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Multiple Sclerosis of the Spinal Cord: Diagnosis and Follow-up with Contrast-Enhanced MR and Correlation with Clinical Activity

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PURPOSE: Although MR findings in multiple sclerosis (MS) are well known, the relationship between MR-detected lesions and clinical activity has not been studied in the spinal cord. The purpose of this study was to determine whether serial MR imaging provides evidence of disease activity unsuspected on clinical examination and to determine whether it is useful in monitoring patients with MS primarily affecting the spinal cord.

METHODS: Twenty-five consecutive patients with MS and with signs and symptoms of myelopathy underwent a full neurologic examination and contrast-enhanced MR imaging of the spinal cord at intervals of 0, 2, 6, and 12 months. Disability was rated according to Kurtzke's functional systems and the expanded disability status scale (EDSS). Clinical status of myelopathy (improved, deteriorated, or stable) was also assessed. Hyperintense lesions were counted on T2-weighted images and a weighted lesion load was calculated for each patient. The number of enhancing lesions was also determined.

RESULTS: We found a moderate correlation between lesion load and sensory function and EDSS. Seventy percent of patients with new clinical manifestations of myelopathy had one or more enhancing lesions. Agreement between MR findings and clinical examination in evincing disease activity was found in 60% of follow-up examinations. MR images showed lesion progression in seven (44%) of 16 occurrences of clinical deterioration and in 21 (35%) of 60 occurrences of clinical improvement or stability.

CONCLUSION: Serial MR imaging provides evidence of disease activity unsuspected on clinical examination and could be useful in monitoring patients with MS primarily affecting the spinal cord.

MR imaging is sensitive in detecting demyelinating lesions in the brain. In fact, white matter hyperintense lesions are detected on T2-weighted images in 90% to 97% of patients with clinically definite multiple sclerosis (MS) (1–3). Correlation between disability and MR-detected lesions in the brain is weak (4–9). Most intracranial lesions are asymptomatic, and new lesions appear on serial MR studies five to 10 times more often than evidenced by clinical exacerbations (4, 10–15). Contrast enhancement shows breakdown of the blood-brain barrier associated with perivascular inflammation and thus is a marker of histologic activity (16–18).

The spinal cord is frequently and often extensively involved in MS. In one autopsy series, Ikuta and Zimmerman (19) found cord lesions in 86% of 70 randomly selected patients with MS. In 13%, cord lesions were present without concomitant involvement of the brain. Early studies with MR imaging showed spinal cord lesions in 47% to 65% of patients with MS (20–22). Using multiarray coils and fast spin-echo pulse sequences, Kidd et al (5) identified lesions in 74% of their patients. Tartaglino et al (23) described the characteristic MR imaging features of spinal cord lesions in 68 patients with MS. The majority of plaques were two body segments in length or less, located at the periphery, and occupied less than

50% of the cross-sectional area of the cord. Although the spinal cord may be the earliest and, in some cases, the only site of involvement by MS (21, 24), few prospective studies with follow-up MR imaging have been carried out in patients with signs and symptoms of myelopathy.

The traditional measure of therapeutic outcome in patients with MS depends on relatively subjective and locomotion-weighted clinical rating scales. Serial MR imaging of the brain, when added to the clinical examination, allows more sensitive and objective monitoring of these patients (25–28). Serial MR imaging of the spinal cord could potentially play the same role in monitoring patients with myelopathy; but before recommending its use, we think it important to determine what additional information MR will provide concerning disease activity. The purpose of this study, therefore, was to compare disability and clinical activity with MR-detected spinal cord lesions in patients with MS and myelopathy.

Methods

Twenty-five consecutive patients with MS who had signs and symptoms of myelopathy on neurologic examination were included in this study. Selection of patients was based solely on the presence of myelopathy. The study group consisted of 15 women and 10 men, aged 20 to 56 years (mean, 36 years). Disease duration (measured from the time of the first clinical episode) ranged from 0 to 14 years (mean, 7 years). The study was reviewed and approved by our institutional review board and informed consent was obtained from each patient.

