
Acute Transverse Myelitis: A Retrospective Study Using Magnetic Resonance Imaging

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Abstract: The prognostic value of magnetic resonance imaging (MRI) in the syndrome of acute transverse myelitis had not been evaluated. After retrospective study of 14 cases, we found that MRI is valuable for both diagnosis and prognosis in this illness. The criteria for the diagnosis of acute transverse myelitis consisted of acute onset (over less than 3 weeks) of symmetrical motor and sensory dysfunction referable to a distinct spinal cord level, with sphincter dysfunction. Patients with abnormal MRIs of the spinal cord had significantly worse outcomes than patients with normal MRIs.

Résumé: L'imagerie par résonance magnétique nucléaire dans la myélite transverse aiguë: une étude rétrospective. La valeur de l'imagerie par résonance magnétique nucléaire pour établir le pronostic dans le syndrome de la myélite transverse aiguë n'a jamais été évaluée. Suite à une étude rétrospective de 14 cas, nous avons constaté que l'imagerie par résonance magnétique nucléaire était utile tant pour établir le diagnostic que pour évaluer le pronostic de cette maladie. Les critères diagnostiques de la myélite transverse aiguë étaient les suivants: début subit (sur une période de moins de trois semaines) d'une dysfonction motrice et sensitive symétrique reliée à un niveau spinal bien précis, avec dysfonction sphinctérienne. Les patients dont l'imagerie était anormale avaient une issue significativement plus sombre que ceux dont l'imagerie était normale.

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The pathogenesis of acute transverse myelitis (ATM) is poorly understood but is likely demyelinating and/or inflammatory, perhaps similar to that of multiple sclerosis (MS). Studies of transverse myelopathies are sparse and often contain patients with various systemic diseases, Devic's disease, multiple sclerosis, or other illnesses more specific than idiopathic ATM.^{1,2}

The term acute transverse myelitis (ATM) refers to an acute transverse spinal cord syndrome which cannot be attributed to a mass lesion, infarction, trauma, radiation, infection, or other identifiable cause. The term has been used synonymously with acute transverse myelopathy; some advocate the exclusive use of this latter term because of the lack of proof of an inflammatory process.³ However, the pathophysiologic evidence favors such a process. For example, ATM and experimental allergic encephalomyelitis (EAE) produce similar pathologic abnormalities; the latter is characterized by an inflammatory reaction (induced by a hypersensitivity to myelin basic protein).⁴ Furthermore, the term ATM has come into common usage in recent reports.^{5,6} For these reasons we favor the continued use of the term ATM in patients with this syndrome.

The term ATM may also encompass the entity of progressive necrotic myelopathy, otherwise known as subacute necrotizing myelitis.⁷ The distinction between these entities on pathological grounds has generally been made according to the degree of necrosis, but the clinical features may be very similar, making them clinically indistinguishable. Postinfectious encephalomyelitis, which most often occurs in children, also is associated

with similar pathologic changes. It is not clear if some cases of idiopathic ATM are a variant of this, simply lacking cerebral involvement. The relationship between these processes, MS, and Devic's disease is also unclear. We suggest reserving the term ATM for those patients not exhibiting evidence of any of these entities.

METHODS

The records of all patients presenting with an acute transverse spinal cord syndrome who were diagnosed as having either ATM or acute transverse myelopathy and who were admitted to one of the three cooperating hospitals between 1987 and 1991 were retrospectively reviewed. ATM was defined as a syndrome manifested by symmetrical motor and sensory dysfunction with sphincter involvement referable to a distinct spinal cord level, reaching its peak over a period of three weeks or less. All patients had MRI at the clinically appropriate spinal cord level. Patients with a prior history of carcinoma, radiation

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Table 1.

