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Original Article

Is methyl prednisolone useful in acute transverse myelitis?

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Study design: Hospital based observational study.
Objectives: To evaluate the role of methyl prednisolone (MPS) in the management of acute transverse myelitis (ATM).
Methods: Twenty-one patients with ATM were included in a prospective hospital based study during 1992–1997. All the patients underwent neurological examination, spinal MRI, somatosensory and motor evoked potentials of both upper and lower limbs and concentric needle EMG study. Twelve consecutive patients did not receive MPS therapy who were managed during 1992–1994 and nine consecutive patients during 1995–1997 received MPS therapy in a dose of 500 mg i.v. for 5 days. The clinical and neurophysiological studies were repeated 3 months later. The outcome was defined on the basis of Barthel index (BI) score at the end of 3 months into good ($BI \geqslant 12$) and poor ($BI < 12$).
Results: The age of MPS group was 25.5 years (range 12–42) and three were females. The age of non MPS group was 33.5 years (range 16–70) and two were females. In the MPS group 33% had poor outcome compared to 67% in the non MPS group. In the MPS group mean admission BI score was 7.3 which improved to 14.6 after MPS therapy. In the non MPS group, the admission BI score was 3.2 which improved to 9.6 at 3 month follow-up. In patients with complete paraplegia, evidence of denervation on EMG and unrecordable central motor conduction time to lower limb and tibial SEP were associated with poor outcome irrespective of MPS treatment. Global test statistics did not suggest a beneficial role of MPS therapy in the outcome of ATM.
Conclusion: Our results do not suggest a beneficial role of methyl prednisolone on the 3 month outcome of ATM.
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Keywords: methyl prednisolone; myelitis; denervation

Introduction

Acute transverse myelitis (ATM) is attributed to an immunlogical response against the central nervous system.[1] It usually follows infectious diseases, vaccination and occasionally occurs without any definite antecedent event. High dose MPS therapy has been shown to be beneficial in acute transverse myelitis.[2,3] Steroid or ACTH treatment, however, were not found to be beneficial in patients with necrotising myelitis in an earlier study.[4] A controlled trial on the use of corticosteroids in ATM is lacking. High dose methyl prednisolone has been found to be more useful than prednisolone or ACTH in controlling acute exacerbations of multiple sclerosis and a number of other immunological disorders.[5–7] We have reported clinical and evoked potential changes in ATM following methyl prednisolone therapy and suggested a possible beneficial role.[8] In this study, we compare the effect of MPS therapy in ATM with those not receiving any form of corticosteroids.

Subject and methods

The patients with acute transverse myelitis based on the following criteria have been included.[9]

(1) Acute or subacutely developing motor, sensory and sphincter disturbance.
(2) Spinal segmental level of sensory disturbance with a well defined upper limit.
(3) No clinical or laboratory evidence of spinal cord compression.

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(4) Absence of other known neurologic diseases such as syphilis, previously diagnosed multiple sclerosis, malignant neoplasm, spinal cord arteriovenous malformation, sarcoidosis and HTLV-1 infection.
(5) Lack of clinical progression beyond 4 weeks.

All the patients underwent a detailed neurological examination. Weakness was assessed by Medical Research Council (MRC) scale, tone by Ashworth scale,[10] and tendon reflex, plantar response and sensations were also recorded. Haemoglobin, blood counts, ESR, blood chemistry, serum test for syphilis, HIV, rheumatoid factor and antinuclear antibodies (ANA) were studied in all the patients. CSF was examined for protein, sugar, cell, bacteria and fungi. Spinal MRI was carried out on a 2T superconducting system operating at 1.5 T using a flat oval surface coil. All images were obtained employing multislice spin echo (SE) sequences which included gradient motion rephasing to reduce motion induced artefacts. T1 (500/15/3-TR in ms/TE in ms/excitations), proton density (2200–2500/15–20/1) and T2 weighted (2200–2500/80–90/1) SE images were obtained in the sagittal plain with slice thickness 3 mm, inter slice gap 0.3 mm and $220/256 \times 256$ matrix. The whole spinal cord imaging was completed in two to three examinations.

Neurophysiological investigations
Neurophysiological investigations included median and tibial somatosensory evoked potentials and motor evoked potentials to upper and lower limbs.[11] Nerve conduction studies of peroneal and sural nerve and concentric needle electromyography in a number of upper and lower limb muscles were also carried out.

