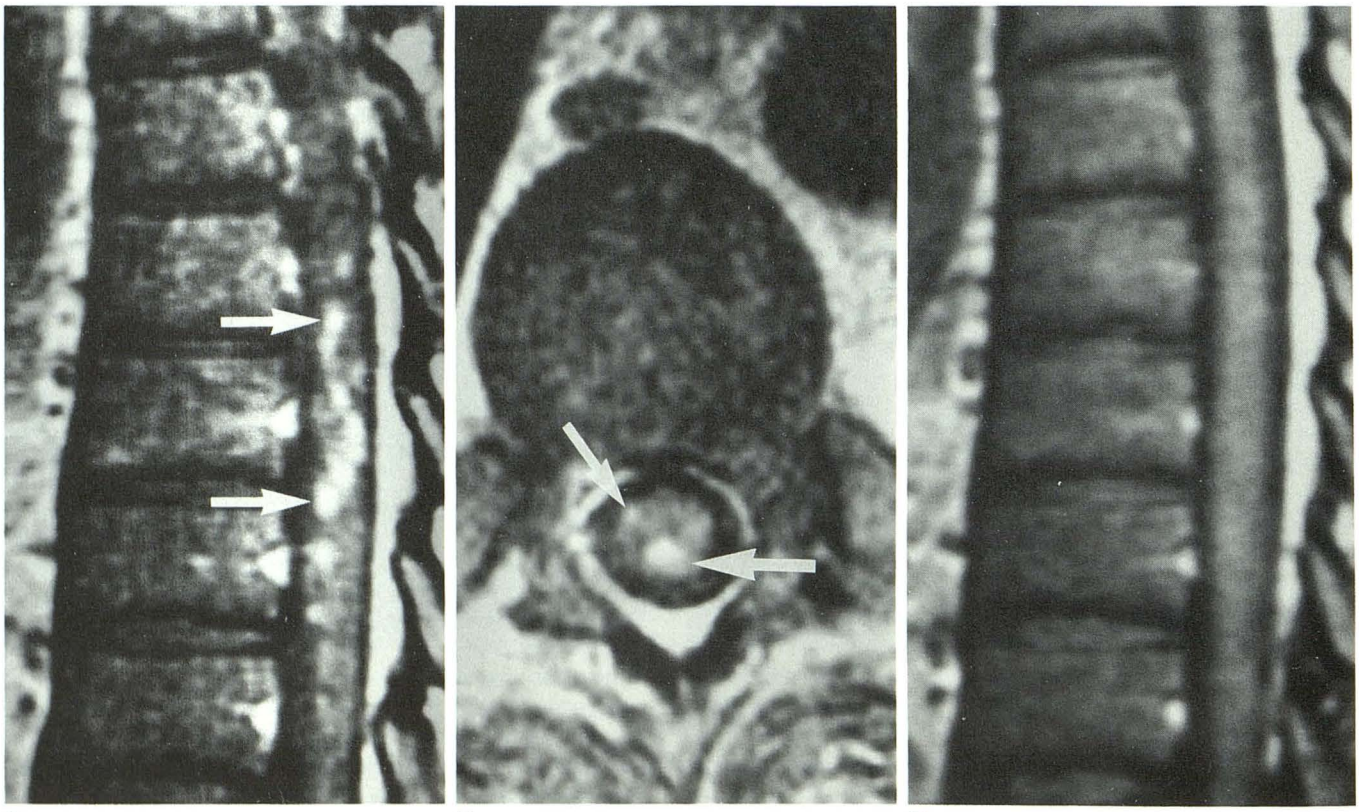


Fig. 5.—Case 3 (Table 4): Herpes zoster myelitis in 41-year-old man with progressive urinary retention and bilateral leg weakness. The patient had concomitant herpes zoster skin papules in some of the involved dermatomes. Acyclovir therapy resulted in moderate clinical improvement.

A and *B*, Sagittal precontrast T1-weighted (600/30/4) (*A*) and T2-weighted (1500/50/2) (*B*) images show minimal swelling of thoracic cord (*arrows* in *A*), although high signal in spinal cord is present on T2-weighted image (*arrows* in *B*). Some subtle high signal, possibly due to punctate hemorrhage, may also be present on T1-weighted image.

C and *D*, Sagittal (*C*) and axial (*D*) postcontrast T1-weighted images (600/30/4) disclose a speckled pattern of enhancement (*arrows*).

E, Follow-up postcontrast T1-weighted image (600/30/4) obtained after therapy shows decreased enhancement within spinal cord.



