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ORIGINAL ARTICLE

Neurological and functional recovery in acute transverse myelitis patients with inpatient rehabilitation and magnetic resonance imaging correlates

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Objective: The objective of this study was to observe neurological and functional recovery in patients with acute transverse myelitis (ATM) with inpatient rehabilitation and correlate with magnetic resonance imaging (MRI) changes.

Patients and methods: The study was conducted with 43 ATM patients (19 males) admitted in the tertiary university research hospital from July 2012 to June 2014. Detailed MRI findings were noted. Neurological status was assessed using the ASIA impairment scale (AIS) and functional recovery was assessed using the Barthel Index score (BI) and Spinal Cord Independence Measure (SCIM).

Results: Patients showed significant neurological and functional recovery with inpatient rehabilitation using AIS, BI and SCIM scales when admission and discharge scores were compared ($P<0.001$). Thirty-one patients (72.1%) had rostral level in the cervical region according to MR imaging, but clinically, 17 patients had tetraplegia, whereas 26 patients had lower-limb weakness only. No definitive pattern or correlation was found between level (MRI or clinical) and neurological status (AIS).

Conclusion: The neurological outcome in patients with ATM cannot be predicted on the basis of imaging findings. There is a great variation in the imaging level and clinical presentation. Patients show significant improvement with inpatient rehabilitation even with poor functional ability in acute and sub-acute phase of illness.

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INTRODUCTION

Myelitis is a neurological disorder of the spinal cord, caused by inflammation.[1] It is often referred to in the literature as 'transverse myelitis' or 'acute transverse myelitis-ATM'. It typically presents acutely, but sub-acute presentations have been described.[2,3] Incidence of ATM varies between developed and developing countries with higher incidence in the latter. It affects individuals of all ages with some studies reporting peaks in second and fourth decade of life.[4–7] Patients mostly have a clearly defined rostral border of sensory dysfunction, and spinal magnetic resonance imaging (MRI) and lumbar puncture often show evidence of acute inflammation.[2] At its peak, patients would have motor deficits with partial or complete loss of motor power in limbs. Sensory deficits present in the form of loss of light touch and pin prick, numbness, paresthesias or band-like dysesthesias.[4–9] Autonomic symptoms consist of increased urinary urgency, bowel or bladder incontinence, difficulty or inability to void, incomplete evacuation or bowel constipation.[10]

The American spinal cord injury association (ASIA) impairment scale (AIS)[11] is commonly used to assess neurological status in spinal cord-injured patients. It has been used extensively to correlate between MR imaging and neurological status because of traumatic spine injuries but less commonly used in non-compressive lesions. Moreover, there have been few studies to assess the relationship between the MRI findings and the neurological deficit as assessed by clinical examination and AIS. In addition, upper extremity, lower extremity motor scores (UEMS and LEMS) and total motor scores (TMS) can be used to assess motor status and recovery in these patients during admission and discharge. Functional recovery in these patients is commonly assessed using the Barthel Index scale (BI)[12] and Spinal Cord Independence Measure (SCIM-II).[13]

MRI is an invaluable imaging tool in the diagnosis of ATM. Studies suggest dorsal spinal cord involvement to be more common as compared with cervical cord.[7–10,14] MRI findings include pronounced signal alterations, which are diffuse hypointense to isointense on T1 and hyperintense on T2. Although these changes are consistently observed in patients with ATM, they are not specific. Similar changes have been reported in other lesions involving spinal cord, such as neoplastic infiltration, vascular malformation, sarcoidosis, acute spinal injury, AIDS myelopathy and multiple sclerosis.[15] In the literature, there are only a few reports on MRI changes in ATM, with contradictory findings.[15–18] Extensive signal alteration in MRI suggests more extensive and severe involvement, and these patients are likely to have worse recovery compared with those with less pronounced MRI changes. Depending on the extent, severity and duration of insult, the end result after treatment may vary from complete recovery to permanent deficit (Figure 1).