Motor evoked potential (MEP) Motor evoked potentials were recorded from both upper and lower limbs bilaterally following transcranial electrical stimulation of cortex and spine. A Digitimer D-180 stimulator delivering electrical 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 the 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 the vertex to the tragus. For activating the tibialis anterior (TA) the anode was kept at the vertex, and the cathode 7 cm posterior. For cervical and lumbar stimulation the cathode was placed below the spinous process of seventh cervical (C_7) and twelfth thoracic vertebra (T_{12}) respectively and the anode proximal. Motor evoked potentials were recorded by surface electrodes, placed on ADM or TA in a belly tendon montage. During the cortical stimulation, the patient was asked to contract the target muscle slightly (10% of the maximum force irrespective of degree of weakness), whereas during the spinal stimulation, the patient was asked to relax. Electromyogram signals were filtered through 20 Hz–2 KHz at a gain of 0.5–1 mV/division. The stimulus intensity was 90% to 100% for cortical and 50% to 60% of the maximum output for spinal stimulation. Three responses were obtained at 10-s intervals and the one with the shortest latency was recorded. Onset latency and the amplitude of the negative phase were measured. Central motor conduction time was calculated for the upper limb (CMCT-ADM) by subtracting the latency on C_7 stimulation from that on cortical stimulation and that for the lower limb (CMCT-TA) by subtracting the latency on L_1 stimulation from that on vertex stimulation.[11]

Somatosensory evoked potentials (SEPs) Median SEPs were obtained by stimulating the median nerve at the wrist by a 0.1 ms square wave pulse at 3 Hz, at an intensity to produce a painless twitch of the thumb. The active surface recording electrode was placed at Erb's point and at contralateral parietal cortex 3 cm behind and 7 cm lateral to vertex using a midfrontal reference. For tibial SEP, the posterior tibial nerve was stimulated below the medial malleolus at 3 Hz, sufficient to produce a painless twitch of the great toe. The recording electrode was placed on the spinous process of the first lumbar vertebra (L_1) and 2 cm caudal to Cz (Cz′). The reference electrodes were placed at L_3 and Fz respectively. The impedence of the electrode was kept below 5 KΩ, frequency bandpass was 2–3000 Hz and analysis time 100 ms. Five hundred and twelve responses were twice averaged at a gain of 1–2 μV/division to ensure reproducibility. Median SEPs were analyzed by the latency of N_9, N_{20} and interpeak latency N_9-N_{20}. For tibial SEPs latencies of N_{21}, N_{40} and $N_{21}-N_{40}$ conduction were measured.[11]

The results of evoked potentials were compared with the normal values of our laboratory, which were obtained from 32 healthy adult volunteers. Their age ranged between 15–60 years. The upper limit of normal was defined by mean $\pm$ 2.5SD of controls. The upper limit (mean $\pm$ SD) of central motor conduction time to abductor digiti minimi ADM was 8.1 (5.1 ± 1.2) ms, central motor conduction time to tibilalis anterior (CMCT-TA) 16.1 (12.1 ± 1.6) ms, median N_9-N_{20} conduction time (CSCT) was 11.3 (8.3 ± 1.2) ms and tibial $N_{21}-N_{40}$ conduction time was 27.1 (20.1 ± 2.8).

In the follow-up studies, the change in evoked potential results was considered abnormal if it exceeded the normal intra-individual variation. To determine the normal intra-individual variation, 15 randomly selected healthy volunteers were subjected to MEP and SEP studies for 3 consecutive days and the maximum difference in CMCT and CSCT was analyzed. The mean (SE) of maximum difference for CMCT-TA was 0.79 (0.15) ms and that for tibial CSCT 0.53 (0.14) ms. The upper limit of normal intra-individual variability was defined as mean + 2.576 SE which covers 99% of normal variability.[12]

Treatment and follow-up
Twelve patients who were managed during 1992 and 1994 did not receive MPS therapy (group A). Nine patients during 1995–1997 received methyl prednisolone 500 mg i.v. slowly over 6 h for 5 days within 2 weeks of illness (group B). The clinical and evoked potential changes in these patients following MPS therapy have been reported earlier.[8] The clinical and evoked potential studies were repeated after 3 months. The outcome was defined on the basis of Barthel Index (BI) score at the end of 3 months into poor ($BI < 12$) and good ($BI \geqslant 12$).[13]

