

MRI CHARACTERISTICS OF ACUTE TRANSVERSE MYELITIS WITH CLINICOTHERAPEUTIC CORRELATION

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Abstract : Acute transverse myelitis is an acute or sub acute inflammation of spinal cord characterized by motor, sensory & autonomic dysfunction resulting from the involvement of both halves of spinal cord. 50 patients of noncompressive myelitis admitted in the department of neurology between January 2002 to June 2004 were included our study. 40 patients were diagnosed as transverse myelitis on the basis of criteria laid down by Jaffery & Madlee. All patients were subjected to routine biochemical investigations, CSF X-RAY of spine M R imaging spine and scan of brain if indicated. The presenting features were quadripareisis in 24 patients (60%) paraparesis in 16 patients (40%). Bladder & bowel involvement was seen in 35 patients (87%) sensory loss in 29 patients (72%). All the patients had acute onset of symptoms which peaked by 6.5 (range 1-15) days. High signal intensity on T2 weighted non contrast M R imaging was seen in 32 patients (80%), 5 patients showed only cord expansion while it was normal in 3. On contrast M R imaging, hyper-intense shadow was seen in 3 patients having normal M R imaging, 3 patients showed hypointensity on T1. The lesion extended for a mean of 6 spinal segments (range 4-12 segments). The high signal intensity occupied more than 2/3 of the cross sectional area of the cord in 34 patients (85%), 30 patients also had a small dot known as central dot sign. It was observed that the neurological deficit was severe and response to treatment was poor in patients having diffuse involvement of the cord (more than 8 spinal segments) and in the patients having necrosis of the cord. This study has shown a beneficial effect of methylprednisolone.

INTRODUCTION

Spinal cord is the site of infectious and non infectious inflammatory process¹. Acute transverse myelitis is an acute or subacute inflammation^{1,2} of spinal cord, characterized by motor, sensory and autonomic dysfunction resulting from the involvement of both halves of spinal cord after secondary causes such as compressive lesions, tuberculosis, syphilis were arteriovenous malformation, trauma, malignant infiltration excluded. The essential features in acute transverse myelitis include oedema, necrosis and demyelination. White matter changes are more pronounced than than in the grey matter. The brunt of disease falls on the thoracic or lumbar spinal cord although oedema and demyelination extend upwards to cervical cord or even higher³. Many reports of MR imaging in patients of acute transverse myelitis have revealed local enlargement of the cord and increased signal intensity on long repetition time/echo time (TR/TE) sequences^{1,2}. However these findings cannot differentiate this condition from other lesions such as intramedullary tumours, multiple sclerosis, hematoma and vascular ischaemia.

There are very few reports on the prevalence and pattern of contrast enhancement in patients with transverse myelitis. This study was carried out to differentiate transverse myelitis from intramedullary abnormalities using contrast and to correlate these changes with neurological deficit at the onset and response to treatment.

MATERIAL AND METHODS

Fifty (50) patients of noncompressive myelitis admitted in the Department of Neurology, between January 2002 to June 2004, were included in our study. 40 patients were diagnosed as transverse myelitis on the basis of criteria by Jeffery et al⁴.

1. Acute or subacutely developing motor, sensory and sphincter disturbances.

2. Spinal segmental level of sensory disturbances with a well defined upper limit.
3. No clinical or laboratory evidence of spinal cord compression.
4. Absence of other known neurologic diseases such as syphills, previously diagnosed multiple sclerosis, malignant neoplasm, spinal cord arteriovenous malformation, saecoidosis and HIV infection.
5. Lack of clinical progression over 4 weeks.

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 (clinically and radiologically). Neurological examination was carried on all patients. Weakness was assessed by Medical Research Council (MRC) scale and spasticity by Ashworth scale⁵.

Routine laboratory investigations included complete blood cell count, fluorescent antinuclear antibodies test, Rheumatoid factor, VDRL, test for vitamin B₁₂, folate assay; cerebrospinal fluid examination that included white blood cells with differential count, protein, sugar, gram stain, acid fast bacillus, India ink preparation, enzyme-linked immunosorbent assay for parasites, electrophoresis, human T-cell leukemia/lymphotropic virus-1 and viral titre.

All patients underwent X-ray of spine depending on the level of neurologic deficit. Magnetic Resonance Imaging spine was obtained on 1.5 Tesla super conducting magnet. Both T1 and T2 weighted images were taken. Contrast study was done where ever required. Scanning of brain was done in 8 patients who had non specific symptoms like headache and dizziness in order to exclude possible intracranial lesion.