All patients had definite MS as supported by either the clinical (n = 23) or laboratory (n = 2) criteria of Poser et al (29). All had a sensory deficit and, in 18 patients, a sensory level was present. Nineteen patients had paraparesis or monoparesis and 18 had sphincter dysfunction.

At the onset of the study, a detailed medical history was obtained and a full neurologic examination was carried out. Disability was rated according to Kurtzke's functional systems and the expanded disability status scale (EDSS) (30). Pyramidal, sensory, and bowel/bladder functions were graded on a 0 to 6 scale. The ambulation index (from 0 to 9) and EDSS score (from 0 to 10) provided global assessment of disability. The clinical examination was repeated at 2, 6, and 12 months. At each visit, any new symptoms or signs were sought and the Kurtzke scores were reassessed. All clinical examinations were performed by one of two neurologists who were blinded to the MR results. Clinical deterioration of myelopathy was defined as the occurrence of one or more symptoms of neurologic dysfunction lasting more than 24 hours. Clinical improvement was defined as a definite improvement of symptoms, signs, or both lasting for at least 1 month (30).

The mean functional scores of patients on initial examination were as follows: pyramidal, 2.28/6; sensory, 2.68/6; bowel/bladder, 1.20/6, EDSS score, 4.34/10; and ambulation index, 3.28/9. All patients except one had known MS and were previously symptomatic from lesions in the brain, optic nerves, or spinal cord. In one patient, MS was diagnosed upon entry in the study. Seventeen patients had relapsing-remitting MS, seven had secondary-progressive MS, and one patient had primary-progressive disease. All but three patients with relapsing-remitting MS had one or more previous episodes of myelopathy before they entered the study.

Each patient underwent MR imaging of the brain and spinal cord within 10 days of the initial clinical evaluation. In addition, imaging of the cord was performed at 2, 6, and 12 months, and

imaging of the brain at 12 months. All imaging was done on a 1.5-T system. Brain MR was carried out using a standard quadrature head coil. Axial and sagittal proton density– and T2-weighted fast spin-echo images (5000/17–102/1 [TR/TE/excitations]) were obtained with the following parameters: a section thickness of 5-mm, an echo train length of 8, a matrix of 192×256, and a field of view (FOV) of 24 cm. Imaging of the cord was performed with a phased-array multicoil. Sagittal T1-weighted spin-echo images (500/20/2) were obtained through the cord using a 3-mm section thickness, a 256×512 matrix, and a 24×48-cm FOV. Then, sagittal proton density– and T2-weighted fast spin-echo images (2000/34–119/2) were obtained through the cervical and thoracic segments with an echo train length of 8, a matrix of 256×256, and an FOV of 26 cm. In the second part of the examination, sagittal T1-weighted spin-echo images (500/20/2) were obtained through the cervical and thoracic segments 5 to 12 minutes (early) and 15 to 22 minutes (delayed) after injection of 0.1 mmol/kg gadopentetate dimeglumine. For all images, a 3-mm section thickness, a 256×256 matrix, and a 26-cm FOV were used. Axial images were not obtained in this study. Total examination time was approximately 1 hour for imaging of the brain and cord.

At the end of the study, two neuroradiologists who were blinded to the clinical history reviewed all images and reached a consensus decision. Only the patient's identification number appeared on the imaging studies, and a standardized questionnaire was used for interpretation. Hyperintense lesions were counted in five periventricular and eight nonperiventricular white matter regions on proton density– and T2-weighted images of the brain obtained at 0 and 12 months. Lesions were counted if they were present on both proton density– and T2-weighted images. Lesions were judged according to their longest diameter (31). They were categorized as 5 mm, 6 to 10 mm, more than 10 mm, or confluent, and scored 1 to 4, respectively, depending on this measurement. Each lesion within a region was scored and a total score was calculated for that region. The total brain lesion load was obtained by summing the lesion size scores over all 13 regions. The five periventricular and eight nonperiventricular regions encompassed almost the entire brain except for the caudal part of the posterior fossa and the upper convexity. Lesions were also counted on proton density– and T2-weighted images of the spinal cord obtained at 0, 2, 6, and 12 months. Lesions were counted if they were present on both proton density– and T2-weighted images. Cord lesions were categorized as 5 mm, 6 to 10 mm, 11 to 30 mm, or more than 30 mm in diameter and scored 1 to 4, respectively, depending on this measurement. A weighted spinal cord lesion load was calculated by summing the scores. The number of enhancing lesions and the type of enhancement pattern (nodular, ring, or patchy) were determined on postcontrast sagittal T1-weighted images. Follow-up examinations were reviewed for presence of new or enlarging lesions on T2-weighted images and postcontrast T1-weighted images. Disappearance, decrease in size, or loss of enhancement was also noted on the same images.