Statistical analysis
The prognostic variables such as severity of lower limb weakness, upperlimb involvement, evidence of denervation on EMG, CMCT-TA an tibial CSCT since were discrete, therefore each variable was given an arbitrary score for both group A and group B patients. The scores were added for each patient and the total score was compared between the two groups. The comparisons were also made between respective good and poor recovery subgroups of the two groups employing global test statistics.[14]

Results

Between 1992 and 1997, 24 patients with ATM were managed by us, out of which 21 have been included in the present study. Two patients had received dexamethasone and one died before neurophysiological studies could be undertaken; hence they were excluded from the present study. Twelve patients who were admitted during 1992 and 1994 did not receive MPS therapy (group A). Nine patients admitted during 1995 and 1997 received MPS therapy (group B). The clinical characteristics of these two groups are as follows:

Group A (Non MPS)
The mean age of group A patients was 33.5 years (range 16–70) and two were females. The onset to peak time of neurological deficit was 4.1 days (range 1–14). In five patients fever preceded neurological deficit. All the patients had grade 0 power in the lower limbs except one who had grade IV power. Upper limb weakness was present in four patients which ranged between grade II and IV. Horizontal level of sensory loss extended up to the lumbar region in one and the thoracic region in the remaining patients. Joint position sensation in the lower limbs was impaired in all the patients. Spinal MRI revealed abnormalities in nine out of 10 patients. Hyperintense signal changes in T2 were found in all the patients and extended from four segments to the whole spinal cord (mean 14.3 spinal segments). Electromyographic evidence of denervation in lower limb muscles were present in eight patients. Central motor conduction time to tibialis anterior (CMCT-TA) was abnormal in 11 patients (unrecordable in nine and prolonged in two). Tibial central sensory conduction time (CSCT) was abnormal in 10 patients (unrecordable in nine and prolonged in one). Upper limbs CMCT was abnormal in two patients only (unrecordable in three slides and prolonged in one) whereas median CSCT was prolonged in one patient (one side) only. At 3 months follow-up, lower limb muscle power had improved in five patients and the improvement ranged between grade I and IV. This improvement correlated with improvement in CMCT in four patients. In one patient, CMCT remained unrecordable in spite of improvement in muscle power. Joint position sensation improved in two patients although tibial CSCT improved in one patient only. In another patient, clinical sensory testing remained abnormal but CSCT improved. At 3 months follow-up, eight patients had poor and four had good recovery. The mean Barthel Index score on admission was 3.2 which improved to 9.6 at 3 months follow-up. The sensory and motor function and the outcome of untreated patients at the end of 3 months are shown in Table 1.

Group B (MPS group)
The mean age of group B patients was 25.5 years (range 12–42) and three were females. The onset to peak time of neurological deficit was 4.1 days (1–14). Preceding history of fever was present in three patients. All the patients had paraparesis, which was complete in four and partial in five. Upper limbs were weak in four patients; the weakness was mild (grade IV) in all except in one in whom it was grade II. The latter patient had respiratory paralysis necessitating artificial ventilation. Eight of these patients had a transverse level of sensory loss which was present in the dorsal region and one in whom sensory loss reached to the sixth cervical spinal level. Joint position sensation was abnormal in all the patients. Eight patients underwent an MRI study, which revealed diffuse hyperintense signal changes in T2 ranging from 2 to 17 spinal segments (mean 10). Electromyography revealed denervation potentials in lower limb muscles in four patients. Central motor conduction to TA was abnormal in eight patients; unrecordable in five (nine sides) and prolonged in three patients (six sides). Upper limb CMCT however was prolonged in two patients only (three sides).

At 3 months follow-up, the muscle power improved in all the patients. The improvement ranged between grade I and V (mean 2.3). The improvement in lower limb power correlated with CMCT-TA in eight patients. In one patient, the muscle power improved to grade III but CMCT remained unrecordable at 3 months follow-up. The motor and sensory functions and the outcome of ATM patients receiving methyl prednisolone are summarised in Table 2.