The present study was conducted to observe Neurological and functional recovery in ATM patients with inpatient rehabilitation by

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Figure 1 Sagittal MR Image of cervical and dorsal spine showing 'myelitic' changes.

comparing their admission and discharge AIS, Barthel index (BI) and Spinal Cord Independence Measure (SCIM-II) scores. Detailed MRI findings in patients with ATM were noted. We tried to observe correlation between MRI findings and neurological recovery in these patients.

PATIENTS AND METHODS

This study was conducted in a tertiary university research hospital between July 2012 and June 2014. During this period, a total of 143 patients were referred to the department of neurological rehabilitation, with acute onset weakness and a clear sensory level on clinical examination. Patients were screened and worked up for myelopathies in the department of neurology. On MRI, 53 patients had compressive lesions, whereas 90 patients were diagnosed as having non-compressive myelopathy. Further, 47 patients (out of 90) were found to have relapsing remitting neurological illness, neuromyelitis optica, neuromyelitis optica spectrum disorder or multiple sclerosis on subsequent detailed work-up. They were excluded from the study. The remaining 43 patients who were diagnosed with ATM on the basis of the criteria of Transverse Myelitis Consortium Working Group (TMCWG) 2002[2] were recruited in the study. Institutional ethics committee approval was obtained, and informed written consent was taken from the patients before including them in the study. They underwent detailed clinical and neurological examination at the time of admission and were classified according to the AIS, BI and SCIM-II to assess

Table 1 Correlation between upper (rostral) level of spinal lesion (magnetic resonance imaging) and neurological status according to the ASIA impairment scale-AIS (admission vs discharge)

Neurological status-AIS		*Upper level of spinal cord lesion (according to MRI scan at the time of admission)*		
		Cervical (C1-8)	*Dorsal (D1-12)*	*Lumber/Sacral*
AIS-A	Adm.	10	4	0
	Dis.	6	4	0
AIS-B	Adm.	7	2	0
	Dis.	1	1	0
AIS-C	Adm.	12	3	1
	Dis.	4	5	0
AIS-D	Adm.	2	1	1
	Dis.	7	12	1
AIS-E	Adm.	0	0	0
	Dis.	0	1	1

Abbreviations: Adm., admission; AIS, American spinal cord injury association impairment scale; Dis., discharge.

neurological and functional status. Admission goals in most of the patients were to achieve maximum independence in performing activities of daily living, locomotion, management of bladder and bowel-related issues, and vocational and psychosocial rehabilitation. Patients received a standard rehabilitation program consisting of passive and active range of motion-strengthening program for weaker muscles. Ambulation devices and orthosis were given as necessary. Immunomodulation was received by patients during their stay in the department of neurology. Antispastic medications to control spasticity, clean intermittent catheterization for bladder care and regular bowel program were advised when appropriate. Details of MRI findings were recorded and analyzed by one of the co-authors (with Neurology background) to observe key features of spinal cord and brain involvement.

Statistical analysis

Statistical Package for the Social Sciences version 17 for Windows (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. The results were expressed as mean ± s.d. with range or median with range. Descriptive statistics included frequency, mean and standard deviation for quantitative variables such as age, duration of illness, duration of stay, BI and SCIM scores. Paired Student's *t*-test was used for the assessment of functional recovery using mean BI and SCIM scores at admission and discharge, as well as motor recovery using UEMS and LEMS. The Wilcoxon non-parametric test was used for the assessment of neurological recovery by comparing admission and discharge AIS scores. $P<0.05$ was considered to be significant.

RESULTS

Forty-three patients were included in the study (19 males and 24 females). The mean age was 30.1 years ± 15.3 (range 10–69 years). Median duration of illness at admission in the rehabilitation ward was 28 days (range 22–921 days). Mean length of stay in the rehabilitation unit was 32.6 days ± 17.2 (range 14–78 days). Patient distribution based on clinical presentation was as follows: tetraplegia in 17 (39.5%) and paraplegia in 26 (60.5%).