The vertebral segmental length of the hypertense 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 was determined by finding on the axial T2 weighted images. Cord expansion was evaluated with respect

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to adjacent normal cord on T1 weighted images. The central linear high signal intensity that was seen in some cases above and below the diffuse high intensity on T2 weighted images was not considered in determining the extent of abnormality. Location and extent of the intramedullary contrast enhancement were confirmed on sagittal T1 weighted images 20 patients were treated with intravenous methylprednisolone at a dose of 1 gm/day for 5 days and the other 20 were not given methyl-prednisolone and the patients were followed for one year. The main outcome criteria were- (1.) proportion of patients able to walk independently at one month; (2.) proportion of patients with full recovery within one year; (3.) proportion of patients developing complications-namely corticosteroids side effects (persistent hyperglycemia, high blood pressure) bed sore and urinary tract infections.

RESULTS

The study included 24 females and 16 males (F:M =1.5:1). Mean age was 40 years (range 15-50 years). The *antecedent event* was non specific infection of upper respiratory tract seen in 19 patients (47.5%) while it was *idiopathic* in rest of the cases.

The presenting *features* were quadriplegia in 24 patients (60%), paraparesis in 16(40%). Bladder and bowel

Table-1: Clinical presentation of Acute Transverse Myelitis

Clinical features	No. of patients
Paraparesis	16(40%)
Quadriplegia	24(60%)
Symmetrical	32(80%)
Bowel and bladder	35(87.5%)
Sensory level	29(72.5%)

involvement was seen in 35 patients (87.5%), sensory level in 29 patients (72.5%). The symptoms were symmetric in 32 (80%) while there was asymmetry in 8 patients (20%) as shown in table 1.

All the patients had acute onset of symptoms which peaked by 6.5 (range 1-15) days. 4 patients (10%) had suffered from similar attack some years back. In these patients screening of brain was also done to rule out any possibility of multiple sclerosis. *Cerebrospinal fluid* was abnormal in 36 patients (90%); it revealed mild to moderate lymphocytic pleocytosis with mean of 7 cells/³ (range -10 cells/min) mild to moderate rise in proteins with a mean of 65 mg% (range 35-105 mg%). The CSF sugar was normal and there were no bacteria or fungi on smear or culture.

High signal intensity on T2 weighted non contrast Magnetic Resonance Imaging was seen in 32 patients (80%); 5 patients showed only cord expansion while it was normal in 3. On contrast M R Imaging, hyper-intense shadow as seen in the 5 patients having normal M R Imaging. 3 patients showed

Table -2: Spectrum of Magnetic Resonance Imaging changes

S.No	Magnetic Resonance Imaging findings	No. of Patients
1.	High signal intensity on T2 weighted images	32(80%)
2.	Hypointensity on T1	5(2.5%)
3.	Hyperintensity occupying more than 2/3 rd of cross section	34(85%)
4.	Mean spinal segments involved by hyperintensity	6

hypointensity on T1. The lesion extended for a mean of 6 spinal segments (range 4-12 segments). The high signal intensity on axial T2 weighted images was centrally located in all the patients and occupied more than 2/3rd of the cross sectional area of the cord in 34 patients (85%) as shown in table 2. Thirty (30) of the 32 patients having hyperintensity also had a small dot, isointense with the cord in the core of hyperintensity which is known as *central dot sign*. It was observed that the neurological deficit was severe and response to treatment was poor in the patients having diffuse involvement of the cord (more than 8 spinal segments) and in the patients having hypointensity on T1 weighted images signifying necrosis of the cord. Among 20 patients who were treated with methylprednisolone 14 patients (70%) walked independently after one year. Full recovery was seen in 10 patients (50%); the mean time of walking was 30 days. While in the non prednisolone group only eight patients (40%) walked independently after one year. Full recovery was seen in 7 patients (28%); the mean time of recovery was 45 days.

DISCUSSION

Acute transverse myelitis, a fragment of disseminated vasculomyelinopathy^{6,7}, is pathologically identical with acute disseminated encephalomyelitis^{8,9}. The hallmark lesion in *acute transverse myelitis* is perivenular inflammation and surrounding demyelination¹⁰. Middle aged adults are commonly affected and the most common site of involvement is the thoracic spinal cord. There are many etiological associations such as viral illness assignments, demyelinating processes such as multiple sclerosis, collagen vascular disease, vascular disorders and paraneoplastic syndromes but most cases are **idiopathic**¹¹. M R imaging is the investigation of choice to delineate such lesions in the cord^{12, 21}.

In literature various studies have documented M R imaging changes in transverse myelitis but few studies have correlated the radiological changes with the severity of clinical presentation, response to methylprednesolone and prognosis¹³. Largest series of M R imaging changes in transverse myelitis was reported by Choi etal¹⁴. Earlier reports have documented local enlargement of the spinal cord and increased signal intensity on long repetition time/echo time sequences. In one series, high signal intensity over several spinal segments along with focal cord enlargement, has been observed.

