Idiopathic transverse myelitis: MR characteristics.

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Idiopathic Transverse Myelitis: MR Characteristics

Kyu Ho Choi, Kwang Soo Lee, Sun Ok Chung, Jung Mee Park, Young Joo Kim, Hyun Sook Kim, and Kyung Sub Shinn

PURPOSE: To describe the MR characteristics that can distinguish idiopathic transverse myelitis from other intramedullary lesions. **METHODS:** A total of 32 initial and follow-up MR studies in 17 patients with clinically proved transverse myelitis were reviewed retrospectively. The location, size, pattern, and segmental length of areas of hyperintensity were estimated on T2-weighted axial and sagittal images. In 15 of the patients, whose neurologic abnormalities were limited to the spinal cord, the location and pattern of intramedullary contrast enhancement were evaluated on sagittal and axial T1-weighted images. Follow-up MR studies were available for 10 patients. The statistical significance of cord enhancement between the groups with and without cord expansion was calculated. **RESULTS:** Common MR findings of idiopathic transverse myelitis included a centrally located hyperintensity occupying more than two thirds of the cross-sectional area of the cord (88%); a length of 3 to 4 vertebral segments (53%), with variable presence of cord expansion (47%); a small central area of intensity, isointense with normal cord, in the core of hyperintensity (47%); focal, peripheral cord enhancement (53%), particularly in patients with cord expansion; and a slow regression of T2 hyperintensity with an enhancing nodule. Although no linear correspondence was observed between MR findings and neurologic signs and symptoms, all but 4 patients improved clinically as MR findings improved or remained stable. **CONCLUSIONS:** MR findings are helpful in detecting transverse myelitis and in differentiating this entity from multiple sclerosis and cord tumors, but clinical assessment and observation of MR changes over time are essential in making the diagnosis.

Index terms: Myelitis; Spinal cord, magnetic resonance

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The spinal cord is the site of infectious and noninfectious inflammatory processes. Transverse myelitis, or myelopathy, is diagnosed when both halves of the cord are involved with an inflammatory process (1, 2). The syndrome has multiple causes; it interrupts both motor and sensory tracts at one level, commonly the thoracic; and it is known to be associated with various viral infections, vaccinations, autoimmune diseases, and carcinomas, although most cases are idiopathic (3).

Reports of the magnetic resonance (MR) imaging findings in patients with transverse myelitis, specifically in the acute stage, have described local enlargement of the cord and increased signal intensity on long repetition time/echo time (TR/TE) sequences (1, 2). However, these findings are not sufficient to differentiate this condition from other similar lesions, such as intramedullary tumors, compressive spinal cord disease, multiple sclerosis, hematoma, and vascular ischemia. In particular, there is a paucity of published material on the prevalence and pattern of contrast enhancement in patients with transverse myelitis. To help differentiate transverse myelitis from other intramedullary abnormalities, we sought findings on unenhanced and enhanced MR images that were characteristic of transverse myelitis.

Materials and Methods

The initial MR studies of 17 patients with clinically proved transverse myelitis were reviewed retrospectively.

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Follow-up MR examinations, available for 10 of these patients, were also reviewed. The study group consisted of 16 men and 1 woman whose ages ranged from 28 to 56 years (mean age, 41 years). Strict exclusion criteria included a history of spinal trauma, compressive myelopathy, cardiac disease, aortic aneurysm, a space-occupying lesion in the spinal cord or spine, systemic malignancy, syringomyelia, multiple sclerosis, collagen-vascular disease, and respiratory infection or common cold within a few months of the onset of the neurologic manifestation of myelitis. No patient had received a vaccination in recent years.

Routine laboratory studies included complete blood cell count, fluorescent antinuclear antibodies test, rheumatoid factor, VDRL test, tests for vitamin B_{12}, folate, and cerebrospinal fluid that included white blood cell with differentiation, protein, sugar, gram stain, acid-fast bacillus, India ink, enzyme-linked immunosorbent assay for parasites, electrophoresis, myelin basic protein, human T-cell leukemia/lymphotropic virus-1, and viral titer. If one of these laboratory studies, except for lymphocytic-pleocytosis (9 to 130/μL) and/or elevated protein (0.050 to 0.139 g/dL) in cerebrospinal fluid, was positive, the patient was excluded from the study. All patients had a radiographic series of the thoracic and/or cervicothoracic spine, depending on the level of neurologic deficit. No positive plain radiographic finding was found in any patient who was included in the study. Inclusion in the study required the presence of an area of high signal intensity in the cord on axial and sagittal T2-weighted MR images.

Neurologists obtained the patients' histories and performed the neurologic examinations. All initial MR images were obtained immediately after the patient's first visit to the hospital for neurologic disorders and before the start of steroid therapy. A follow-up MR study was considered necessary when the patient did not improve with steroid therapy after approximately 4 weeks or when the clinical manifestations worsened during the observation period. Patients who did not have a follow-up MR examination either recovered sufficiently to refuse further study or failed to visit the clinic.

MR studies of the brain were obtained in two patients who had nonspecific symptoms, such as headache or dizziness, in order to exclude possible intracranial lesions. Brain MR imaging was not performed in the other 15 patients because their neurologic manifestations were limited to the spinal cord. MR imaging of the spine was done on a 1.5-T unit. T1-weighted (350–550/11–17/2) (TR/TE/excitations) and double-echo proton density–weighted and T2-weighted (1800–2200/30,90/2) spin-echo images were obtained with a 3-mm section thickness, 1-mm intersection gap, 256 × 192 matrix size, 32-cm field of view for the sagittal plane, and 20-cm field of view for the axial plane. In addition, in 15 patients, axial and sagittal T1-weighted spin-echo images were obtained immediately after intravenous infusion of 0.1 mmol/kg gadopentetate dimeglumine.