	Age, Sex	Clinical (Deficit/Onset)	MRI (time from onset)	MRI (Brain)	Outcome (and follow-up period)	Cerebrospinal Fluid
1)	14 Male	neck pain and paresthesias, severe quadriparesis after 1 day, urinary retention	abnormal (day 8), swollen cervical cord, bright signal on T2 at C-4 to C-7, patchy enhancement	Normal	some recovery (1 year)	normal*, no oligoclonal bands, IgG concentration normal
2)	63 Male	paresthesias, severe quadriparesis after 2 days, urinary retention	abnormal (day 3) punctate focus of hyperintensity, C3 sensory level	Normal	some recovery (5 years)	normal
3)	29 Male	2 weeks of LE* paresthesias, then weakness progressing to severe paraparesis over 2 days, urinary retention	normal (day 15), thoracic	Normal	complete recovery (16 months)	protein 105 (mg/dl), mild pleocytosis, normal IgG concentration, no oligoclonal bands
4)	61 Male	Low back pain and gradual moderate paraparesis over 3 weeks, distal LE parasthesis, urinary frequency	normal (day 21), thoracolumbar	Not Done	complete recovery (4 years)	normal
5)	43 Male	interscapular pain, C-5 sensory level, moderate quadriparesis over 1 day, urinary retention	normal (day 1), cervical, thoracic	Not Done	complete recovery (5 years)	normal, IgG concentration normal
6)	72 Female	UE** paresthesia, moderate paraparesis, incontinence, over 1 day	normal (day 3), thoracic	Normal	complete recovery (5 years)	mild pleocytosis
7)	69 Female	mild UE weakness for 2 weeks, then rapid severe quadriplegia, urinary retention T-5 sensory level	abnormal (day 25), bright signal on T2, C3; initial MRI normal (day 18)	Normal	poor recovery, (10 months)	normal IgG concentration, no oligoclonal bands
8)	33 Female	moderate paraparesis, T12 sensory level, over 3 days, urinary retention	abnormal (day 4) bright signal on T2 at multiple thoracic levels	Normal	poor recovery, relapse after 2 years (4 years)	mild pleocytosis, normal IgG concentration, no oligoclonal bands
9)	54 Female	12 hours of ascending numbness and moderate paraparesis; areflexic; T12 sensory level, absent anal wink	thoracic normal (day 1 and 8)	Normal	near complete motor and sensory recovery (18 months)	normal IgG concentration normal no oligoclonal bands
10)	49 Male	2 weeks LE stiffness; then urinary retention severe paraparesis, T-7 sensory level	normal thoracic (day 14)	Normal	strength, urination normal (4 years)	mild pleocytosis, mildly elevated protein
11)	30 Male	rapid onset over 24 hours of mild paraparesis, decreased LE sensation, urinary retention	normal thoracic (day 3)	Normal	full motor recovery, minor sensory loss (8 mos.)	moderate pleocytosis, mildly elevated protein
12)	46 Female	rapid onset over 24 hours C4 sensory level, moderate paraparesis, urinary retention	normal thoracic and cervical (day 7)	Normal	complete motor recovery, some sensory recovery (5 years)	normal, no oligoclonal bands
13)	17 Male	low back pain, L3 paresthesias, numbness and moderate quadriparesis over 1 day, urinary retention	normal cervical and thoracic (day 7)	Normal	near complete recovery (3 months)	moderately elevated protein, no oligoclonal bands
14)	29 Female	moderate paraparesis and with T-10 sensory level and urinary retention over 24 hours	normal thoracic and cervical (day 7)	Normal	complete recovery, minor relapse after 4 years (4 years)	normal, IgG concentration normal no oligoclonal bands

* Lower extremities

** Upper extremities

+ Normal cells, protein, glucose

therapy to the spine, a diagnosis of MS, or other identifiable cause of transverse myelopathy were excluded. Patients who presented with substantially asymmetric motor involvement, suggestive of acute partial transverse myelopathy, were also excluded, because of the demonstration that many of them are found to have MS on long-term follow-up. (The term acute partial transverse myelitis is used to refer to an acute myelopathy with asymmetric motor and sensory findings, and does not refer to the degree of dysfunction in regard to severity or completeness.)⁸ Follow-up was obtained by the authors personally in most cases and by medical records in the remainder. MRI

scanning was performed with relative T1 and T2 weighted images sagittally with 5 mm cuts and 5 mm gaps using either a Siemens 1.5 magnetom unit (D13B) or a General Electric signa unit (1.5 Tesla). All scans were interpreted by neuroradiologists who were unaware of the nature of this research.

RESULTS

Patient data are presented in Table 1. Patients 11 and 12 reported upper respiratory tract infection 1-2 weeks prior to ATM symptoms. The degree of weakness and the clinical course

were variable. All patients had some degree of either paraparesis or quadriparesis; of the 14 patients, 1 had mild, 9 moderate, and 4 severe weakness. Paresthesias and mild to moderate sensory loss occurred in all of the 14 patients; complete loss of sensation up to the spinal cord level occurred in none of the patients. One patient had some loss of pain and touch sensation but sparing of vibratory and position sense. All patients had sphincter dysfunction, seen usually as urinary retention. The interval between onset and peak of illness ranged from 12 hours to 3 weeks.