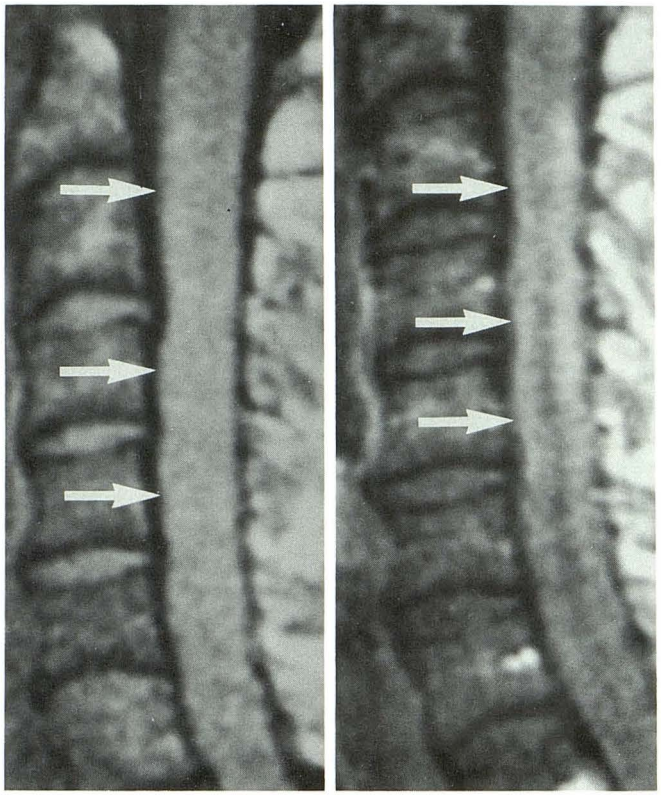


Fig. 6.—Case 4 (Table 4): Tropical spastic paraparesis (HTLV-1 myelopathy) in 34-year-old man from Barbados who presented with progressive quadriparesis, proved by CSF titers.

***A*, Sagittal precontrast T1-weighted image (600/20/4) shows diffuse swelling of cervical spinal cord (*arrows*).**

***B*, Sagittal postcontrast T1-weighted image (600/20/4) shows peripheral enhancement of cord (*arrows*).**

The appearance and location of the enhancement in the cases of intradural extramedullary disease make it likely that the enhancing structures were inflamed meninges, similar in appearance to intracranial meningeal enhancement [12]. The three patterns of enhancement identified (linear enhancement coating the nerve roots and spinal cord, nodular enhancement on the surface of the spinal cord, or nerve roots and thick intradural enhancement completely filling the subarachnoid space) most likely reflect the degree and nature of the meningeal inflammation. Cranial meningeal enhancement has been shown to correlate well with severe meningeal inflammation on histopathologic sections; areas of less intense meningeal inflammation histologically did not exhibit enhancement [12]. While this trend may be the same in the meninges of the spine, in this series no correlation was found between the patterns of enhancement—that is, linear, nodular, or thick intradural—and the initial severity of neurologic symptoms.

All the enhancement patterns noted in the patients with intradural extramedullary inflammatory changes have been seen in patients with leptomeningeal spread of tumor; the MR findings alone could not differentiate infection from tumor [13]. Although the IV administration of gadopentetate dimeglumine greatly increased the sensitivity of MR in detecting an abnormality and localizing it to the intradural extramedullary compartment, it did not improve the diagnostic specificity. In addition, no enhancement pattern characterized a specific inflammatory origin. Fine linear enhancement on the cord surface was seen in patients with lesions varying from sarcoidosis to bacterial meningitis.

Intramedullary Disease

In the subgroup of patients with intramedullary disease of granulomatous origin, the MR characteristics were focal cord swelling, high signal on long TR images, and nodular enhancement with contrast administration. On both the long TR images and the contrast-enhanced scans, differentiation of granulomatous disease from an intramedullary tumor could not be made.

In the subgroup of patients, however, with nongranulomatous intramedullary lesions, the MR characteristics were often sufficiently unique to suggest the probability of an inflammatory intramedullary lesion and to make consideration of an intramedullary tumor less likely. Of course, the clinical presentation alone is often suggestive. Patients with inflammatory myelitis usually present acutely in contradistinction to the usual insidious onset of symptoms associated with spinal cord tumors, although exceptions in both instances do occur. MR, however, can further help in the differentiation of the two. Cord tumors are generally characterized by a significant amount of cord swelling, which is usually of low intensity on short TR images and high intensity on long TR images. If hemorrhage is present, it is usually focal. After the administration of contrast agent, focal nodular enhancement is usually seen. Of the 55 reported gliomas of the cord studied with contrast material, 54 enhanced [14–19]. Nonenhancing gliomas appear to be comparatively unusual. In addition, gliomas that enhance in a peripheral, diffuse, or speckled pattern have not been described.