The interval between the onset of neurologic symptoms and signs and the initial MR examination varied from 3 days to 4 months. Our cases were arbitrarily classified according to the time between onset of the neurologic deficit and the initial MR study: less than 4 weeks, acute (five patients); between 4 and 8 weeks, subacute (six patients); and more than 8 weeks, chronic (six patients). The vertebral segmental length of the high signal was estimated on the basis of findings on sagittal T2-weighted images. Cross-sectional location, size, and pattern of the high signal in the cord were determined by findings on the axial T2-weighted images. Evaluation of the abnormal signal intensity of the cord, segmental extent, and contrast enhancement was based on the initial MR examination; for the follow-up studies, these features were compared with the initial findings. The central linear high signal intensity that was, in some cases, seen above and below the diffuse high-intensity area on T2-weighted images was not considered in determining the extent of the abnormality. Cord expansion was evaluated with respect to adjacent normal cord on T1-weighted sagittal images.

A total of 32 MR studies were obtained, including follow-up studies (6 patients had 1 follow-up, 3 had 2 follow-ups, and 1 had 3 follow-ups). The time from initial examination to first follow-up ranged from 20 days to 7 months; from initial examination to last follow-up, the time ranged from 3 to 13 months. The location and extent of the intramedullary contrast enhancement were confirmed on contrast-enhanced axial and sagittal T1-weighted images. Usual treatment for the patients consisted of methylprednisolone 1 g per day for 3 to 5 days followed by prednisone 1 mg/kg for 1 to 2 months, depending on the patient's clinical response.

To determine the differences in MR findings between transverse myelitis and cord tumors, we evaluated the MR findings of 13 patients with proved cord tumor. These included 6 cases of ependymoma, 6 cases of astrocytoma, and 1 case of intramedullary metastasis from lung carcinoma in 9 men and 4 women who had a mean age of 36 years. The cervical cord was involved in 6 patients and the thoracic cord in 7 patients (2 cervical and 4 thoracic ependymomas, 4 cervical and 2 thoracic astrocytomas, and 1 thoracic metastasis). For better comparison, we selected the patients in whom MR studies were performed on a 1.5-T unit with the same parameters as those used for our patients with transverse myelitis. Sagittal and axial T1-weighted images were obtained immediately after intravenous infusion of 0.1 mmol/kg gadopentetate dimeglumine in all patients. The MR studies were examined for cord expansion on T1-weighted sagittal images; for segmental body length of the high signal on T2-weighted sagittal images; for cross-sectional location, size, and pattern of the high signal on T2-weighted images; and for the location, extent, and pattern of contrast enhancement on T1-weighted axial and sagittal images.

Results

The high signal intensity on axial T2-weighted images was centrally located in all patients and was well marginated in all but 5

