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Original article

Cognitive Changes After Methylprednisolone Pulse Therapy in Relapsing-Remitting Multiple Sclerosis: Association with Cerebrospinal Fluid Biochemical Parameters

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Abstract

Cognitive decline severely impacts the quality of life in patients with relapsing-remitting multiple sclerosis, yet the specific effects of methylprednisolone pulse therapy on cognitive performance remain unclear.

The objective of this study was to evaluate cognitive changes following methylprednisolone pulse therapy in patients with relapsing-remitting multiple sclerosis and to assess cerebrospinal fluid biochemical parameters as predictors of these changes.

Methods. Forty-one patients with relapsing-remitting multiple sclerosis and twenty-seven healthy controls were examined. Patients were assessed before, immediately after, and three months following methylprednisolone pulse therapy; controls were assessed at comparable intervals without treatment. Cognitive assessment included tests of global cognition, information processing speed, visuospatial memory, verbal fluency, and executive function. Cerebrospinal fluid analyses included protein, glucose, lymphocytes, neutrophils, total cell count, and oligoclonal immunoglobulin G synthesis pattern.

Results. Patients showed immediate improvement in visuoconstructive abilities (χ^2 = 8.07, p = 0.005) and executive functions (χ^2 = 10.5, p = 0.005), followed by improvement in visuospatial memory after three months (χ^2 = 10.9, p = 0.004). Improvement in visuoconstruction and absence of progress in information processing speed distinguished patients from controls (χ^2 = 11.6, p < 0.001). Cerebrospinal fluid parameters and the number of previous methylprednisolone courses explained 42–46% of visuospatial memory variance, 71% of executive function variance, and 35% of global cognitive performance variance.

Conclusions. Methylprednisolone pulse therapy was associated with improvements in visuoconstructive and executive functions in relapsing-remitting multiple sclerosis. Cerebrospinal fluid biochemical parameters significantly predicted post-treatment cognitive dynamics.

Keywords: multiple sclerosis, relapsing-remitting, methylprednisolone, cerebrospinal fluid, cognitive dysfunction, biomarkers, neuropsychology.

1. Introduction

Multiple sclerosis (MS) is an autoimmune demyelinating and neurodegenerative disease of the central nervous system and the leading cause of non-traumatic neurological disability in young adults [1]. Over recent decades, the prevalence of MS has been increasing, and currently, about 2.5 million people worldwide live with this condition [2].

The disease typically manifests between the ages of 20 and 40. In women, MS tends to appear earlier than in men, and women are affected more frequently, with a sex ratio of approximately 3:1 [3]. The etiology of MS is multifactorial: risk increases due to a combination of genetic predisposition and environmental factors such as smoking, vitamin B and D deficiency, residence in northern latitudes, and viral infections [4].

The pathological processes in MS are defined by four key features: inflammation, demyelination, reactive gliosis, and neuroaxonal damage. During relapses, foci of inflammatory demyelination form in the brain and spinal cord [5]. Typical lesion locations include juxtacortical, infratentorial, periventricular regions, and the spinal cord [6].

Cognitive impairment occurs in up to 93% of MS patients at both early and late stages [7]. The variability in prevalence stems from differences in psychometric instruments and the heterogeneity of cognitive profiles across MS subtypes and disease stages. Mild cognitive dysfunction can appear early but more commonly it develops within the first five years [8]. Cognitive decline affects multiple domains: learning and memory (up to 80%), praxis and gnosis (up to 30%), attention (up to 70%), language (up to 58%), executive functions (up to 80%), and social cognition (up to 66%) [9].

Intravenous methylprednisolone (a synthetic corticosteroid) is widely used to treat MS relapses [10]. Corticosteroids are powerful modulators of higher mental functions [11]. According to Spannenburg L.V., corticosteroid use can reduce verbal memory and visuospatial abilities [12]. However, earlier studies have also reported improved memory performance following corticosteroid administration [13,14].

A meta-analysis of corticosteroid effects on cognition divided patients into three groups: chronic users, short-term users, and current users. Corticosteroids had no effect on attention span or attention stability in any group, but negatively affected executive functions in current users. Short-term and chronic users demonstrated impaired short-term memory, and long-term memory declined during active treatment. No significant effects were found on processing speed, and neither dose nor age influenced cognitive change [15].

