Paraplegia **32** (1994) 593–596

Transverse myelitis—neurophysiological and MRI correlation

U K Misra DM, J Kalita DM

Department of Neurology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Post Box no. 375, Lucknow, India.

Acute transverse myelitis (ATM) rarely involves the cervical spinal cord. We report on two patients with ATM and cervical cord involvement. Magnetic resonance imaging (MRI) revealed high intensity signals in T2 extending far beyond the sensory level in both patients. Motor evoked potential (MEP) was unrecordable in all but one limb, and there it was marginally prolonged. Tibial somatosensory evoked potential (SEP) was unrecordable while median SEP was normal in both patients. Both MEP and SEP correlated with their respective clinical deficits but MEP seemed to correlate better with the MRI changes.

Keywords: transverse myelitis; MRI; evoked potentials.

Introduction

Acute transverse myelitis is defined as acute loss of sensory, motor and bladder functions without evidence of antecedent neurological disease or evidence of spinal cord compression. It commonly affects the lower thoracic cord and the cervical spinal cord is involved in 10% patients only.[1] There is limited experience of MRI and evoked potential changes in ATM.[2–5] With MR imaging, the structural alterations in ATM can be studied with greater detail and provide a sound basis for analysing the functional alterations. We report two patients with ATM, correlating MRI and evoked potential changes.

Case reports

Patient no. 1 (CR no. 20419)

A 40 year old male had an acute onset of lower limb weakness, which reached its peak on the third day. He also noticed retention of urine and hiccups. His symptoms remained static from the fourth day onwards. There was no history of neurological illness, tuberculosis, diabetes, collagen disease or STD, but he had fever 10 days prior to the present illness. The patient had a flaccid paraplegia of grade 0, in a 0–5 Medical Research Council (MRC) scale. The plantar responses were extensor and all sensations were absent below T8. The upper limbs were slightly weakened (MRC 4). Biceps and triceps reflexes were reduced. There was no vertebral tenderness. Blood counts, urinalysis, liver function tests, serum electrolytes and serum creatinine were normal. On admission his erythrocyte sedimentation rate (ESR) was 26 mm for the first hour. Antinuclear antibodies (ANA), rheumatoid factor, VDRL and ELISA test for HIV were negative. Cerebrospinal fluid (CSF) revealed protein 495 mg/dl, sugar 88 mg/dl and cells 310/mm^3. Spinal MRI revealed diffuse hyperintense signals on T2 extending from C2 to conus. There was no widening of the cord or gadolinium enhancement.

Patient no. 2 (CR no. 120505)

A 15 year old boy developed weakness of both legs which progressed to involve the upper limbs and there was also retention of urine, within 24 h. On the second day, he noticed breathing difficulty; thereafter his symptoms remained unchanged. There was no significant antecedent medical history. On examination he was dyspnoeic, single breath count was 16, respiratory rate 25/min, BP 120/80 mmHg. He had a flaccid hyporeflexic quadriparesis: lower limb power was grade 0 and upper limb 2–3 (MRC). The plantar response was bilaterally extensor. Pinprick was reduced by 75% below T4, joint position and vibration sense were absent in the lower but normal in the upper limbs. His blood counts, urinalysis, serum electrolytes, blood sugar, blood urea, serum creatinine and liver function tests were normal. ESR was 28 mm in the first hour; ANA, rheumatoid factor amd VDRL were negative. CSF exami-

nation revealed protein 132 mg/dl, sugar 45 mg/dl and 172 cells/mm^3. Spinal MRI revealed diffuse hyperintensity on T2 extending from the lower dorsal region to the cervicomedullary junction which was hypointense on T1. There was no gadolinium enhancement. The vertebral bodies and the disc spaces were normal (Fig 1).

Summary

Both the patients started improving after 10 days of illness. Two weeks after onset patient no. 1 had normal and patient no. 2 had gained one grade in upper limb power and had no breathing difficulty. The lower limb power and bladder dysfunction remained unchanged in both patients. Nerve conduction and electromyography (EMG) were performed in patient no. 1 after 1 month. Median, ulnar motor nerve conduction velocity (NCV) and sural sensory conduction velocity were normal but peroneal motor conduction was unrecordable. Concentric needle EMG revealed fibrillation in the tibialis anterior (TA), gastrocnemius, vastus medialis, right first dorsal interosseus (FDI) and biceps. No motor unit potential was recorded from TA, gastrocnemius and vastus medialis. Biceps and