Spearman rank correlation coefficients were calculated to assess the relationship between lesion load and functional scores at time 0. The relative frequency of cord enhancement in patients with and without recent clinical manifestations was compared using a χ^2-test. Correlation between evolution of MR-detected lesions and clinical status of myelopathy was analyzed with a χ^2-test. An analysis of variance for repeated measures was used to compare functional scores at time 0, 2, 6, and 12 months. Finally, Pearson correlation coefficients were calculated to evaluate the relationship between the change in brain lesion load and the change in cord lesion load in patients with evidence of lesion progression on follow-up MR examinations.

FIG 1. 44-year-old woman with recurrent episodes of myelopathy. Sagittal T2-weighted image of the thoracic cord shows multiple hyperintense lesions measuring 7, 10, and 4 mm in length and scored 2/4, 2/4, and 1/4, respectively. Other lesions were present in the cervical cord as well (not shown). Despite these lesions, disability was mild (EDSS score = 1.0).-

FIG 2. 21-year-old man with recent onset of myelopathy, sensory level at T-9, and EDSS score of 3.5.

A, Sagittal T2-weighted image of the thoracic cord shows solitary hyperintense lesion at T-9 with swelling. Lesion measured 13 mm in length and was scored 3/4. There was no enhancement after contrast administration.

B, Patient showed clinical improvement at 6 months, with EDSS score of 0. Sagittal T2-weighted image at this time shows a decrease in size and signal intensity of lesion and disappearance of swelling.

Results

Baseline Hyperintense Lesions of the Spinal Cord and Brain on T2-Weighted MR Images

Twenty-four (96%) of 25 patients had hyperintense lesions in the spinal cord on proton density– and T2-weighted images. A total of 108 plaques were identified (mean, 4 per patient; range, 0 to 8). Twenty-two patients (88%) had more than one plaque (Fig 1). The weighted lesion load in the spinal cord ranged from 0 to 19, with a median of 10. The total spinal cord lesion load in the sample of 25 patients was 235 at time 0.

Twenty-four (96%) of 25 patients also had hyperintense lesions in the brain on proton density– and T2-weighted images. Lesions in the brain and spinal cord were found in 23 patients, one patient had lesions in the brain only, and another had lesions in the spinal cord only. The weighted lesion load in the brain ranged from 0 to 123, with a median of 49. The total brain lesion load in the sample was 1228 at time 0. The total lesion load in the CNS (spinal cord plus brain) was 1463, with 81% of the lesion load located in the brain and 19% in the spinal cord.

We found a moderate correlation between spinal cord lesion load and sensory function ($r = .553$; $P < .001$) and EDSS score ($r = .422$; $P < .05$). Eight (44%) of 18 patients with a defined sensory level deficit had a hyperintense lesion in the cord that corresponded anatomically with the level (Fig 2). There was no significant correlation among the following: 1) spinal cord lesion load and ambulation index and pyramidal or bowel/bladder function scores; 2) brain or total CNS lesion load and EDSS score, ambulation index, and pyramidal, sensory, or bowel/bladder function scores; or 3) spinal cord and brain lesion loads ($P > .05$).