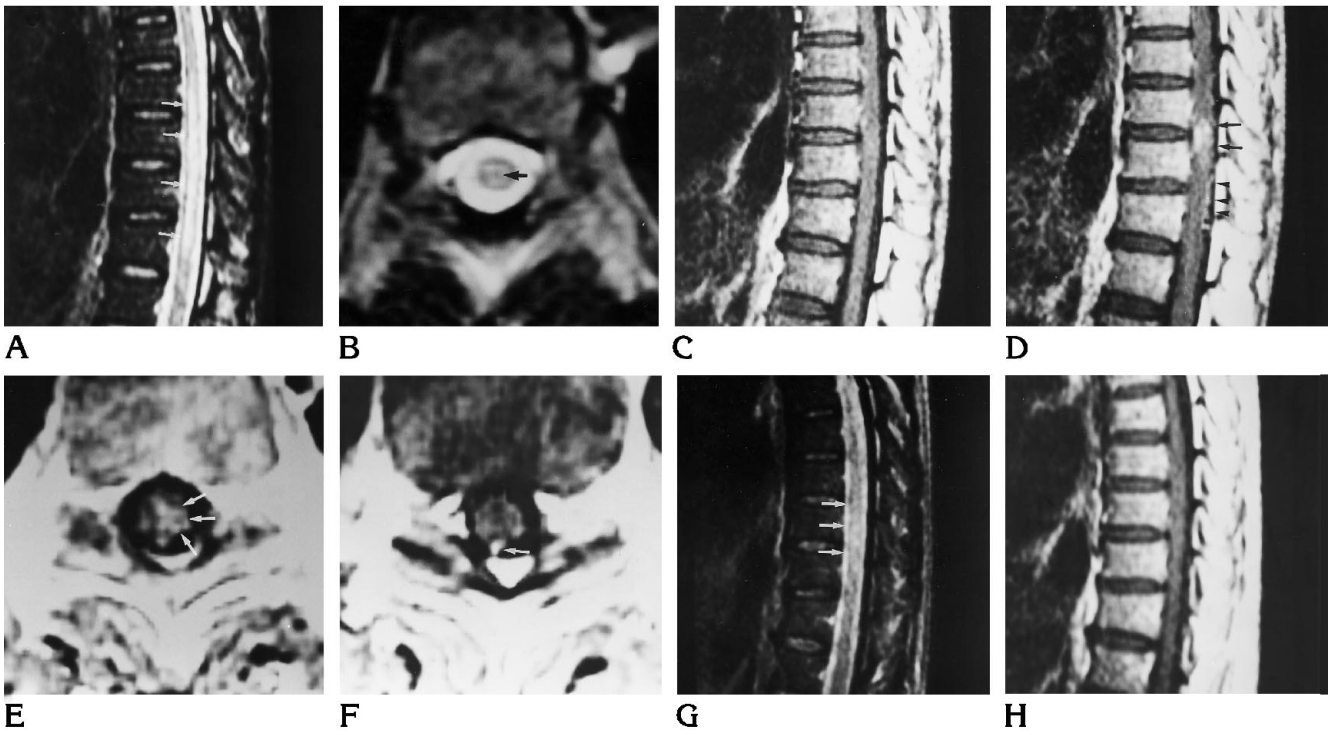

Fig 1. Case 4: 48-year-old man with a 6-month history of sensory dysesthesia below the T-10 level and newly developed motor impairment of the left lower extremity.

A, Sagittal T2-weighted image of the thoracic spine shows long segmental intramedullary high signal intensity (*arrows*).

B, On axial T2-weighted image, hyperintensity is located centrally and occupies more than two thirds of the cross-sectional area of the cord, and a central dot sign (*arrow*) is seen.

C, On sagittal T1-weighted image, no segmental enlargement is evident.

D, Postcontrast sagittal T1-weighted image reveals a poorly marginated area of contrast enhancement in the cord (*arrows*). The dorsally located enhancement along the posterior surface of the cord below the cord lesion (*arrowheads*) most likely represents a prominent posterior medullary vein.

Postcontrast axial T1-weighted images through the cord lesion and dorsally located enhancement confirm intramedullary enhancement (*arrows* in *E*) and posterior medullary vein (*arrow* in *F*).

G, Six months later, sagittal T2-weighted image shows markedly improved segmental intramedullary hyperintensity (*arrows*).

H, The intramedullary contrast-enhancing area has resolved on this postcontrast sagittal T1-weighted image.

patients; it occupied more than two thirds of the cross-sectional area of the cord in 15 of the 17 patients (less than half the cross-sectional area in the other 2). In 8 of the 15 patients who had a large area of central hyperintensity, a small dot, isointense with the cord, was present in the core of the hyperintensity (central dot sign, Fig 1).

On the first follow-up MR studies (10 patients), the extent of the area of high T2 signal intensity in the cord either improved or remained unchanged. Of the 4 patients who had no interval improvement, 2 patients had the same extent for 4 and 6 months, respectively, and the other 2 patients had eventual improvement on later follow-up studies.

Expansion of the spinal cord was defined better on sagittal than on axial T1-weighted images. Cord expansion was observed in eight patients, tapered smoothly to the normal cord, and was of lesser extent than the high T2 signal abnormality (Fig 2). The difference in degree of cord expansion among the eight patients was minimal, with the cerebrospinal fluid intensity around the cord clearly visible on the axial images in all cases.

Among the 15 patients who had contrast-enhanced studies with the initial MR examination, 8 patients had a clear contrast-enhancing area on both axial and sagittal T1-weighted images. The enhancement was nodular (6 patients) or diffuse (2 patients), occupying a portion of the segmental cord, eccentric on one side, and did not bulge outside the contour of

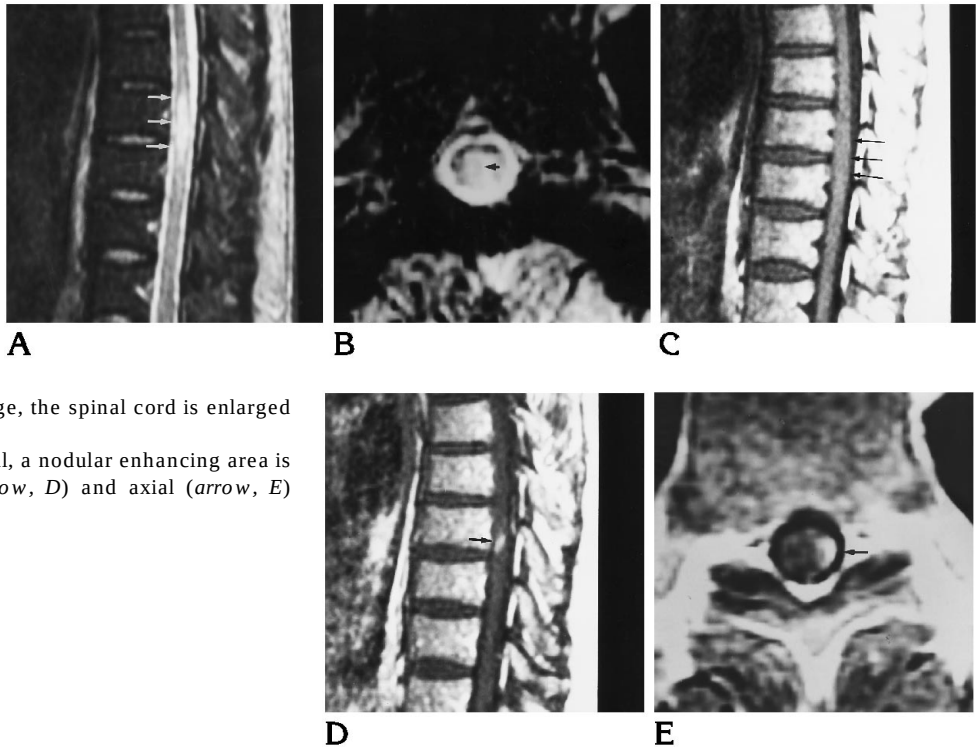

Fig 2. Case 7: 42-year-old man with intolerance to cold, progressive weakness of both lower extremities, and difficulty urinating for 5 months.