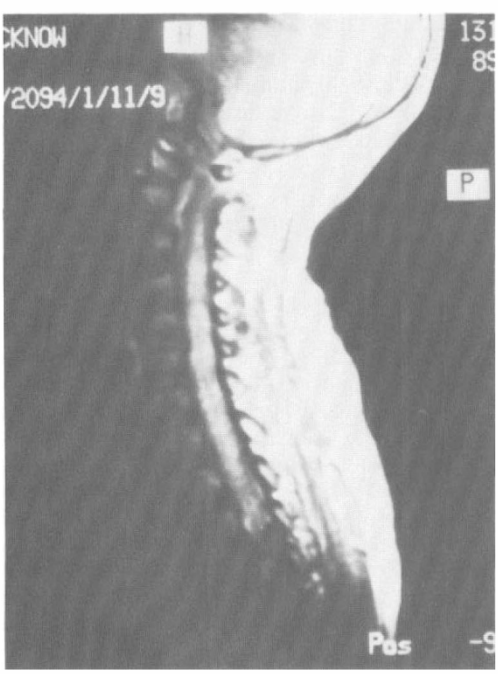

Figure 1 Spinal MRI of patient no. 2, sagittal section showing hyperintense signals in T2 extending up to cervicomedullary junction.

FDI revealed long duration, 20–40% polyphasic motor unit potentials with poor recruitment. Electromyographic findings were suggestive of widespread and asymmetric denervation and reinnervation which were consistent with anterior horn cell involvement.

Evoked potentials. Conductions in motor and sensory pathways were evaluated by respective evoked potential study.

Motor evoked potential (MEP). To stimulate the motor cortex, cervical and lumbar spine a Digitimer D180 (Digitimer Ltd, UK) stimulator delivering a single electrode shock up to 750 V with a time constant of 50–100 μs was used. The stimulating electrode was a 1 cm diameter saline soaked felt pad mounted on a plastic handle. To activate abductor digiti minimi (ADM) the cathode was placed at the vertex and anode 7 cm laterally and 1 cm anterior to a line drawn from vertex to tragus. To activate the tibialis anterior (TA) the anode was kept at the vertex and cathode 7 cm posterior to it. For cervical and lumbar stimulation the cathode was placed below the spinous processes of C7 and T12 respectively, with the anode proximal to the cathode. Motor evoked potentials (MEPs) were recorded by a surface electrode placed on the ADM or TA in a belly tendon mountage. During cortical stimulation the patient was asked to contract the target muscle slightly (10% of maximum force) whereas during spinal stimulation the patient was asked to relax. EMG signals were filtered through 20 KHz–2 KHz, at a gain of 0.5–2 mV/division. Stimulus intensity was 90–100% for cortical and 50–60% of maximum output for spinal stimulation. Onset latency and amplitude of the negative phase of the MEP was recorded. Central motor conduction time (CMCT) was calculated for the upper limb (CMCT-ADM) by subtracting the latency on C7 stimulation from that on cortical stimulation, and that for the lower limb (CMCT-TA) by subtracting the latency on L1 stimulation from that on vertex stimulation.[6]

Somatosensory evoked potential (SEP). Median and tibial SEPs were recorded by stimulating the median nerve at the wrist and the tibial nerve at the ankle by 0.1 ms square wave pulse at 3 Hz, sufficient to produce a painless twitch. The recordings were made from contralateral parietal cortex and ipsilateral Erb's point referred to Fz for median SEP, while Cz referred to Fz and L1 referred to L3 for tibial SEP. Five hundred and twelve responses were twice averaged at a gain of 2 μv/division, sweeptime 100 ms, filter setting 2–3000 Hz. Central sensory conduction time (CSCT) for median SEP was calculated by the difference between the

latencies of Erb's point and N20 potentials. Central sensory conduction time for the tibial nerve was calculated as the difference between cortical potential P40 and lumbar potential.[7]

The evoked potential results were compared with our laboratory controls which were derived from 32 healthy adult volunteers. The upper limit was defined by mean ± 2.5 SD. The mean ± SD and the cut off points in controls are shown in Table I. The results of the evoked potential studies in the patients are summarised in Table I. Motor pathways to the lower limbs were inexcitable and tibial SEPs were also unrecordable in both the patients. Motor pathways to the upper limbs were inexcitable in both the patients except in the left side of patient no. 1, in whom CMCT-ADM was marginally prolonged (Fig 2). Median SEPs were normal in both the patients. Repeat MEP and SEP study after 1 month did not reveal any change.

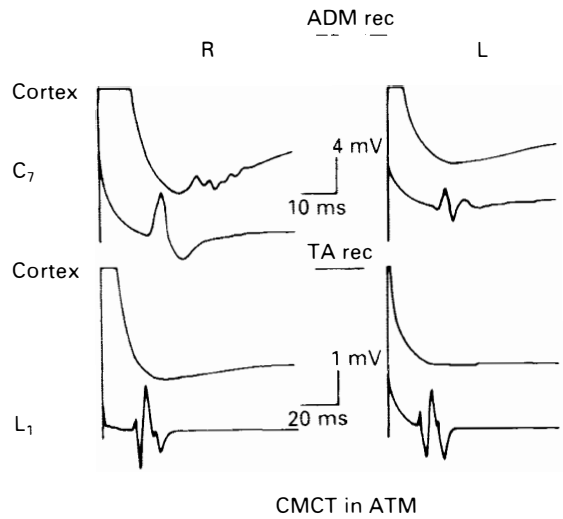

Figure 2 Motor evoked potential studies in patient no. 2, showing inexcitable central motor pathways to both left and right upper limbs. CMCT to the left upper was prolonged (8.4 ms).