Table 1 Sequential changes in motor and sensory functions in the patients with acute transverse myelitis who did not receive methyl prednisolone therapy

Sl. No	Age/ sex	Lower limb Power I	Lower limb Power F	EMG Fibs	CMCT-TA I (R/L)	CMCT-TA F (R/L)	JPS I	JPS F	Tibial CSCT I (R/L)	Tibial CSCT F (R/L)	Outcome	BI
1	43 M	0	0	+	NR/NR	NR/NR	Ab	Ab	NR/NR	NR/NR	Poor	3/5
2	22 M	0	4	–	33.6/25.2	25.2/14.4	Ab	Ab	18.0/15.0	18.0/15.0	Good	2/19
3	48 M	0	0	+	NR/NR	NR/NR	Ab	Ab	NR/NR	NR/NR	Poor	2/7
4	57 M	0	0	+	NR/NR	NR/NR	Ab	Ab	NR/NR	NR/NR	Poor	6/7
5	15 M	0	0	+	NR/NR	NR/NR	Ab	Ab	NR/NR	NR/NR	Poor	0/7
6	40 M	0	4	–	19.2/18.8	18.8/16.4	Ab	Ab	23.0/32.0	25.0/24.0	Good	3/20
7	16 M	0	0	+	NR/NR	NR/NR	Ab	Ab	NR/NR	NR/NR	Poor	3/3
8	20 M	0	0	+	NR/NR	NR/NR	Ab	Ab	NR/NR	NR/NR	Poor	3/3
9	14 M	0	4	–	NR/NR	16.4/16.0	Ab	N	NR/NR	26.0/28.0	Good	3/18
10	45 M	0	3	+	NR/NR	NR/NR	Ab	Ab	NR/NR	NR/NR	Poor	3/3
11	70 M	0	0	+	NR/NR	NR/NR	Ab	Ab	NR/NR	NR/NR	Poor	3/3
12	31 F	0	5	–	14.2/13.2	14.4/14.0	Ab	N	23.0/25.6	22.4/25.0	Good	7/20

I = initial, JPS = joint position sense, Ab = abnormal, N = normal, F = final (3 months), NR = not recordable, BI = Barthel index

Table 2 Sequential changes in motor and sensory functions in the patient with acute transverse myelitis receiving methyl prednisolone therapy

Sl. No	Age/ sex	Lower limb Power I	Lower limb Power F	EMG Fibs	CMCT-TA I (R/L)	CMCT-TA F (R/L)	JPS I	JPS F	Tibial CSCT I (R/L)	Tibial CSCT F (R/L)	Outcome	BI
1	20 M	4	5	–	14.0/13.6	12.0/12.6	Ab	Ab	24.4/23.2	20.0/20.4	Good	20/20
2	29 F	0	2	+	NR/NR	NR/14.0	Ab	Ab	NR/NR	NR/NR	Poor	1/4
3	30 M	3	5	–	36.4/36.0	25.6/27.6	Ab	Ab	NR/35.0	NR/24.4	Good	10/20
4	30 M	0	5	+	NR/NR	14.0/17.2	Ab	N	24.0/31.2	19.6/19.8	Good	8/20
5	42 F	0	2	+	NR/NR	13.6/NR	Ab	N	17.6/18.2	15.6/15.6	Poor	4/4
6	12 M	2	4	–	14.4/NR	12.8/14.8	Ab	Ab	NR/NR	NR/NR	Good	4/19
7	35 M	0	3	+	NR/NR	NR/NR	Ab	Ab	NR/NR	NR/NR	Poor	0/4
8	20 F	4	5	–	18.0/20.0	12.4/17.6	Ab	N	NR/19.2	21.2/22.4	Good	11/20
9	12 M	2	5	–	32.0/24.0	9.6/8.8	Ab	N	21.4/22.0	18.0/18.8	Good	5/20

I = initial, JPS = joint position sense, Ab = abnormal, N = normal, F = final (3 months), NR = not recordable, BI = Barthel index

Comparison of prognostic variables in MPS group and non MPS group

In the present study, 67% patients in the non MPS group (group A) had a poor outcome compared to 33% receiving methyl prednisolone. In the non MPS group, eight out of 11 patients (73%) and in the MPS group, three out of four patients (75%) with complete paraplegia had a poor outcome. All the patients with partial lower limb weakness in the non MPS group (one patient) and the MPS group (five patients) had a good recovery. The upper limbs were involved in one patient in the control group who had a poor recovery. All patients in the non MPS and MPS groups who had evidence of denervation on EMG had a poor outcome except one patient in the MPS group (no 4) who had a good outcome. CMCT-TA if unrecordable was associated with a poor outcome. Eight out of nine patients in the non MPS group and three out of four patients in the MPS group with unrecordable CMCT-TA had a poor recovery. Similarly unrecordable tibial CSCT was associated with a poor outcome. Eight out of nine patients in the non MPS group and two out of three in the MPS group with unrecordable tibial CSCT had a poor outcome.