A, Sagittal T2-weighted image of the thoracic spine shows an area of intramedullary high signal intensity at the T8–9 level (*arrows*).

B, On axial T1-weighted image, high signal intensity is seen to be associated with a faintly seen central isointensity (*arrow*).

C, On sagittal T1-weighted image, the spinal cord is enlarged at the same level (*arrows*).

After infusion of contrast material, a nodular enhancing area is noted in the cord on sagittal (*arrow*, *D*) and axial (*arrow*, *E*) T1-weighted images.

the cord (Fig 3). Enhancing lesions were located at or near the midpoint of the segmental T2 hyperintensity. The frequency of positive contrast enhancement was greater in patients in the subacute stage (4 of 6 patients) than in those in the acute (1 of 4) and chronic (3 of 5) stages. Among the 8 patients who had follow-up contrast-enhanced studies 1 to 7 months after the initial study (4 patients had enhancement and 4 had no enhancement at the initial study; 3 patients had 2 or 3 follow-up MR examinations), 3 patients with positive contrast enhancement had interval improvement in the size of the enhancement (3 months later in 1 patient and 7 months later in 2 patients). One patient with positive enhancement had progression 3 months later at the second follow-up; and 2 months after that, the progression was further improved (Fig 4). Among the 4 patients in whom there was no initial contrast enhancement, abnormal enhancement was absent in 3 patients and the other patient had a new enhancing nodule at the 1-month follow-up followed by improvement at the 2-month follow-up. No connection could be found between progression or new appearance of an enhancing area and the time course of neurologic features. Despite temporary marked clinical progression in 2 patients, the initial enhancing lesion was improved in 1 patient on the first follow-up study and showed persistent lack of enhancement by the second month in the other patient.

Six of the eight patients with cord expansion had initial contrast-enhanced MR studies. Enhancement was seen in five of these patients, and no enhancement was present in the other patient. Of nine patients with no cord expansion, three had enhancement and six had no enhancement. The frequency of cord enhancement in the group with cord expansion was significantly greater than in the group without cord expansion, but our analysis is based on a small number of cases. The findings are summarized in the Table.

MR findings of cord tumors were significantly different from those of transverse myelitis. In all patients with cord tumors, the cord expansion was present and accompanied a heterogeneous high T2 signal intensity that occupied the entire cross-sectional area of the cord. The average segmental length of the high signal on T2-weighted sagittal images was 4.5. Cavitary lesions were associated in six cases of ependymoma and in two cases of astrocytoma. In two patients, a small central area of intensity, isointense with the cord, was seen outside the area of contrast enhancement in the core of hyperintensity on axial T2-weighted images. Intratu-