Baseline Enhancing Lesions of the Spinal Cord

Thirteen (52%) of 25 patients had enhancing lesions in the cord after contrast administration (Fig 3). A total of 15 enhancing plaques were present, representing 14% of the hyperintense lesions. The mean length of the enhancing lesions was 7 mm, with a range of 2 to 12 mm. All lesions were present on delayed images (15 to 22 minutes after contrast administration) but only 13 were identified on early

FIG 3.   A 20-year-old man with new clinical manifestations of myelopathy, sensory level at T-12, and EDSS score of 2.0.
A, Sagittal T2-weighted image of the cervical cord shows multiple hyperintense lesions. Thoracic cord was normal.
B, Contrast-enhanced T1-weighted image shows nodular enhancement at C-4.
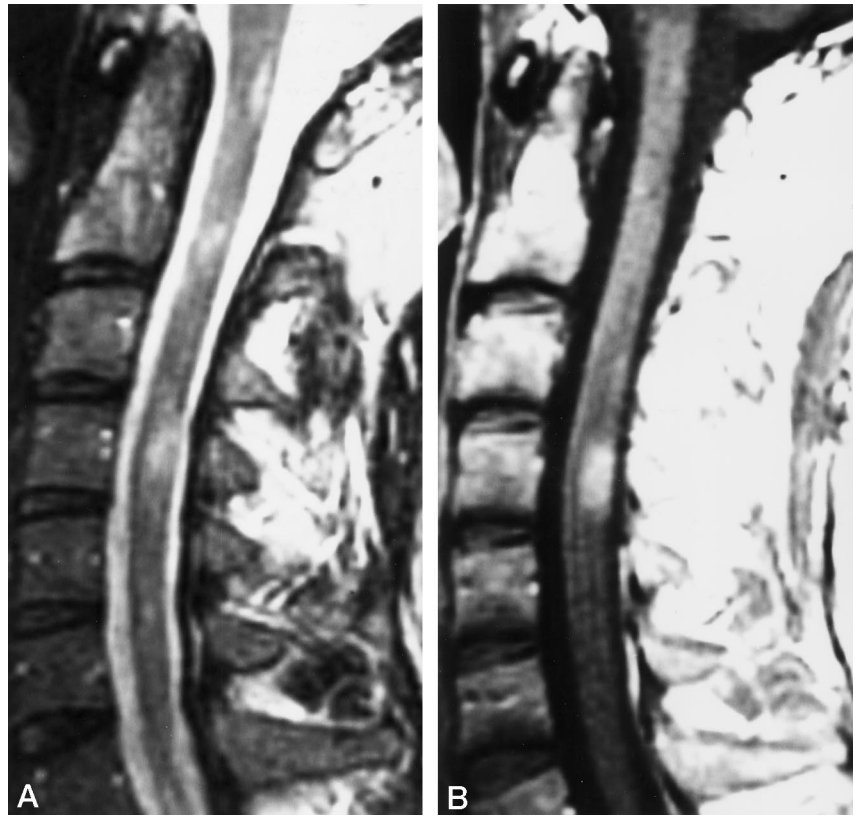
images (5 to 12 minutes after contrast administration). In addition, four lesions were more conspicuous on delayed images than on early images. Three patterns of enhancement were present: nodular (n = 10), patchy (n = 3), and ring (n = 2). Two lesions evolved from a ring pattern on early images to a nodular pattern on delayed images. All lesions were hyperintense on proton density– and T2-weighted images. Six enhancing lesions (40%) were associated with swelling of the cord.

Seventeen of 25 patients had new clinical manifestations of myelopathy within 2 months of entry in the study and were included in the new clinical manifestations (NCM) group. The remaining eight patients were clinically stable during the 2 months prior to entry in the study and thus were included in the no new clinical manifestations (NNCM) group. The NCM group included 10 women and seven men, aged 20 to 50 years (mean, 34 years), with a mean disease duration of 7 years. The NNCM group included five women and three men, aged 24 to 56 years (mean, 40 years), with a mean disease duration of 6 years. The NCM group included significantly more patients with enhancing lesions than did the NNCM group ($P <$ .01). Twelve (71%) of 17 patients presenting with new clinical manifestations had at least one enhancing lesion in the spinal cord on initial MR imaging, while only one (13%) of eight NNCM patients had enhancement. The single NNCM patient with enhancement had clinical deterioration 10 weeks before entering the study, which barely excluded her from the NCM group.

