The Role of Magnetic Resonance Imaging in Acute Transverse Myelitis

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ABSTRACT: Eighteen adult patients presenting with acute transverse myelitis (ATM) were evaluated using magnetic resonance imaging. Only 7 had abnormal scans showing an area of increased signal intensity within the cord solely on T2 weighted images; T1 weighted images were normal. The MRI abnormality did not correlate with the cause of the transverse myelitis, the extent of maximum neurological deficit, or the prognosis. A scan performed more than 5 days after the onset of disease was most likely to be positive. Even though the prognostic value of MRI in ATM may be limited, it remains a valuable technique for ruling out other causes of noncompressive spinal cord lesions, such as hemmorhage, vascular malformation, or tumor.

RÉSUMÉ: Le rôle de l'imagerie par résonance magnétique dans la myélite transverse aiguë. Nous avons évalué, au moyen de l'imagerie par résonance magnétique (NMR), dix-huit patients adultes qui se sont présentés avec une myélite transverse aignë. Seulement 7 avaient un scan anormal montrant une zone où l'intensité du signal était augmentée dans le moelle épinière, seulement sur les images pondérées par T2; les images pondérées par T1 étaient normales. L'anomalie à la NMR n'était pas corrélée à la cause de la myélite transverse, à l'étendue maximale du déficit neurologique ou au pronostic. Un scan effectué plus de 5 jours après le début de la maladie était plus susceptible d'être positif. Bien que la valeur prédictive de la NMR dans la myélite transverse aiguë puisse être limitée, cette technique demeure valable pour éliminer d'autres causes de lésions non-compressives de la moelle épinière, telles une hémorragie, une malformation vasculaire ou une tumeur.

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Acute transverse myelitis (ATM) has been defined as an "acute intramedullary dysfunction of the spinal cord, ascending or static involving both halves of the cord and appearing without any history of previous neurological disease."[1] Multiple etiologies have been postulated, including post infectious,[2] infectious (herpes zoster, herpes simplex virus, cytomegalovirus, echo-2 virus, syphilis, mycoplasma pneumoniae and Epstein-Barr virus),[3-9] vascular insufficiency,[10] paraneoplastic syndromes,[11] heroin use,[12] post-vaccinial,[13] irradiation, and collagen-vascular disease.[14,15] Since the advent of MRI, several previous causes can now be ruled out, including vascular malformations, hemorrhage, and usually tumor. Of the more than 15 reported cases of ATM with MRI findings we reviewed, all scans were abnormal.[16-24]. Lesions consisted of either cord swelling without abnormal signal,[16] or more commonly, diffuse increased signal on T2 weighted images within the center of the cord.[17] One specific case with gadolinium enhancement was also reported.[22]

Many questions remain about the meaning of MRI findings in patients presenting with ATM. It is not clear what proportion of patients having ATM will have an abnormal MRI. If it is abnormal, can it be used to predict the likelihood of continued neurological deterioration or the prognosis for recovery? Also, does an abnormal scan indicate a higher risk of later developing a disseminated disease such as multiple sclerosis?

We reviewed the records of 18 patients presenting with ATM who had spinal cord MRI's performed at our institution over the past 4 years. The results of the MRI are correlated with clinical findings, including initial and residual disability. The presumed cause of the ATM, the presence or absence of CSF pleocytosis and the level of cord affected are also analyzed.

Materials and Methods

Between Feb. 1986 and Dec. 1990, we identified 18 patients at the Los Angeles County – University of Southern California Medical Center who had acute transverse myelitis evaluated with MRI. Histories and physical findings were obtained by a retrospective review of medical records and imaging studies were reviewed with a neuroradiologist. An outline of cases along with MRI results are summarized in Table 1.