One patient had no changes in MRI, whereas most of the patients showed isointense changes on T1 image and hyperintense in T2 images. Cervical cord was involved in 31 (72%) and dorsal cord in 11 (26%) patients when classified on the basis of the rostral level of the involvement on MRI. The involvement of cord was single with continuity (more than three segments) in 33 (77%) patients, whereas multiple with patchy involvement was found in 9 (21%). On analysis of the transverse section of MRI of patients, less than one-third of cord

Table 2 Distribution of patients based on neurological status (AIS score) at admission and discharge with topographical and clinical presentation

AIS (ASIA impairment scale)	*Admission*	*Discharge*
AIS-A		
Tetraplegia/paresis	5	5
Paraplegia/paresis	9	5
AIS-B		
Tetraplegia/paresis	3	1
Paraplegia/paresis	6	1
AIS-C		
Tetraplegia/paresis	8	4
Paraplegia/paresis	8	5
AIS-D		
Tetraplegia/paresis	1	7
Paraplegia/paresis	3	13
AIS-E		
Tetraplegia/paresis	0	0
Paraplegia/paresis	0	2
P value	<0.05	

was involved in 19 (44%), whereas less than half of cord was involved in 21 (49%), 1 had total involvement and 1 had involvement of discrete tracts. Some form of enhancement of cord or surrounding structures was seen in 12 patients (28%), and no enhancement was visualized in 28 (66%).

Correlation between upper level of spinal lesion (MRI) and neurological status (admission vs discharge) is presented in Table 1. No pattern or correlation was observed between the level of lesion and neurological status of the patients. The MRI scan was performed only once (at or just before admission to Neurorehabilitation unit). No MRI scan was performed at the time of discharge. The neurological recovery and status was compared using the AIS scale (admission vs discharge). At admission to the neurological rehabilitation unit, 14 patients were AIS-A, 9 were AIS-B, 16 were AIS-C and 4 patients were AIS-D. At discharge, close to 50% patients were showing good neurological status recovery (AIS-D). Majority of patients with AIS-A score at admission with cervical cord demyelination did not show recovery at discharge.

The admission and discharge AIS score after rehabilitation are presented in Table 2 (irrespective of the MRI findings at admission). In tetraplegic patients, there was a significant number of patients improving from ASIA B and C to ASIA D, whereas ASIA A showed no change. On the contrary, in paraplegic patients, there was a significant number of patients improving from all levels even including ASIA A to higher levels mostly to ASIA D.

In addition, motor recovery was assessed using UEMS, LEMS and TMS (out of maximum 100 with 25 for each extremity). Mean UEMS score at admission was 39.8 ± 12.1 (11–50), whereas discharge mean score was 43.1 ± 10.5 (12–50). Mean LEMS admission score was 11.6 ± 13.2 (0–40) and discharge score was 21.8 ± 15.4 (0–43). Mean TMS at admission was 51.2 ± 16.8 (11–90) and discharge mean TMS was 63.9 ± 19.1 (12–90). While comparing admission vs discharge scores in UEMS, LEMS and TMS, significant recovery was observed ($P = 0.001$, $P < 0.001$ and $P < 0.001$, respectively) at the time of discharge. The admission and discharge BI and SCIM scores after rehabilitation are presented in Table 3. In tetraplegic and paraplegic patients, mean BI and SCIM score improved significantly, indicating better functional status after inpatient rehabilitation. Interestingly, mean BI and SCIM score of tetraplegia lagged behind the mean SCIM score of paraplegics by few points, indicating that the functional status achieved in both groups was more or less same.

Table 3 Functional recovery at admission and discharge following inpatient rehabilitation

Functional recovery	*Admission*	*Discharge*	P *value*
BI in tetraplegia	20.2 ± 24.5	55.6 ± 27.4	<0.001
SCIM-II in tetraplegia	19.6 ± 6.7	57.8 ± 18.8	<0.001
BI in paraplegics	25.4 ± 9.6	61.5 ± 20.6	<0.001
SCIM-II in paraplegics	29.4 ± 13.6	64.2 ± 15.6	<0.001

Abbreviations: BI, Barthel Index score; SCIM, Spinal Cord Independence Measure.