Follow-up Examinations of the Spinal Cord and Brain

Each patient had follow-up clinical examinations and MR imaging at 2, 6, and 12 months after entry in the study. One patient with a clinical relapse had an additional examination 2 weeks after the 6-month examination; thus, a total of 76 follow-up examinations were performed in 25 patients. Myelopathy showed clinical deterioration on 16 follow-up examinations, improvement on 20 examinations, and stability on 40 examinations. By comparison, lesion progression (new, recurrent, or enlarging hyperintense lesions and new enhancing lesions) was seen on 13 follow-up MR examinations (Fig 4); lesion diminution (disappearance or decrease in size of hyperintense lesions or loss of enhancement) was evident on 28 examinations (Figs 2 and 5); both lesion progression and lesion diminution were seen on 15 examinations; and no change was noted on 20 examinations. A total of 47 new, recurrent, or enlarging hyperintense lesions were present on T2-weighted images. Enhancement was detected in nine plaques: six in pre-existing T2-weighted hyperintense lesions and three in new T2-weighted lesions.

Table 1 shows the relationship between MR imaging findings and clinical status of myelopathy in all 76 follow-up examinations. In this table, MR examinations with lesion progression include those with lesion progression only as well as those with both lesion progression and lesion diminution. Lesion progression was identified on 28 follow-up MR examina-

FIG 4. 26-year-old man with chronic myelopathy and EDSS score of 3.0.

A, Sagittal proton density–weighted image shows a hyperintense lesion from C-2 to C-5 measuring 48 mm in length, which was scored 4/4. A second lesion is present at C-1. There was no enhancement after contrast administration.

B, Clinical deterioration was present at 6 months with an increase in EDSS score to 5.0. Sagittal T1-weighted image shows enhancement at C-2. Enhancement disappeared at 12 months without change in clinical status.

tions and was associated with concurrent clinical deterioration in seven cases and with subsequent deterioration in another five cases. Lesion diminution or stability was identified on 48 follow-up MR examinations and was associated with concurrent clinical deterioration in nine cases. MR findings showed lesion progression on seven (44%) of 16 examinations with clinical deterioration and in 21 (35%) of 60 examinations with clinical improvement or stability. Agreement between MR and clinical examinations was found in only 46 (61%) of 76 follow-up examinations. Statistical analysis showed no apparent relationship between change in MR-detected lesions and clinical activity of the disease over the 1 year follow-up period.

Of 15 enhancing plaques present on the first examination, 11 (73%) had lost enhancement at 2 months (Fig 5). An additional two lesions lost enhancement at 6 months, and a third lesion lost enhancement at 12 months. Only one lesion still enhanced at 12 months. Loss of enhancement was accompanied by concurrent loss of swelling in all patients. Lesions persisted longer on T2-weighted images. Nine (60%) of 15 enhancing cord lesions identified on the initial examination were still hyperintense at 12 months, although six were less intense. Four enhancing lesions disappeared on both T1- and T2-weighted images at 2 months, one lesion disappeared at 6 months, and another disappeared at 12 months. Loss of enhancement was accompanied by clinical improvement in nine (69%) of 13 patients in whom enhancement was

seen on the first examination (Fig 5). An additional two patients were clinically stable despite loss of enhancement. Of the remaining two patients, one had clinical deterioration despite loss of enhancement, while the other improved despite persistent enhancement of one lesion. Table 2 shows the relationship between the percentage of patients with at least one enhancing lesion and mean functional scores, ambulation index, and EDSS score at time 0, 2, 6, and 12 months in all 25 patients. The percentage of enhancement decreased from 52% at time 0 to 16% at 2 months. Loss of enhancement was accompanied by improvement in sensory function, pyramidal function, bowel/bladder function, ambulation index, and EDSS during the same time interval.

Nine new enhancing lesions were identified in six patients on follow-up examinations (Figs 4 and 5). Three patients each had two new enhancing lesions while three others had a single enhancing lesion. All new enhancing lesions were hyperintense on T2-weighted images. Six (67%) of nine new enhancing lesions were either new or had enlarged on T2-weighted images. Six enhancing lesions were associated with clinical activity while three lesions appeared in asymptomatic patients.