Global test statistics did not reveal a beneficial effect of methyl prednisolone in the outcome of ATM. There was no difference in the global scores of controls and the study group ($Z = 1.51$, NS). Similarly in the good recovery subgroup the score was higher in the MPS group compared to the control group which also did not achieve statistical significance ($t = 1.93$, NS).

Discussion

In our study, 67% patients in the control group had a poor outcome compared to 33% receiving methyl prednisolone. These results apparently suggest a beneficial role of methyl prednisolone in ATM. Methyl prednisolone has been reported to be beneficial in two out of three patients with ATM in an earlier study. In this study, the clinical details were provided about one patient only who had partial weakness and minimal posterior column impairment. This patient dramatically improved 48 h after methyl prednisolone therapy.[2] In a recent study, five children

with severe ATM treated with i.v. methyl prednisolone were compared with a historical group of 10 patients. There was significant improvement in the treated group as evidenced by reduced time for independent walking and a higher proportion of patients with full recovery within 12 months.[3] In this study the extent of spinal cord involvement was not evaluated by evoked potential or EMG studies. In another study, three patients with recurrent acute transverse myelopathy, responded well to corticosteroid therapy initially.[15] These patients however represent a different disorder compared to our patients. A beneficial effect of prednisolone and methyl prednisolone in lupus myelitis has been reported.[16,17] None of our patients however had features of systemic lupus erythematosus. In our study, the methyl prednisolone was not found to be beneficial. In the non MPS group, eight out of 11 patients with complete paraplegia (73%) and in the MPS group three out of four (75%) had a poor outcome. All the patients having EMG evidence of denervation also had a poor outcome irrespective of methyl prednisolone therapy except one in the MPS group who recovered well. Eight out of nine patients with unrecordable MEP and SEP in the non MPS group and three out of four patients in the MPS group had poor recovery, suggesting a lack of beneficial effect of a methyl prednisolone in ATM. In an earlier study also, a patient with necrotic myelitis did not improve in spite of corticosteroids.[4]

Histologically the changes in ATM are variable and extend both horizontally and vertically. The lesion may be confined to the white matter which usually shows patchy demyelination along the course of veins. Perivenous lymphocytic and plasma cell infiltration are seen. Lesions are more common in ventrolateral compared to the dorsal white matter which may account for the more frequent MEP abnormalities in our study. The neurons may appear normal or there may be necrosis of not only neurons but of the spinal cord as a whole.[18] Oedema and demyelination are likely to have a better outcome compared to necrotic changes. White matter changes in spinal cord can be assessed by evoked potentials and grey matter by EMG. Prolongation of CMCT and CSCT may be due to demyelination and oedema which results in dispersion of descending volleys.[12] Severe demyelination, however, may result in conduction block and unrecordable evoked potentials. Axonal damage can also result in unrecordable evoked potentials. The damage to anterior horn cells results in evidence of denervation on EMG. The presence of denervation on EMG has been associated with poor prognosis.[19] Bilaterally unrecordable evoked potentials and evidence of denervation on EMG may suggest a more severe illness compared to the patients who have recordable evoked potentials and normal EMG. In the present study, patients with recordable CMCT-TA, tibial SEP and absence of denervation fared better compared to the remaining patients. If the comparisons are made with severely and mildly affected subgroups of the control group with corresponding treatment groups, there was no beneficial effect of methyl prednisolone. Our results raise doubts about the efficacy of methyl prednisolone in ATM. The failure of methyl prednisolone to show any benefit may well have been due to the fact that two groups of patients were by no means comparable in terms of severity of their disease. Corticosteroids may help in reducing oedema by their anti-inflammatory action especially in demyelinating diseases. In multiple sclerosis, methyl prednisolone helps in shortening the acute exacerbation, although long-term prognosis is not influenced.[5,20] We have not been able to assess the speed of recovery between the two groups as their outcome was determined at the end of 3 months. Long-term outcome of these patients depends on remyelination, axonal regeneration, sprouting of collaterals and neuronal plasticity. From this study it can be concluded that methyl prednisolone may not alter the prognosis of ATM as assessed by 3 months Barthel index score.

Our results are based on a relatively small sample size. A larger study classifying patients into poor and good prognosis groups may be warranted to clarify the role of corticosteroids in ATM.

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