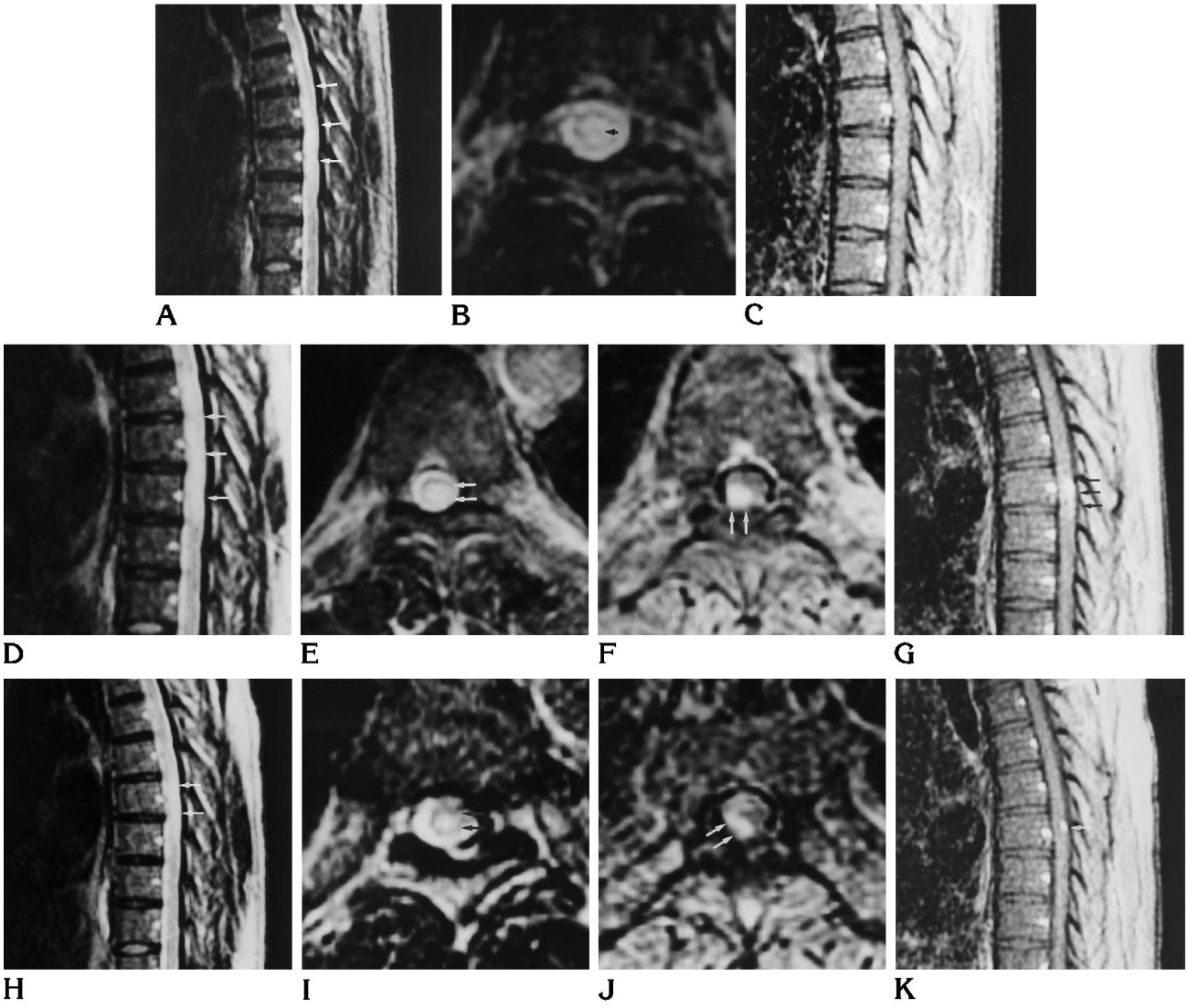

Fig 3. Case 15: 48-year-old man with left leg paresthesias for 3 months and right leg weakness for 3 days.

A, Sagittal T2-weighted image of the thoracic spine shows an area of intramedullary hyperintensity at the midthoracic level (*arrows*). A herniated disk is present three levels below the intramedullary abnormal signal compressing the cord.

B, On axial T2-weighted image, a small area of isointensity relative to the cord (*arrow*) is seen in the center of the large intramedullary hyperintensity.

C, Postcontrast sagittal T1-weighted image shows no definitive enhancing area.

Sagittal (*D*) and axial (*E*) T2-weighted images at 1-month follow-up show almost the same extent and size of high signal intensity in the cord (*arrows*).

Postcontrast axial (*F*) and sagittal (*G*) T1-weighted images show an eccentric contrast-enhancing area in the cord (*arrows*).

Two and a half months later, the extent of high signal in the cord has reduced on a sagittal T2-weighted image (*arrows, H*), and the significant central hyperintensity is still present with better visibility of the cord along the periphery on axial T2-weighted images (*arrows, I*). The contrast-enhancing area has reduced on postcontrast axial (*arrows, J*) and sagittal (*arrow, K*) T1-weighted images. The patient improved clinically as the contrast enhancement lessened.

moral T2 hypointense areas that suggest presence of hemosiderin deposition, a result of old bleeding, were noted in three cases of ependymoma and astrocytoma. In cases of cord tumors, the contrast enhancement was present in all patients; it was larger in size, occupying the entire cross-sectional area of the cord on axial images except in one patient (in whom it still occupied more than half the cross-sectional area); and it showed a variable, heterogeneous pattern in all but two patients. Sharp superior and inferior enhancing margins on T1-weighted

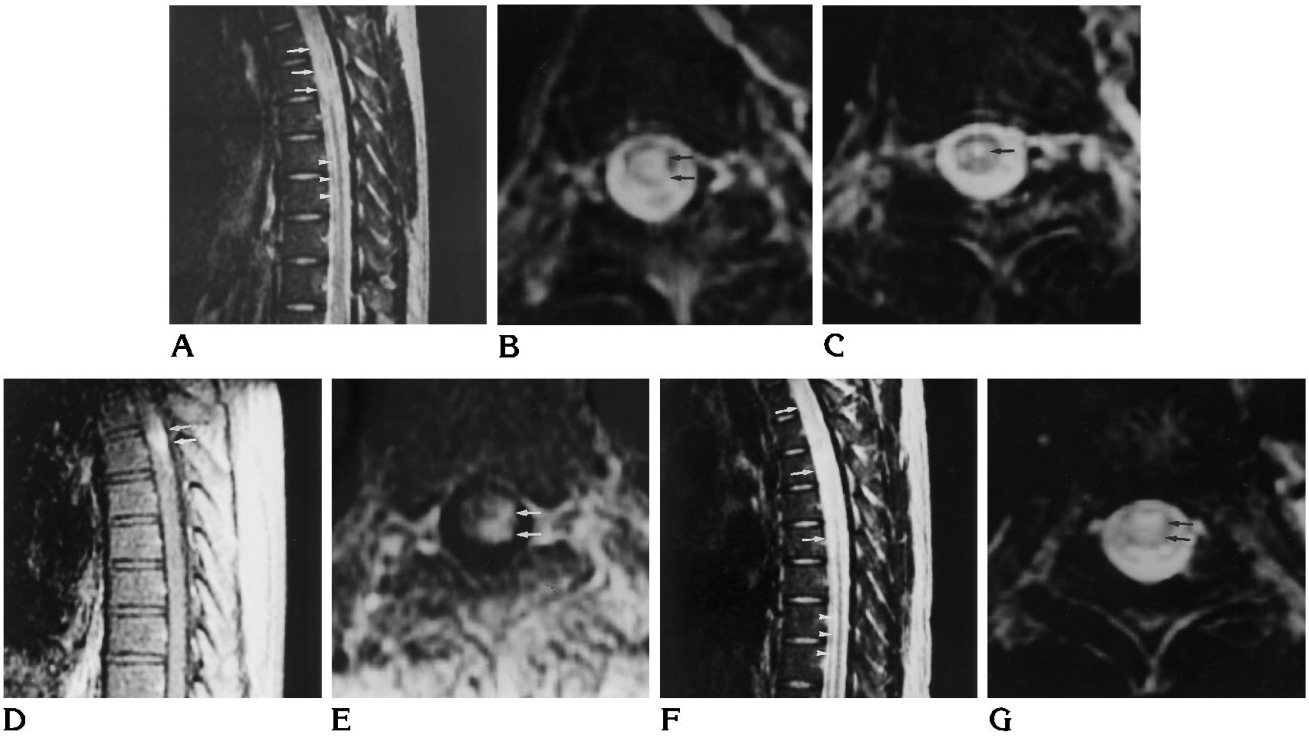

Fig 4 Case 16: 31-year-old man with a history of paraparesis and paresthesias of both lower extremities for 5 months.