In our study, nongranulomatous myelitis was characterized on MR by two sets of findings. In the first set, there was diffuse swelling of the spinal cord over numerous segments, sometimes with skip areas, and a subtle increase in the signal of the cord on short TR scans, possibly due to petechial hemorrhage. This was accompanied by a diffuse high signal on long TR scans in the enlarged areas of the spinal cord. In all cases except one, diffuse, peripheral, or speckled enhancement was seen. Any of these enhancement patterns would be most unusual in cases of intramedullary tumor where large nodular enhancement is generally seen. The presence of skip areas would also be atypical. In this number of cases, if these were tumors, one might also have expected to see cases with definite cyst formation.

In the second set of findings that characterized nongranulomatous myelitis, the cord did not show enlargement on the short TR scans. Focal areas of high signal intensity were seen on the long TR scans and nodular enhancement after contrast administration was noted in these regions. Tumor was considered less likely in these cases owing to the fact that there was no swelling despite the relatively large focal lesion.

Pathologically, patients with myelitis exhibit perivascular inflammation, primarily perivenular lymphocytic infiltration [20, 21]. The myelin sheaths adjacent to the perivascular inflam-

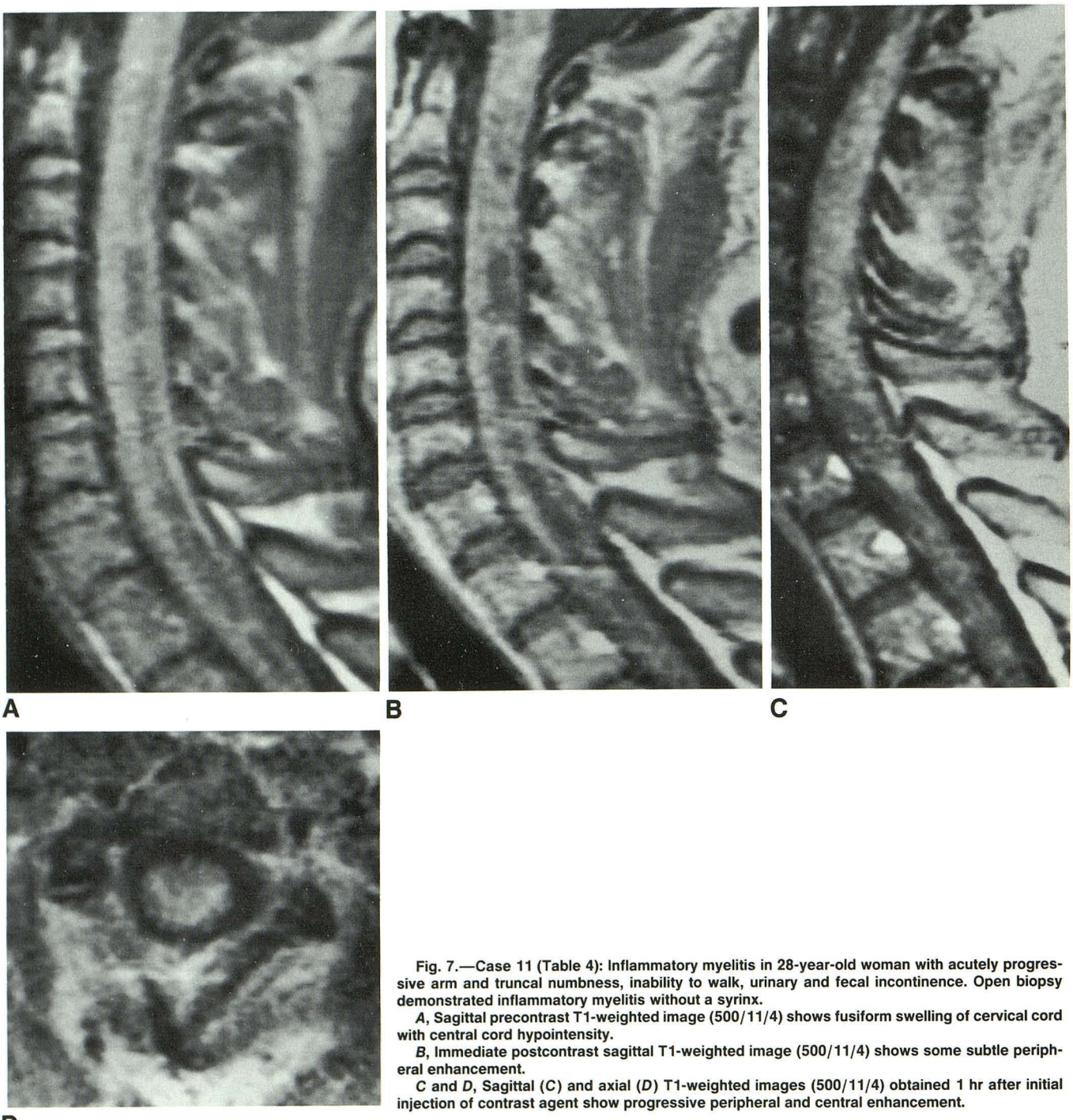


Fig. 7.—Case 11 (Table 4): Inflammatory myelitis in 28-year-old woman with acutely progressive arm and truncal numbness, inability to walk, urinary and fecal incontinence. Open biopsy demonstrated inflammatory myelitis without a syrinx.

***A*, Sagittal precontrast T1-weighted image (500/11/4) shows fusiform swelling of cervical cord with central cord hypointensity.**

***B*, Immediate postcontrast sagittal T1-weighted image (500/11/4) shows some subtle peripheral enhancement.**

***C* and *D*, Sagittal (*C*) and axial (*D*) T1-weighted images (500/11/4) obtained 1 hr after initial injection of contrast agent show progressive peripheral and central enhancement.**

mation show degradation with relative sparing of the axons. An inflammatory exudate bathes this process. Ischemia may occur in association with arterial spasm in the pia or other spinal arteries and may result in myelomalacia [5, 22, 23]. In the chronic stage, the inflammatory exudate is replaced by fibrous gliosis. Depending on the severity of the inflammation and the time from onset of the disease, cord swelling may or may not be seen.

These pathologic findings correlate with the MR findings encountered in this study. Diffuse swelling of the cord or no swelling of the cord was seen on short TR images. High signal intensity seen on T2-weighted images most likely reflected the inflammatory exudate, edema, and ischemia in the acute and subacute cases or areas of gliosis in the more chronic examples.