All patients received pulsed therapy of parenteral steroids (methylprednisolone 1 g for 5 days) followed by oral steroids in tapering doses. In addition, four patients showing no recovery received plasmapheresis and two patients received IVIg. Among 43 patients, 2 patients developed urinary tract infection, 2 had cystitis and 2 had hydroureteronephrosis on sonography. Moreover, bladder was managed with clean intermittent catheterization by self in 38 patients (88%), by assistance in 4 (10%) and 1 patient was able to void self. Eight patients (20%) had developed at least grade II pressure sore in dependent areas at the time of admission, which was managed conservatively. Prevalence of neuropathic pain was high, with as many as 31 patients reporting it, and was managed with pharmacotherapy, apart from counseling, coping strategies and physical methods. No patient developed complications such as deep vein thrombosis and heterotopic ossification during inpatient rehabilitation.

DISCUSSION

The rehabilitation approach to ATM patients requires a multidisciplinary team involving the neurologist, physiatrist, physical therapist, occupational therapist, orthotist, psychologists, nurses and social worker. Rehabilitation has to be introduced very early in the course of illness and should continue for long term, depending on the recovery status of the patients and residual deficits.[19] The goal is to improve the functional ability of the patient in performing activities of daily living through improving available joint range of motion, effective compensatory strategies and relieving pain.[20] The neurological and functional outcomes in ATM patients vary with up to two-thirds of the patients being left with significant sensory-motor deficits with bladder involvement.[21] Many studies suggest that steroids are mostly effective in the acute stage; all patients with ATM are offered immunomodulation with steroids and plasmapheresis before referring them to rehabilitation set-up. Our study showed significant neurological and functional recovery following inpatient rehabilitation, as suggested by some previous studies also.[22,23] Whether the recovery occurring as a result of pharmacotherapy including immunomodulation or rehabilitation therapy alone is difficult to ascertain. In all probability, final neurological and functional status achieved is a result of both.

There was no definitive neurological status observed in the patients based on the rostral imaging level of spinal cord in the study (Table 1). Patients showed different neurological recovery status even with the same imaging level. The role of rehabilitation becomes even more important in such an unpredictable scenario. There are studies with ATM patients that suggest that initial poor scores on functional measures would result in poor eventual recovery.[24–26] In the present study, patients with both tetraplegia and paraplegia had poor mean functional scores (using BI and SCIM) at the time of admission to rehabilitation unit. However, discharge scores showed significant increase in both the groups showing vast improvement in functional status, as reported by some earlier studies also.[27,28]

MRI showed isointense changes on T1 image and hyperintense in T2 Images in all patients in the study barring one, agreeing with previous studies.[29–32] MRI performed early in the course of illness would help in differentiating the causes of myelopathy other than ATM.[33] As the metabolic demand of cell bodies in spinal gray matter is high, this is the site most sensitive to ischemia. With increasing severity, it involves the anterior columns, central gray matter or central cord with a rim of spared peripheral white matter.[34] MRI performed immediately in patients with ATM may be normal as T2 hyperintensity and expansion evolve over hours, with enhancement developing in the sub-acute setting.[35,36] The mid-to-lower thoracic cord is most vulnerable to occlusion of the artery of Adamkiewicz,[37,38] whereas watershed ischemia typically involves the mid-to-upper thoracic cord.[36] Because of these reasons, dorsal cord involvement is reported to be more common as compared with cervical cord. However, in our series, most of the patients had involvement of cervical cord on MRI (31/43–72.1%), although many of them presented clinically as paraplegia. This pattern with the rostral level in cervical spinal cord according to imaging and clinical involvement of only lower limbs has been a common observation in the unit but difficult to explain.

Idiopathic ATM involves long segments and hence also called as 'long segment myelitis'. The lesion tends to be longitudinally extensive, spanning over two segments. In our study, we also observed that more than 95% patients had longitudinally extensive lesions spanning more than three segments.

Another important issue, which needs attention in rehabilitation of ATM patients, is bladder dysfunction, as its involvement is almost an integral part of the illness. Patients should be assessed with urodynamics and bladder management should be planned, on the basis of the findings. Family/caregiver education is essential during the early recovery period to develop a strategic plan for dealing with the challenges of independence following discharge from hospital and return to the community.

This study was conducted with selected ATM patients, and importantly, all patients consented for inpatient rehabilitation with no drop-out. Lack of follow-up to observe further recovery (or lack of it) among these patients is an issue that should have been addressed, but financial concerns of the patients and traveling could be the hindrance for reporting in the follow-up.