A, Sagittal T2-weighted image of the thoracic spine shows expansile high signal intensity in an enlarged cord (*arrows*). In addition, the central hyperintensity extends inferiorly to the midthoracic level (*arrowheads*).

B, Axial T2-weighted image at the level of superiorly located expansile hyperintensity again shows a large central high signal (*arrows*).

C, An inferiorly located central hyperintensity is seen slightly anterior to the midpoint of the cord on axial T2-weighted image (*arrow*), suggesting that this hyperintensity is unlikely to be the dilated central canal.

Postcontrast sagittal (*D*) and axial (*E*) T1-weighted images show a contrast-enhancing area in the cord (*arrows*).

F, Two and a half months later, the expansile intramedullary hyperintensity (*arrows*) and inferiorly located central hyperintensity (*arrowheads*) appear more extensive and obvious on sagittal T2-weighted image.

G, Axial T2-weighted image again shows the central expansile hyperintensity (*arrows*). (*Figure continues.*)

sagittal images were seen in four cases of ependymoma and in one case of astrocytoma.

Discussion

Transverse myelitis, or myelopathy, is an uncommon syndrome characterized by bilateral motor, sensory, and autonomic dysfunction resulting from the involvement of both halves of the spinal cord in the absence of a preexisting neurologic or systemic disease (2, 4). The disease commonly begins with back or radicular pain followed by the abrupt onset of bilateral paresthesias of the legs, an ascending sensory level, and a paraparesis that often progresses to paraplegia (5). Middle-aged adults are commonly affected, and the thoracic spinal cord is the most frequent site of involvement. Although transverse myelitis has been recognized for several decades, it is a poorly understood syndrome pathologically and etiologically. There are many etiologic associations—such as with viral diseases, vaccinations, demyelinating processes such as multiple sclerosis, collagen-vascular diseases such as systemic lupus erythematosus, vascular disorders, and paraneoplastic syndromes—but most cases are idiopathic (3, 6). Polio virus, herpes zoster, and human immunodeficiency viruses are known to involve the spinal cord directly. Whenever possible, a precise etiologic diagnosis should be established on the basis of clinical, laboratory, and radiologic findings. None of our 17 patients recalled a history of upper respiratory infection in the 3 to 4 months preceding the onset of the neurologic deficit.

Classification, as described by Adams and Victor (7), into acute, subacute, or chronic varieties was not feasible with our patients because the time interval between the onset of

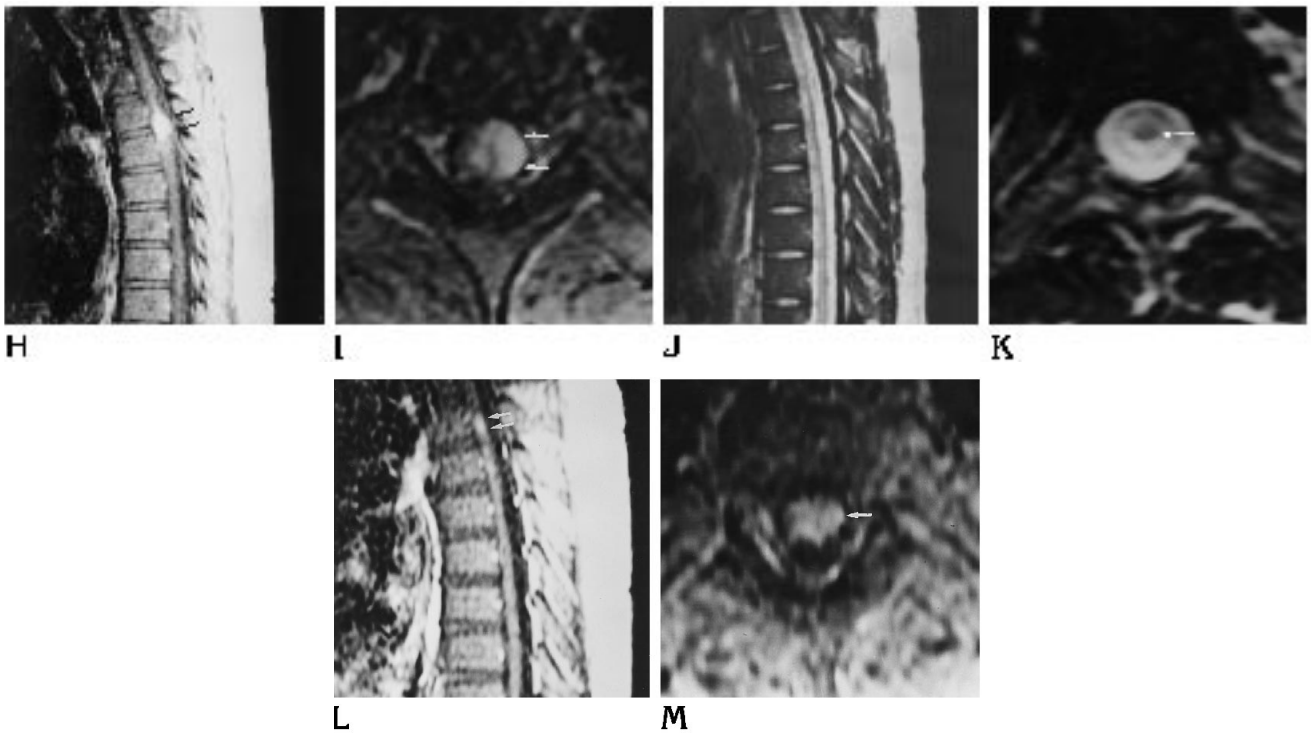

Fig 4, *continued.*

The enhancing nodule has also enlarged on postcontrast sagittal (*arrows, H*) and axial (*arrows, I*) T1-weighted images.