In this series, initial enhancement was not seen or was

minimal in two patients despite marked abnormalities on the unenhanced sequences. Follow-up scans, however, within a few weeks depicted marked enhancement. Thus, findings in patients with myelitis may progress fairly rapidly, unlike the usual appearance of tumors. In addition, delayed scans may also be of use. The immediate postcontrast image may demonstrate only peripheral cord enhancement, with progressive central enhancement seen later (Fig. 7).

The identification of a causative agent producing myelitis remains difficult. CSF analysis, specifically viral antigen titers and viral cultures, are helpful if positive but often take weeks to return. Even open biopsy, as shown in two of our cases in which the preoperative diagnosis was intramedullary tumor, demonstrated nonspecific changes consistent with myelitis; isolation of the causative agent was unsuccessful.

One of the dilemmas facing clinicians involves the determination of whether an acute myelitis may be the initial presentation of multiple sclerosis (MS). MS as described by Poser and colleagues [24] is a multiphasic disease. In as many as one third of patients, the initial presentation of MS, however, may be an isolated spinal cord syndrome [25]. Hence, an acute myelitis may herald the onset of MS. On the basis of spinal MR findings alone, myelitis resulting from MS could not be differentiated from myelitis of an infectious or inflammatory origin in patients presenting with isolated spinal cord syndrome. This is not surprising considering that MS also induces an inflammatory condition. The presence of coterminous white matter lesions in the brain, however, at the time of initial presentation is known to increase the likelihood of MS as the eventual diagnosis [26].

Of our 16 patients with inflammatory cord lesions, 10 were given a specific diagnosis. Of the remaining six, five showed no evidence of MS on clinical follow-up a year later. Their myelopathic symptoms were stable or improved and they developed no symptoms referable to previously uninvolved portions of the CNS. In addition, two had negative MR studies of the brain. One was lost to follow-up.

In conclusion, contrast-enhanced MR images proved essential for visualization, characterization, and localization of intradural extramedullary inflammatory diseases; however, the enhancement patterns were nonspecific. In patients with granulomatous cord lesions, MR disclosed focal lesions in the spinal cord but differentiation from tumor was difficult. In patients with nongranulomatous cord lesions, two sets of MR findings that were sufficiently characteristic to suggest infection or inflammation rather than tumor were identified.

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