All patients showed evidence of an acute, intramedullary spinal cord process with onset over less than 4 weeks. Patients were previously healthy without prior evidence of neurological disease. Patients were not excluded if they subsequently showed

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Table 1. Summary of the 18 Patients with Transverse Myelitis

Pt. No.	Age/Sex (Years)	Cause	Initial Grade	Final Grade	Time of Follow-Up (Months)	CSF WBC's	Cord Level	Timing of MRI in Relation to Onset	MRI Results, T2 Weighted Images of the Spinal Cord
1.	36M	unknown	2	0	3mo	0	T5	3 days	negative
2.	38F	MCTD***	2	2	24mo	—	T8	7 days	negative
3.	40M	unknown	1	0	2mo	28	none	10 days	negative
4.	25M	unknown	1	1	1mo	0	T1	5 days	negative
5.	23M	syphilis	3	—	—	66	T2	4 days	negative
6.	51F	unknown	2	1	2mo	24	L1	10 days	negative
7.	15M	unknown	2	1	1mo	0	T7	5 days	negative
8.	39M	Neuromyelitis optica	3	3	1mo	72	C5	6 weeks	negative
9.	51F	Sjogrens	2	2	6mo	6	T5	24 days	negative
10.	59F	infarction	3	2	4mo	0	T10	3 mos	negative
11.	17M	EBV	3	—	—	38	T4	—	negative
12.	26M	Mumps	3	0	4mo	359	T4	6 days	mildly increased signal at conus, poorly defined
								30 days	enhancement at T6 only, conus normal
13.	30M	unknown	2	2	2mo	0	T10	10 days	mildly increased signal at T12, poorly defined
14.	20M	unknown	2	1	8mo	0	C4	7 days	increased signal at C2-3, see figure
15.	27F	post-infectious	2	2	12mo	5	T9	4 days	faintly increased signal from T7-T10, poorly defined
16.	37F	ADEM**	3	3	12mo	70	C4	4 days	negative cord
								2 mos	high density lesion at C4-C6 with swelling of the cord
17.	23F	possible MS	2	1	12mo	172	T5	14 days	high density lesion at C2-4 with mild cord swelling, no enhancement
18.	23F	unknown	2	1	9mo	0	T10	8 days	moderate increased intensity lesion at T8-T9, no enhancement

* Patients are not listed in chronological order.
** Acute disseminated encephalomyelitis.
*** Mixed connective tissue disease.

other central nervous system signs as long as their initial problems were localized to the spinal cord. Those persons positive for HIV or less than 15 years old were excluded. All patients except one (#3) had a distinct sensory level and bladder or bowel involvement. No patient had evidence of cord compression on the imaging study. MRI was done as soon as possible after the onset of clinical symptoms.

MRI scans were obtained on either the GE Sigma 1.5T unit or the Philips O.5T and 1.5T Gyroscan. Routine protocol included 5mm thick sagittal scans using spin echo pulse sequences with T1 weighted and T2 weighted images. Axial T2 weighted images were also obtained in patients #12-18.

To our knowledge, there is not a standard grading scale for the extent of neurological deficit in patients with ATM. Ropper[1] divided his patients into those who had good, fair, and poor outcomes, but did not grade the initial deficit. For our study, the patients were segregated into three groups based on the extent of neurological impairment at their worst and again at the latest follow-up. Grade 1 was defined as those patients still able to ambulate with or without assistance. Grade 2 indicated patients who were unable to walk, but retained some voluntary muscle movement, tone or reflexes. Patients graded as 3 had flaccid paraparesis with loss of reflexes. We chose this method of grading as it represented information readily available through reviewing the medical record. Also, Lipton, et al.[25] felt that patients who presented with the symptoms of spinal shock fared worse. Therefore, we felt they should be in a different category than those patients with less profound weakness. Patients who made complete recovery were graded as 0 at follow-up. Follow-up ranged from 1 month to 2 years with a mean of 4 months.

Results

Patients ranged in age from 15 to 59 years, with 10 males and 8 females. The average age of those patients with a normal scan was 36 years, compared to an average age of 26 in patients with a positive scan. Time from onset to maximum deficit was from less than 1 hour to 17 days. Sensory levels were found in the cervical region in three patients, the thoracic region in 13 patients, and the lumbar area in one patient. Patient #3, as stated above, did not have a distinct sensory level but had acute onset of increased tone of the lower extremities with pathologically brisk deep tendon reflexes and bladder incontinence.