The central isointensity or dot is believed to represent central gray matter, sequestered by the surrounding edematous cord. In our series and by the series reported by Choi et.al.¹⁴ a significant number of patients had this centrally located high signal

intensity occupying more than $2/3^{\text{rd}}$ of the cross sectional area of the cord. It is not a specific sign for transverse myelitis and has been reported in the cases of intramedullary tumors. When contrast was given in the patient there was a peripheral enhancement with maintenance of the cord contour, this contrast enhancement may be confused with intramedullary tumors but in those cases contrast enhancement is profound and occupies the entire cross sectional area of the cord on at least one T1 weighted axial image and it is heterogeneous associated with central or marginal cavity. Similar observations were made by Choi et.al.¹⁴, Barkos et.al.¹⁵ & Aichner et.al.¹⁶ Commonly 3-4 spinal segments are involved Barkos et al¹⁵ & Bruna J¹⁹ reported signal abnormality extending over at least six spinal segments. Misra and colleagues¹³ have reported unusual cases of acute transverse myelitis with long segment involvement (cervical to conus). In our series the lesions extended for average 6 spinal segment. The length of spinal segments was directly related to the severity of neurological deficit and predicted prognosis as also reported earlier in the series earlier.

3 patients who showed hypointensity on T1 weighted images showed slow recovery suggesting that the hypointensity represent parenchymal or myelomalacic changes. Spinal multiple sclerosis is also associated with similar M R imaging findings but here the hypereintensity extends for not more than 2 segments, enhancement on contrast is central and the plaques are large, multiple, sharply demarcated and sometimes confluent (*kissing plaques*).

Our study showed a good response with high dose methylprednisolone; similar observation was made by Deference et al¹⁷ and sabire et al¹⁸ Chan K H, Tsang KL et al²⁰ but the results were unsatisfactory in few shades. However a multicentric study having a large sample size is needed to have final conclusion.

CONCLUSION

The characteristic findings of transverse myelitis on M R imaging include normal size or segmental enlargement of the cord most commonly thoracic, central hyperintensity

occupying more than $2/3^{\text{rd}}$ of cross sectional area of the cord. It usually affects more than 3 spinal segments central; dot in the core of hyperintensity. There is peripheral contrast enhancement of lesion. Apart from this we can also appreciate necrosis of cord in form of hypointensity on T1 weighted images. These findings can differentiate transverse myelitis from multiple sclerosis, cord tumors or other intramedullary lesions. Prognosis is poor in the patients having long hyperintense signals and necrosis. This study has shown a beneficial effect of methylprednisolone. Our study is one of the few studies that has simultaneously analysed the clinicoradiological correlation of transverse myelitis and response to high dose corticosteroids.

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Renal Transplant Outcome in High Cardio Vascular Risk Recipients

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Cardiovascular (CV) disease is the foremost cause of morbidity in renal transplant recipients. The disease burden is likely to increase as older patients are accepted for transplantation. The outcome of these high-CV risk patients after renal transplantation, especially with known pre-transplant coronary artery diseases (CAD). All renal transplants performed between 1998 and 2002 at our centre, followed up to 2005, were divided into high- and low-risk groups, based on the presences of one or more of the following: pre-transplant angina, myocardial infarction, and positive coronary angiogram. The two groups were compared for post-transplant cardiac events and patient and graft survival. The factors predictive of post-transplant cardiac event were also determined by Cox-regression multivariate analysis.

Forty-five patients (10.5%), out of 429, had post transplant cardiac events; 31.3% in high risk, and 6.5% in the low-risk group ($p=0.001$). Five yr patient survival was lower in the high risk group (82.8% vs. 93.1%, $p=0.004$), while five-yr overall graft survival and death censored graft survival were statistically not different (74.8% vs. 84.1%, $p=0.008$ and 87.3% vs. 90%, $p=0.25$), 41% patients who were treated with angioplasty plus stenting or bypass graft prior to transplantation had post-transplant cardiac events, as compared with 28% of those without intervention in the high risk group and 6.5% of patients in the low risk group ($p=0.001$). Age, pre-transplant cardiac disease, arrhythmias, and low ejection fraction ($\leq 40\%$) were significant independent predictors of post-transplant cardiac event. Post-transplant survival of high-CV risk patients (with Known CAD) is lower than that of low-risk recipients but remain acceptable. Cardiac interventions may reduce perioperative risk but do not reduce the probability of post transplant cardiac events of that of low-risk group.