The total brain lesion load was 1228 at time 0 and 1255 at 12 months. Ten (40%) of 25 patients had an increase in brain lesion load and the increase was greater than 10% in six patients. Nineteen patients had lesion progression in the spinal cord on at least one follow-up MR examination. Table 3 shows the

FIG 5.   22-year-old woman with new clinical manifestations of myelopathy and EDSS score of 3.0.

A, Sagittal T2-weighted image of the cervical cord shows hyperintense lesion at C-5.

B, Contrast-enhanced T1-weighted image shows ring enhancement of the lesion.

C, Patient improved, and EDSS score was 1.5 at 2 months. Sagittal T2-weighted image at this time shows decreased signal of C-5 lesion. There was no more enhancement after contrast administration.

D, At 12 months, new symptoms developed, accompanied by an increase in EDSS score to 2.5. Contrast-enhanced sagittal T1-weighted image shows a new enhancing lesion at C-2.
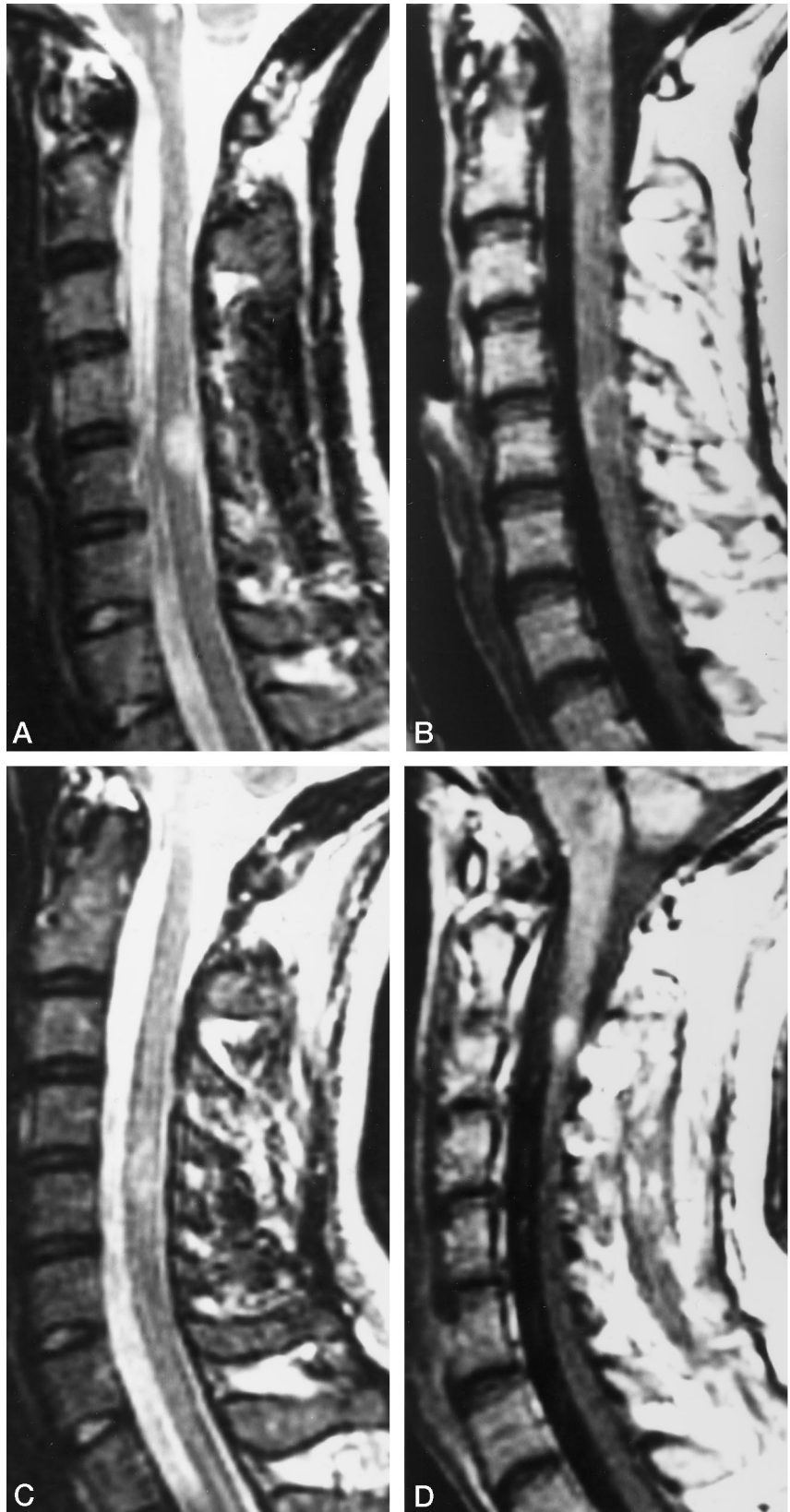
TABLE 1: Relationship between MR findings and clinical status of myelopathy on 76 follow-up examinations