The causes of the transverse myelitis, when known, are listed in Table 1. Three patients went on to develop multifocal neurological disease (#8, #16, and #17), 2 of which had abnormal

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scans. Patient #8 developed optic neuritis soon after he presented with transverse myelitis. His cranial MRI showed several small bright lesions on T2 images in the periventricular areas, although his spinal MRI was negative. Patient #17 developed optic neuritis one year after the onset of ATM. She also complained of a brief episode of diplopia within that year for which she did not see a physician. Patient #16 went on to develop multifocal CNS signs felt to be secondary to acute disseminated encephalomyelitis. Both of the last two patients had areas of markedly increased signal involving the cord on T2 weighted images.

Both cranial and spinal MRI studies were done in 9 patients. Three were abnormal, showing small high intensity lesions of the white matter on T2 images. Patient #8 had neuromyelitis optica and patient #9 was found to have Sjogrens syndrome. Patient #16 had a normal initial cranial and spinal MRI, but a repeat scan at 5 months showed white matter disease of the brain and spinal cord. Six other patients had cranial MRI imaging that was normal, 2 of them had abnormal spinal studies.

Seventeen records reported the time of onset of initial neurological dysfunction. Five patients had MRI done within 5 days of onset. Of these, only 1 had an abnormal MRI (20%). Eleven patients had MRI after 5 days and of these, 6 were positive (45%). Patient #16 initially had a negative MRI, but a follow-up study 2 months later was abnormal.

More than one-half of the patients had a normal scan (11/18). MRI abnormalities were seen only on long repetition time (TR) images. The scan most often showed an ill-defined area of increased signal in the center of the cord spanning one to three spinal segments (Figure 1). There was a gradient of signal intensity of the lesions, some were very faint and others were much more prominent. Patient #14 was atypical. The MRI showed a lesion on T2 weighted images which was relatively well demarcated from surrounding, normal appearing cord, and with higher signal intensity (Figure 2). Gadolinium was given to three patients and resulted in slight enhancement in one (#12).

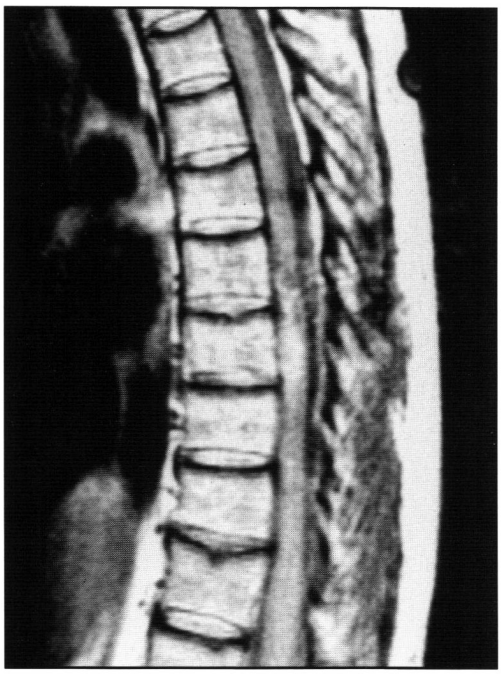

Figure 1 — Sagittal MRI of patient #18 (TR 1800 msec, TE 30 msec) showing an ill-defined area of increased signal at T8-T9 without change in cord diameter.

Five of 13 (38%) patients with thoracic sensory levels and 2 of 3 (66%) patients with cervical levels had abnormal scans. The sensory level correlated with the level of the lesion seen on MRI in 5 of 7 cases. In patient #12, with a clinical sensory level at T4, the initial MRI done at 6 days showed only an area of increased signal involving the conus. However, a second scan done after 30 days showed a thin, longitudinal area at T6 on the post Gd-enhanced image with normal T2 weighted images.

Nine of 11 patients (81%) with a negative MRI, and all 7 patients with a positive MRI were graded as 2 or 3 at their worst. However, by the most recent follow-up, 5 of 11 patients (45%) with a normal MRI, and 4 of the 7 patients (57%) with an abnormal MRI had improved and were graded as 1 or 0.

Seventeen patients had a CSF examination, 10 of which showed a pleocytosis. There was no significant difference (6/10 versus 4/7) between percentages of patients with a positive CSF who had a normal versus abnormal MRI. However, the 2 patients with a WBC count greater than 150/cmm had positive scans. All patients had CSF analysis for oligoclonal bands, and all were negative. During the time of this study, however, high resolution electrophoresis and immunofixation was not performed.