All patients had MRI testing soon after admission to one of the three hospitals involved in this study but at various points in the course of the disease, ranging from 7 to 25 days after onset of symptoms. Patients with severe motor symptoms early in the course of the illness (n = 5) tended to be admitted soon after onset and to be scanned early. Most patients scanned 7 days or more after the onset of symptoms were those who had mild motor involvement and tended to be hospitalized later. One severely involved patient (patient 1) did not have MRI until day 8 because of lack of MRI facilities in his local hospital prior to transfer to us.

Of the 14 patients, 11 had brain MRI; all were normal. All patients had cerebrospinal fluid (CSF) studies. 5/14 had abnormally increased total protein content, ranging from 56-105 mg/dl. 5/14 had CSF pleocytosis, usually mild, but patient 12 had 109 white blood cells per ml. Studies for oligoclonal bands were negative in all of the 8 patients tested. Patient 14 experienced a minor relapse after 4 years, and repeat MRI of the brain and spinal cord were normal.

Patients with spinal cord MRI abnormalities consistently exhibited a less favorable clinical course than did those with normal MRI's. Almost all patients (8/10) with normal MRI's had complete recovery, and 2 had only minor residua. Even those who had moderate to severe motor deficits had complete or near complete recovery when the MRI was normal. Of the 4 patients with abnormal MRI's, 3 were non-ambulatory and one had severely limited upper extremities function at follow-up. Thus, the MRI scan seemed to be a better prognostic indicator than the degree of motor involvement.

Abnormal areas of increased signal intensity of various sizes within the spinal cord were seen on the T-2 weighted MRI scans in 4 patients. In each case, the spinal cord appeared swollen at the site of abnormal signal. One patient had a gadopentetate dimeglumine enhanced MRI; pre-enhancement T-2 weighted images showed diffusely increased signal intensity within the cord from C3 to T7; post-infusion T1 weighted images showed patchy enhancement in this same area (patient 1). Patient 8 had repeat MRI during relapse 2 years after the initial one; mild spinal cord atrophy was the only finding on the second study and the brain was again normal. Thus our study is in agreement with the hypothesis of Tippett et al., that relapse in ATM is probably not rare (in this study 2/14 patients).⁵

DISCUSSION

The syndrome of ATM can be mimicked by a number of other disease entities, including mass lesions compressing the spinal cord. MRI is of great help in diagnosing ATM. In 10 of 14 patients, the MRI was normal; 8 of these made a complete

recovery and 2 had near complete recovery, even when clinical motor involvement was severe. The other 4 patients exhibited increased signal intensity on T2 weighting within the spinal cord, associated with swelling of the cord; these patients had a less favorable clinical outcome. Thus, MRI scanning appears to be of great value both in the diagnosis of ATM and in prognosis.

Authors of single case reports of MRI abnormalities in ATM, either idiopathic or post-infectious, proposed a possible correlation between MRI abnormalities (swelling, ischemic appearing lesions, enhancement) and poor outcome.⁹⁻¹² Barakos et al. described a group of five patients, all with abnormal MRI's, and all but one had a good recovery.¹³ In contrast we found only 29% of patients had abnormal MRI's, and these patients had poor outcomes. Austin et al. reported 8 patients with idiopathic ATM who underwent MRI within 10 days of onset.¹⁴ Only 2 of their patients made complete recoveries, and both had negative MRI's. They reported 1 patient with ATM, abnormal MRI, and severe weakness with little recovery. Despite the appearance presented by these 3 patients (that MRI correlated with outcome) the authors did not feel it was helpful for prognosis, since incomplete recovery was seen in the remaining 5 cases, regardless of MRI appearance (2 abnormal). Unfortunately follow-up time was only 1-2 months in many of the patients.

More data are needed to clarify the prognostic value of MRI in idiopathic or post-infectious ATM. Previous reports weaken but do not refute our findings, since the number of studies available is as yet limited.

Jeffrey et al. suggested clinical findings, MRI and CSF studies might help distinguish ATM from MS.¹⁵ In our cases of ATM no patients had CSF IgG abnormalities, and none developed MS.

The overall patient outcomes in this series appear to be in agreement with previous reports, with most patients having fair or good recoveries.^{16,17} We plan to further study MRI abnormalities in ATM using serial studies prospectively to attempt to verify the present study.

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