Discussion

The usual manifestation of ATM is paraplegia. There was significant upper limb weakness in our patients although it was less marked compared to that of the lower limbs. ATM is regarded as an inflammatory or necrotising disease and the upper sensory level is thought to correspond with the segmental site of the lesion.[8] In both our patients the sensory level which was at T8 and T4 suggested that the disease was involving the thoracic spinal cord, although the motor deficits were consistent with some degree of cervical involvement. The neurological signs however were symmetrical in both patients, but EMG and CMCT-ADM were consistent with asymmetric involvement of the spinal cord. In spite of clinical improvement in both patients, MEP and SEP did not show any change. Autoimmune myelitis primarily affects the white and viral myelitis the grey matter, although this distinction is not always clear.[8] In patient no. 1, widespread asymmetrical denervation seen in the EMG was consistent with anterior horn cell involvement. In primarily demyelinating disorders, CMCT and CSCT are prolonged[7,9] but in our patients the central motor pathways were inexcitable in all lower and upper limbs except one, in which

Table I The result of evoked potential studies in ATM

Evoked potentials	Patient no. 1		Patient no. 2		Control value
	R	L	R	L	Cut off point (mean ± SD) ms
MEP					
CMCT–ADM (ms)	NR	NR	10.4	NR	8.1 (5.1 ± 1.2)
CMCT–TA (ms)	NR	NR	NR	NR	16.1 (12.1 ± 1.6)
SEP					
CSCT median (ms)	8.4	8.4	10.4	10.4	11.3 (8.3 ± 1.2)
CSCT tibial (ms)	NR	NR	NR	NR	27.1 (20.1 ± 2.8)

NR = not recordable.

CMCT-ADM was marginally prolonged. Tibial SEPs were also unrecordable. In 12 patients with ATM, median SEP was normal but peroneal SEP was abnormal in five out of six patients.[5] The role of MEP in the assessment of ATM has not been evaluated in the available literature.

The MRI changes in our patients extended across the cervical region and appeared more extensive than was suggested by the clinical signs. The few reports on the MRI changes in ATM are incompatible. Increased signal intensity and normal cord intensity have been reported on long TR sequences.[3,4] In a recent report, however, all six patients had abnormal increase in signal intensity on T2 which extended at least six segments above the sensory level in five out of six patients.[2] These MRI changes are consistent with the reported histopathological findings of diffuse infiltration and oedema of the spinal cord.[8] The signal changes in MRI correlated better with MEP than with SEP. From this study we conclude that the involvement of the cervical spinal cord in ATM is commoner than is generally appreciated and the structural abnormalities can be better documented by MRI. The evoked potentials are useful in documenting the respective sensory or motor dysfunction, although MEP seems to correlate better with MRI changes than SEP.

References

1 Dawson DM, Potts F (1991) Acute nontraumatic myelopathies. In: Woolsey RM and Young RR, editors. *Neurologic Clinics. Disorders of the Spinal Cord*. WB Saunders company, Philadelphia: 551–603.
2 Barakos JA, Mark AS, Dillon WP, Norman D (1990) MRI imaging of acute transverse myelitis and AIDS myelopathy. *J Comput Assist Tomogr* **21**: 45–50.
3 Bitzan M (1987) Rubella myelitis and encephalitis in childhood: a report of two cases with magnetic reponse imaging. *Neuropediatrics* **18**: 84–87.
4 Merine D, Wang H, Kumar AJ *et al* (1987) CT myelography and MR imaging of acute transverse myelitis. *J Comput Assist Tomogr* **11**: 606–608.
5 Ropper AH, Miett T, Chiappa KH (1982) Absense of evoked potential abnormalities in acute transverse myelopathy. *Neurology* **32**: 80–82.
6 Misra UK, Sharma VP (in press) Central and peripheral conduction studies in lathyrism. *J Neurol Neurosurg Psychiatry*.
7 Chiappa KH (1985) *Evoked Potentials in Clinical Medicine*. Raven Press, New York: 203–234.
8 Walton J (1986): Myelitis (myelopathy). In: Walton J, editor. *Brain's Diseases of the Nervous System*. Oxford University press, Oxford: 420–422.
9 Hess CW, Mills KR, Murray NMF, Schriefer TN (1987) Magnetic brain stimulation: Central motor conduction studies in multiple sclerosis. *Ann Neurol* **22**: 744–755.