CONCLUSIONS

The neurological outcome in patients with ATM cannot be predicted on the basis of imaging findings. There is a great variation in the imaging level and clinical presentation. Patients tend to have poor functional ability, especially in acute and sub-acute phase of illness, and show significant improvement with inpatient rehabilitation. With multidisciplinary approach, clinical, neurological and functional outcomes tend to be better, although the study was lacking in the control group.

DATA ARCHIVING

There were no data to deposit.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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1 West TW. Transverse myelitis: a review of presentation, diagnosis and initial treatment. *Discov Med* 2013; **16**: 167–177.
2 Barnes G, Benjamin S, Bowen JD, Cutter N, de Lateur BJ, Dietrich WD *et al*. Transverse Myelitis Consortium Working Group Proposed diagnostic criteria and nosology of acute transverse myelitis. *Neurology* 2002; **59**: 499–505.
3 Schmalstieg WF, Weinshenker BG. Approach to acute or subacute myelopathy. *Neurology* 2010; **75**: S2–S8.
4 Berman M, Feldman S, Alter M, Zilber N, Kahana E. Acute transverse myelitis: incidence and etiologic considerations. *Neurology* 1981; **31**: 966–971.
5 Jeffery DR, Mandler RN, Davis LE. Transverse myelitis: Retrospective analysis of 33 cases, with differentiation of cases associated with multiple sclerosis and parainfectious events. *Arch Neurol* 1993; **50**: 532–535.
6 Christensen PB, Wermuth L, Hinge HH, Bomers K. Clinical course and long-term prognosis of acute transverse myelopathy. *Acta Neurol Scand* 1990; **81**: 431–435.
7 Altrocchi PH. Acute transverse myelopathy. *Arch Neurol* 1963; **9**: 21–29.
8 Misra UK, Kalita J, Kumar S. A clinical, MRI and neurophysiological study of acute transverse myelitis. *J Neurol Sci* 1996; **138**: 150–156.
9 Lipton HL, Teasdall RD. Acute transverse myelopathy in adults. A follow-up study. *Arch Neurol* 1973; **28**: 252–257.
10 Sakakibara R, Hattori T, Yasuda K, Yamanishi T. Micturition disturbance in acute transverse myelitis. *Spinal Cord* 1996; **34**: 481–485.
11 Ditunno JF Jr, Young W, Donovan WH, Creasey G. The international standards booklet for neurological and functional classification of spinal cord injury. *Paraplegia* 1994; **32**: 70–80.
12 Mahoney FI, Barthel D. Functional evaluation: the Barthel Index. *Md State Med J* 1965; **14**: 56–61.
13 Catz A, Itzkovich M, Tesio L, Biering-Sorensen F, Weeks C, Laramee MT *et al*. A multicenter international study on the Spinal Cord Independence Measure, version III: Rasch psychometric validation. *Spinal Cord* 2007; **45**: 275–291.
14 Dawson DM, Potts FM. Acute nontraumatic myelopathies. In: Wilsey RM, Young RR (eds). *Neurologic Clinic: Disorders of Spinal Cord*, Vol 55. WB Sanders company: Philadelphia, PA, USA, 1991, pp 551–603.
15 Barakos JA, Mark AS, Dillon WP, Norman D. MR imaging of acute transverse myelitis and AIDS myelopathy. *J Comput Assist Tomogr* 1990; **14**: 45–50.
16 Bitzan M. Rubella myelitis and encephalitis in childhood: a report of two cases with magnetic resonance imaging. *Neuropaediatrics* 1987; **18**: 84–87.
17 Merine D, Wang H, Kuma AJ, Zinreich SJ, Rosenbaum AE. CT myelography and M.R. imaging of acute transverse myelitis. *J Comput Assist Tomogr* 1987; **11**: 606–608.
18 Sanders KA, Khandji AG, Mohr JP. Gadolinium MRI in acute transverse myelopathy. *Neurology (Minneapolis)* 1990; **40**: 1614–1616.
19 Sadowsky CL, Becker D, Bosques G, Dean JM, McDonald JW, Recio A *et al*. Rehabilitation in transverse myelitis. *Continuum (Minneap Minn)* 2011; **17**: 816–883.
20 Kaplin AI, Crishnan C, Deshpande DM, Pardo CA, Kerr DA. Diagnosis and management of acute myelopathies. *Neurologist* 2005; **11**: 2–18.
21 Nakipoglu GF, Ozgirgin N. Urodynamic evaluation and rehabilitation outcomes in transverse myelitis. *Neurosciences* 2009; **14**: 37–40.
22 Gupta A, Taly AB, Srivastava A, Murali T. Non-traumatic spinal cord lesions: epidemiology, complications, neurological & functional outcome of rehabilitation. *Spinal Cord* 2009; **47**: 307–311.
23 Debette S, de Sèze J, Pruvo JP, Zephir H, Pasquier F, Leys D, Vermersch P. Long-term outcome of acute and subacute myelopathies. *J Neurol* 2009; **256**: 980–988.
24 Calvo AC, Mané Martínez MA, Palau AA, Escuer JB, Pinel LR, Martínez-Yélamos S. Idiopathic acute transverse myelitis: outcome and conversion to multiple sclerosis in a large series. *BMC Neurol* 2013; **13**: 135.
25 De Seze J, Stojkovic T, Breteau G, Lucas C, Michon-Pasturel U, Gauvrit JY *et al*. Acute myelopathies:clinical, laboratory and outcome profiles in 79 cases. *Brain* 2001; **124**: 1509–1521.
26 Bruna J, Martínez-Yélamos S, Martínez-Yélamos A, Rubio F, Arbizu T. Idiopathic acute transverse myelitis: a clinical study and prognostic markers in 45 cases. *Mult Scler* 2006; **12**: 169–173.
27 Boey K-F, Chan ML. Transverse myelopathy in SLE: clinical features and functional outcomes. *Lupus* 1996; **5**: 294–299.
28 Calis M, Kirnap M, Calis H, Mistik S, Demir H. Rehabilitation results of patients with acute transverse myelitis. *Bratislava Lek Listy* 2011; **112**: 154–156.
29 Choi K, Lee K, Chung S. Idiopathic transverse myelitis. *Am J Neuroradiol* 1996; **17**: 1151–1160.
30 Krishnan C, Kaplin A, Deshpande D, Pardo C. Transverse myelitis: pathogenesis, diagnosis and treatment. *Front Biosci* 2004; **9**: 1483–1499.
31 Alper G, Petropoulou KA, Fitz CR, Kim Y. Idiopathic acute transverse myelitis in children: an analysis and discussion of MRI. *Mult Scler* 2011; **17**: 74–80.
32 Harzheim M. Discriminatory features of acute transverse myelitis: a retrospective analysis of 45 patients. *J Neurol Sci* 2004; **217**: 217–223.