MR findings	Clinical Status of Myelopathy, no. of examinations		
	Deteriorated	Improved or Stable	**Total**
Lesion progression	7	21	**28**
Lesion diminution and/or stability	9	39	**48**
Total	16	60	76

TABLE 2: Relationship between percentage of enhancement and mean functional scores at time 0, 2, 6, and 12 months in 25 patients

	Time, mo			
	0	2	6	12
Percentage of enhancement	52%	16%	24%	12%
Sensory function	2.76	1.53	1.47	1.81
Pyramidal function	2.06	1.70	1.47	1.37
Bowel/bladder function	1.12	0.94	0.88	0.87
Ambulation index	2.82	2.06	2.06	1.87
EDSS score	4.06	2.94	2.88	2.75

Note.—The percentage enhancement is the number of patients with at least one enhancing lesion divided by the total number of patients times 100; there was a significant improvement in sensory function ($P = .002$), ambulation ($P = .029$), and expanded disability status scale (EDSS) score ($P = .002$) between time 0 and 12 months.

TABLE 3: Relationship between lesion load in the brain and cord with time in 19 patients who had lesion progression on follow-up MR examinations

	Time, mo	
	0	12
Mean brain lesion load	54	56
Mean cord lesion load	11	11

relationship between the change in brain lesion load and the change in spinal cord lesion load with time in these patients. There was no significant correlation between the change in brain lesion load and the change in spinal cord lesion load ($r = .217$; $P = .372$). Likewise, there was no significant correlation between the percentage of change in brain lesion load and the percentage of change in cord lesion load ($r = .332$; $P = .165$).

Eleven patients received steroids during this study. One patient was treated with β-interferon. Of five patients who received steroids within 1 month of the first MR examination, only one had enhancement in the spinal cord.

Discussion

We found a moderate correlation between spinal cord lesion load and both sensory function and EDSS scores. There was no significant correlation between lesion load and other functional system scores and the ambulation index. Several factors bear on these re-

sults. First, only lesion length was measured and used for lesion load determination. MR-derived volume measurements provide greater sensitivity and reproducibility (32). They have been used in the brain but not in the spinal cord yet. Second, although significant advances have been made in MR imaging of the spinal cord, image quality is not always optimal and small lesions may be missed. Third, disability is measured by functional scores, which in turn reflect lesions in the brain and spinal cord. We found no significant correlation between total CNS lesion load and disability. Fourth, there are two major types of chronic demyelinating lesions: those with extensive axonal loss and those with extensive gliosis and preserved axons (33). Axonal loss, which causes more disability, cannot be distinguished from extensive gliosis and preserved axons on proton density– and T2-weighted images. The ratio of one type of lesion relative to the other in a given patient presumably has an influence on the degree of disability. Proton spectroscopy and magnetization transfer imaging provide better tissue characterization, and improved correlation between MR-detected lesions and disability has been reported in the brain with the use of these two techniques (34, 35).

Contrast enhancement correlates with the presence of an active inflammatory process with perivascular lymphocytic infiltration (16, 17). Although only a small percentage (14%) of spinal cord plaques enhanced in our patients, the total number of plaques with blood-brain barrier disturbance was probably larger. It is likely that some nonenhancing lesions have subtle blood-brain barrier disturbance not detected with contrast administration. A majority of patients (70%) with new clinical manifestations of myelopathy had enhancing lesions in the cord. Most enhancing lesions were homogeneous, nodular, and relatively small. Patchy and ring enhancement were present in some plaques. Some lesions increased in size, became homogeneous, and were best seen 15 to 22 minutes after contrast administration. Similar findings have been described in the brain (11, 36, 37). Ringlike lesions probably reflect acute inflammation at the edge of chronic demyelinating lesions. The change in the pattern of enhancement from early to delayed scans is explained by a lower blood-brain barrier permeability in the center as compared with the periphery, resulting in a slower rise in concentration of contrast centrally. Demonstration of disease activity through contrast enhancement helps satisfy the criteria of dissemination in time for the diagnosis of MS.