After treatment with corticosteroids for 2 months, the extent of the intramedullary hyperintensity has significantly resolved on sagittal T2-weighted image (*J*), and the central isointensity becomes more apparent on axial T2-weighted image (*arrow, K*).

Contrast-enhanced sagittal (*L*) and axial (*M*) T1-weighted images show markedly improved enhancement (*arrows*). Clinically, the patient improved steadily, in accordance with imaging findings.

neurologic deficit and the first visit to the hospital for medical attention varied greatly. The time at which the disease reached peak severity could not be recalled by every patient, and the time interval between symptom onset and initial MR examination ranged from 3 days to 4 months.

In the literature, MR findings of transverse myelitis have been described for a small number of patients (1, 2, 6, 8). The largest number (seven patients) was reported by Holtas et al (1). In their series, four patients had a focal enlargement of the cord, and all showed an increased signal on long-TR/TE sequences. In our cases, cord enlargement was better appreciated on sagittal than on axial T1-weighted images. Like others (1, 2), we found segmental high signal intensity (usually three to four vertebral segments) on T2-weighted images regardless of cord expansion. Barakos et al (2) reported that in four of their five patients, the signal abnormality extended over at least six spinal segments as opposed to the more common three to four segments in our patients. Patients with cord expansion had no more extensive segmental hyperintensity than did patients without cord expansion. We believe that a centrally located hyperintensity that occupies more than two thirds of the cross-sectional area of the cord on the long-TR/TE images found in most of our patients is characteristic of transverse myelitis.

The central isointensity, or dot (Figs 1B, 3B, and 4K), is believed to represent simply central gray matter squeezed by the uniform, evenly distributed edematous changes of the cord. It may not be a specific sign of transverse myelitis or have any clinical significance. Reviewing our cases of cord tumor, only two had a central isointensity, and in both it was located outside the contrast-enhancing area of the tumor. Because of its destructive nature and massive edema, we think it is less common in the tumors.

The ability to distinguish transverse myelitis from other intramedullary diseases, especially cord neoplasm, is of paramount importance, because it can mean the difference between

Seventeen patients with transverse myelitis

Case	Level of Sensory Deficit	Initial MR Imaging			Follow-up MR Imaging	
		Cord Expansion	Segmental Extent of T2 HSI	Contrast Enhancement	No. of Studies (months since initial study)	Significant Changes
1	T-4	−	T8-12	−	2 (3)	No contrast enhancement, improved T2 HSI
2	L-1	−	T7-11	−		
3	T-10	−	T9-11	−	1 (4)	No change
4	T-10	−	T8-11	+	1 (6)	Improved contrast enhancement and T2 HSI
5	T-7	+	T1-2	+		
6	C-6	+	C5-6	+		
7	None	+	T8-9	+		
8	None	−	T7-10	−		
9	T-5	+	T7-9	. . .	2 (13)	No contrast enhancement Resolved T2 HSI
10	T-5	+	T1-2	. . .	1 (6)	Area of contrast enhancement on follow-up No change in T2 HSI
11	T-9	−	T4-5	+		
12	T-8	−	T2-6	−	1 (7)	No contrast enhancement, improved T2 HSI
13	T-9	+	T2-6	+	1 (5.5)	No contrast enhancement, improved T2 HSI
14	T-9	−	T7-10	−		
15	T-11	+	T6-9	−	2 (2.5)	New contrast enhancement at 1-month follow-up Improved T2 HSI
16	T-9	+	T2-5	+	3 (4.5)	Progressed contrast enhancement at 2.5-month follow-up No contrast enhancement at 4.5-month follow-up Improved T2 HSI
17	None	−	T1-2	+	1 (2.5)	Improved contrast enhancement Improved T2 HSI

Note.—HSI indicates high signal intensity in the cord; +, present; and −, absent.

surgery, possible postsurgical complications, and radiation, or not. The usefulness of gadopentetate dimeglumine has been described in the detection of cord tumors as well as in the delineation of their location and extent in relation to surrounding edema. Pronounced contrast enhancement is usually noted with cord tumors (9–12). A few published reports have described patterns of contrast enhancement in patients with transverse myelitis: a moderate increase in signal intensity of the cord after injection of gadopentetate dimeglumine in one of five patients reported by Barakos et al (2); a diffuse abnormal contrast enhancement in the anterior aspect of the cervical cord that later became patchy in one patient described by Sanders et al (8); and an undefinable, fluffy pattern of enhancement at the midthoracic level in one patient reported by Pardatscher et al (13). Contrast enhancement in our eight cases was focally nodular or diffuse at the peripheral location with maintenance of the cord contour (Figs 2E, 3F, and 4E). The enhancing area was much smaller as compared with the extensive hyperintensity on T2-weighted images, and no case was associated with syringomyelia, a finding that may be associated with cord tumors.