Discussion

Barakos[17] published 5 cases of ATM in which long TR sequences demonstrated an abnormal increase in signal intensity of the cord. Two of 5 patients also had cord widening in the involved area. Merine[16] reported 2 patients with ATM in which the MRI showed cord widening but normal T2 intensities. The

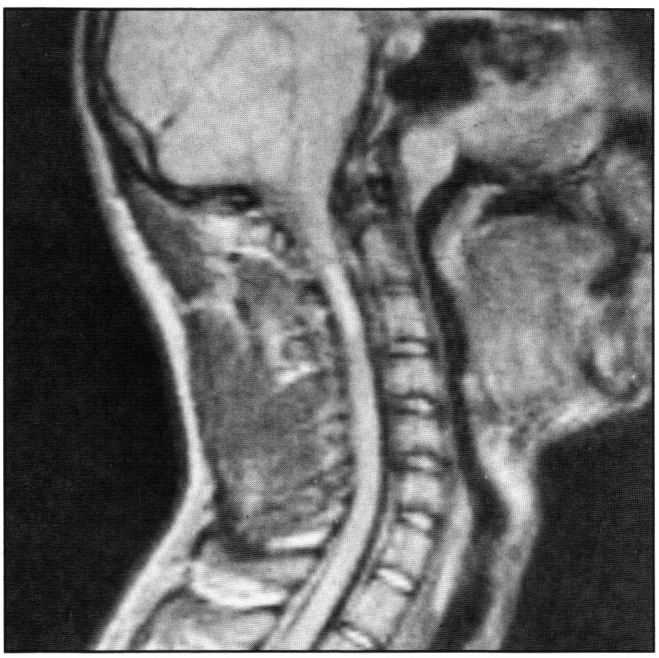

Figure 2 — Patient #14, sagittal proton density image (TR 2000 msec, TE 40 msec) shows a relatively well-defined lesion at C2-C3.

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lack of abnormal signal intensity was cited as an argument for the diagnosis of ATM rather than intramedullary neoplasm. Recently a case of a patient with a Brown-Sequard syndrome and an abnormal MRI was reported.[22] The initial MRI showed an area of cord enlargement on T1 weighted images with diffuse abnormal increase in signal intensity on T2 weighted images over 4 spinal segments. Follow-up scan at 1 month revealed a normal spinal cord and the patient had clinically improved. The author felt that an enhanced MRI may serve to predict residual dysfunction. There have been numerous other case reports, usually describing an area of mildly increased signal within the cord similar to our findings.[17-24]

We were unable to determine a cause for the transverse myelitis in one-half of the patients. An etiology was not more likely to be apparent in patients with an abnormal scan. Of the patients with a known etiology, 3 had demyelinating disease. The others were diagnosed with Sjogren's disease, Epstein-Barr virus, mumps, mixed connective tissue disease, and syphilis.

In our patients, most positive scans were obtained 5 or more days after the onset of the neurological deficit. In Sander's case report, he noted that the appearance of the lesion in the spinal cord changed over time.[22] Since we used MRI mainly to rule out other, possibly treatable causes of acute spinal cord dysfunction, we did not obtain serial scans. Perhaps, as Sanders suggested, the appearance of the lesion on serial scans may help to predict outcome.

In keeping with earlier studies,[26] those patients with cervical cord sensory levels were more likely to have an abnormal scan. The only correlation with the presence or absence of CSF pleocytosis was that both patients with more than 150 cells/cmm had areas of increased signal on their MRI. Perhaps this signifies a more profound inflammatory response with increased intramedullary edema.

We were unable to correlate the appearance of the MRI with the likelihood of progression to multifocal neurological deficits. Because our follow-up was brief, further studies will be required to evaluate this.

MRI remains a valuable tool for the evaluation of spinal cord dysfunction because of its ability to visualize intrinsic and compressive spinal cord lesions noninvasively. Based on our observations in this study, an initial MRI does not provide prognostic information in those patients presenting with ATM. Its major impact in patients with noncompressive spinal cord syndromes is to reveal tumor or hemorrhage.

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