Our study shows that the natural history of MS primarily affecting the spinal cord is characterized by fluctuation in disease activity with time. Myelopathy was clinically improved or had deteriorated in 47% of follow-up visits and there was evidence of lesion progression, lesion diminution, or both in 74% of follow-low-up MR examinations. A number of patients even had lesion progression and lesion diminution on the same examination. Previous studies in the brain have shown similar lesional fluctuation with time, presum-

ably reflecting a natural disease process (6, 12, 38). According to McFarland et al (38), new lesions do not develop at a constant rate but tend to occur in bursts of increased frequency, with a cyclical trend. The fluctuation in disease activity observed in our series was probably accentuated by the relatively large number of patients with relapsing-remitting disease.

Agreement between MR and clinical evidence of disease activity was found in only 60% of follow-up examinations. Lesion progression was often present on MR images despite an absence of clinical deterioration. One explanation is that many patients have sequelae from previous myelopathic episodes and, in these patients, new signs and symptoms of myelopathy may not be clinically apparent. We believe that lesion progression on MR images is most likely a significant development in patients with MS, even in the absence of concurrent clinical deterioration. With the current level of technology, it is not possible to determine if the process is reversible or irreversible. Our study also showed that clinical deterioration frequently occurs despite an absence of lesion progression on MR images. This finding is not surprising in view of the poor correlation between MR appearance and some functional systems. Discrepancy between MR and clinical findings has been reported by other investigators, particularly in the brain (4, 10, 12, 15, 33, 39). Serial MR imaging and clinical examinations are probably needed for adequate monitoring of MS disease activity in the spinal cord.

MR lesion progression was detected mainly by identifying new or enlarging hyperintense lesions on T2-weighted images, and the majority of these lesions were nonenhancing. In the brain, by comparison, most active lesions enhance (11, 14, 25, 33, 36). Since follow-up visits took place only at 2, 6, and 12 months, it is possible that enhancement was present at one point and had disappeared by the time MR imaging was performed. In addition, blood-brain barrier disturbance may be relatively subtle, requiring a double dose of contrast material for demonstration. When present, enhancement of spinal cord lesions was a marker of disease activity. It usually lasted less than 2 months, and loss of enhancement was accompanied by clinical improvement. Knowledge that enhancement is transitory is useful in clinical practice when the nature of an enhancing spinal cord lesion is in doubt. If the lesion is a consequence of MS, a follow-up examination is expected to show disappearance or decrease in enhancement with time. Besides T2-weighted hyperintense lesions and contrast enhancement, cord atrophy is another potential marker of disease activity. Kidd et al (40) found that the cross-sectional area of the spinal cord at C-5 decreased over the course of 1 year in 19 patients with progressive MS.

A large percentage of patients with spinal cord lesions also have lesions in the brain (5, 22, 23, 41, 42). Furthermore, in our sample, 81% of the total CNS lesion load was in the brain, compared with only 19% in the spinal cord. Thus, in patients with predominating signs and symptoms of myelopathy, if brain lesion count alone is used, approximately 19% of disease activity will be overlooked.

Conclusion

There is a moderate correlation between spinal cord lesion load and disability (EDSS score and sensory function) in patients with MS and myelopathy. Hyperintense lesions on T2-weighted images and contrast enhancement on T1-weighted images are markers of disease activity. There is a discrepancy between evolution of lesions on MR studies and clinical status of myelopathy on follow-up examinations. MR lesion progression occurs despite absence of clinical deterioration and vice versa. Both MR imaging and clinical examinations are needed to monitor disease activity in patients with MS primarily affecting the spinal cord.

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