33 Goh C, Desmond PM, Phal PM. MRI in transverse myelitis. *J Magn Reson Imaging* 2014; **40**: 1267–1279.
34 Mawad ME, Rivera V, Crawford S, Ramirez A, Breitbach W. Spinal cord ischemia after resection of thoracoabdominal aortic aneurysms: MR findings in 24 patients. *Am J Roentgenol* 1990; **155**: 1303–1307.
35 Hirono H, Yamadori A, Komiyama M, Yakura H, Yasui T. MRI of spontaneous spinal cord infarction: serial changes in gadolinium-DTPA enhancement. *Neuroradiology* 1992; **34**: 95–97.
36 Friedman DP, Tartaglino LM, Fisher AR, Flanders AE. MR imaging in the diagnosis of intramedullary spinal cord diseases that involve specific neural pathways or vascular territories. *Am J Roentgenol* 1995; **165**: 515–523.
37 Fujikawa A, Tsuchiya K, Takeuchi S, Hachiya J. Diffusion weighted MR imaging in acute spinal cord ischemia. *Eur J Radiol* 2004; **14**: 2076–2078.
38 Stepper F, Lovblad K. Anterior spinal artery stroke demonstrated by echo-planar diffusion weighted imaging. *Eur J Radiol* 2001; **11**: 2607–2610.