Different patterns of enhancement in intramedullary tumors were found in our analysis. Common patterns included a large area of enhancement occupying the entire cross-sectional area of the cord on at least one T1-weighted axial image and variably heterogeneous enhancement frequently associated with a central or marginal cavity. Even with these findings, it is not always possible to differentiate between these two conditions. Closely spaced follow-up MR studies may aid in this endeavor. In two of our cases, interval progression of the enhancing lesions eventually improved 3 and 5 months, respectively, after the initial studies. For cases without contrast enhancement, as in seven of our patients, exclusion of a cord tumor would be equally difficult.

Multiple sclerosis is frequently mentioned as a potential underlying condition when transverse myelitis is first diagnosed. Both multiple sclerosis and transverse myelitis commonly involve young adults. Although the prevalence of multiple sclerosis is lower in Asians than in

whites, multiple sclerosis causing transverse myelitis seems to be uncommon. Altrocchi (14) found that only 4 of 67 patients with acute transverse myelopathy had multiple sclerosis. Lipton and Teasdall (15) found only 1 patient of 34 in whom the diagnosis of multiple sclerosis could be made after 5 to 42 years of observation. The frequency with which multiple sclerosis was found to follow acute transverse myelitis in cases reported in the literature ranged from 2% to 8% (mean, 5%) (4). In 4 cases of multiple sclerosis in a series reported by Aichner et al (16), plaques of the spinal cord appeared large, multiple, sharply demarcated, and sometimes confluent (kissing plaques). Although the number of patients they examined was small, the enhancing pattern of multiple sclerosis appears different from the transverse myelitis found in our patients.

The characteristic MR appearance of spinal multiple sclerosis in 68 patients (124 plaques) was documented in an article by Tartaglino et al (17). In their study, multiple sclerosis plaques were located peripherally, were less than 2 vertebral segments in length, and occupied less than half the cross-sectional area of the cord. In comparison, the T2 hyperintense lesions in our cases of transverse myelitis were centrally located, were 3 to 4 segments in length, and occupied more than two thirds of the cord's cross-sectional area. These significantly different findings will help distinguish the 2 conditions.

Although some articles have been written about the role of contrast media in multiple sclerosis of the spinal cord, an analysis of the contrast-enhancing pattern is lacking. In 4 cases of contrast-enhanced multiple sclerosis lesions in articles by Tartaglino et al (17) and Larsson et al (18) (2 cases each), the lesions showed enhancement in the central zone of peripherally located high signal intensity on T2-weighted images. A similar enhancing pattern was found in the brains of 12 patients described by Grossman et al (19). All had enhancement in the same location of high-signal-intensity lesions on T2-weighted images. In cases of transverse myelitis, on the other hand, enhancement is in the periphery of a centrally located area of high T2 signal intensity. Pathologically, cases of idiopathic acute transverse myelopathy may show a nonspecific necrosis that affects gray and white matter indiscriminately and destroys axons and cell bodies as well as myelin. The lesions may be focally transverse or they may diffusely involve a considerable length of the cord (14).

The role of corticosteroid treatment in transverse myelitis is uncertain; but because this disease may stem from an autoimmune mechanism like multiple sclerosis, many neurologists treat transverse myelitis with corticosteroids. Our patients were treated sporadically, mainly during the acute stage, so the treatment's effect on the outcome could not be assessed. Because all initial MR examinations were done before the start of steroid therapy, there could not have been any steroid effect on the degree of cord hyperintensity or on the pattern of contrast enhancement; but in the follow-up studies, steroids may have helped restore the blood–spinal cord barrier and reduce edema in view of the steady improvement or stabilization of imaging findings in all but two of our cases. In one of these patients, a new contrast-enhancing lesion was evident on the first follow-up study (case 15; Fig 3C and G) and in the other patient, an enlarged enhancing lesion was seen on the second follow-up examination (case 16; Fig 4D, E, H, and I). Both lesions were improved on subsequent follow-up studies (Figs 3K and 4L and M). Clinically, these two patients did not show gross worsening. In general, although there was some degree of relapsing-remitting neurologic manifestations during the follow-up period, slow improvement seemed to be the rule. No patient died during the follow-up period.

The clinical outcomes were variable in patients with transverse myelitis studied by us and others. Some of our patients have recovered well enough to lead normal lives while others have improved at a slower rate, with residual neurologic deficits. Follow-up data on 67 patients with acute transverse myelopathy reported by Altrocchi et al (14) showed that about one third had a good recovery, one third a fair recovery, and one third a poor recovery as judged by the criteria of Paine and Byers for classifying degrees of functional recovery (20) (55% of the patients were followed up for 2 years, 22 patients for 6 or more years, and 10 patients for more than 10 years).

In summary, the characteristic findings of transverse myelitis in our patients include normal size or segmental enlargement of the cord, most commonly thoracic; central hyperintensity occupying more than two thirds of the cross-sectional area of the cord on long-TR/TE sequences that usually affected three to four

vertebral levels; a central dot in the core of hyperintensity; not uncommon contrast enhancement; and, when present, a focal nodular or diffuse enhancing area at the periphery of the cord that did not change the cord contour. The prevalence of cord enhancement was significantly higher in patients with cord expansion than in those with normal cord size. These findings can be valuable in distinguishing transverse myelitis from multiple sclerosis, cord tumors, or other intramedullary lesions. Intramedullary hyperintensity and contrast enhancement did not correspond linearly to clinical improvement.

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