When the central nervous system is affected, the composition and concentration of CSF proteins change, making the CSF profile a potential predictor of neurocognitive dysfunction [16]. Gaetani et al. reported a lack of studies linking CSF biomarkers with neuropsychological manifestations in MS. Their systematization suggested that specific CSF biomarkers correspond to distinct cognitive deficits. For example, neurofilament light chain (NfL) correlates with lower information processing speed, verbal fluency, and visuospatial memory, while beta-amyloid peptide shows positive associations with PASAT and SDMT performance [17].

Other investigations of CSF biomarkers also linked YKL-40 glycoprotein to executive dysfunction [18], and stated that interleukin-6 (IL-6) correlates negatively with global cognitive performance [19]. Cognitive deficits have been linked to immune dysregulation [20]. Experimental work by Ziv et al. demonstrated that peripheral T cells play a role in hippocampal neurogenesis and spatial learning [21]. Grauer et al. found that the severity of cognitive impairment was associated with elevated CSF lymphocyte counts [22].

In addition to proteins and lymphocytes, researchers have identified other CSF biomarkers associated with cognitive functioning. Tigchelaar et al. reported a negative correlation between MoCA test scores and cerebrospinal fluid glucose concentration ($\rho = -0.553$) [23]. In patients with neurosyphilis, CSF pleocytosis was found to be associated with more severe cognitive impairment [24].

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Another characteristic marker of multiple sclerosis—and therefore of the disease-specific cognitive disturbances—is the pattern of oligoclonal immunoglobulin G (IgG) synthesis. The most typical pattern for MS is type 2 (intrathecal synthesis), where IgG is present in the CSF but absent in blood plasma [25].

An intriguing question is whether the effect of methylprednisolone pulse therapy on cognition in MS patients can be predicted. In 2002, F. Sellebjerg et al. attempted to predict treatment response using nitric oxide concentrations in CSF. They found that patients with higher CSF nitric oxide levels exhibited better clinical response to methylprednisolone therapy [26].

Similarly, Whitaker et al. demonstrated that myelin basic protein (MBP) could serve as a predictor of response to methylprednisolone [27]. However, Milanese et al., studying both methylprednisolone and dexamethasone pulse therapy, concluded that these treatments did not significantly alter CSF composition [28].

The objective of the present study is to investigate cognitive changes in patients with multiple sclerosis following methylprednisolone pulse therapy and to evaluate the predictive capacity of CSF biochemical composition with respect to cognitive impairment in MS.

2. Materials and methods

Sample and Study Design

The study was conducted between February 2024 and September 2025 at the A.Ya. Kozhevnikov Clinic of Nervous Diseases (Moscow, Russia). The research protocol was approved by the Local Ethics Committee of Sechenov University (Approval No. 19-24). All participants provided written informed consent.

Patients included in the study had no comorbid psychiatric disorders and did not receive psychotropic medications during participation. The sample comprised 41 patients with multiple sclerosis (MS) during relapse: 2 with primary progressive MS, 2 with secondary progressive MS, and 37 with relapsing–remitting MS. The diagnosis was established according to the 2017 McDonald criteria.

The control group (CG) consisted of 27 neurologically and psychiatrically healthy individuals who had never undergone methylprednisolone pulse therapy. All participants of the CG also provided written informed consent.

Data sharing statement: This study did not involve a clinical trial. De-identified data are available from the corresponding author upon reasonable request.

Sociodemographic characteristics of both groups are shown in Table 1.

Table 1 - Sociodemographic characteristics of Patient and Control groups

Variable	**MS Patients**				**Control Group**			
	N	M	Me	SD	N	M	Me	SD
Age (years)	41	36.7	34	11.1	27	40,6	44	10.9
Education (years)	40	15.7	16	2.79	27	15.9	15	3.11
Disease duration (years)	41	6.34	4	7.45	-	-	-	-
Number of prior pulse therapies	41	1.59	1	2.39	27	0	0	0
EDSS score	39	2.26	2	1.61	0	0	0	0
Sex	**N**		**%**		**N**		**%**	
Male	18		43.9		9		33.3	
Female	23		56.1		18		66.7	

Note. N – number of participants; M – mean value; Me – median; SD – standard deviation

Cognitive and affective assessments were conducted before methylprednisolone pulse therapy (1000 mg intravenously for 5–7 days), immediately after, and 3 months post-therapy. Participants in the control group were assessed after 5–7 days in which they did not receive any specific intervention and after 3 months. The study design is illustrated in Figure 1.

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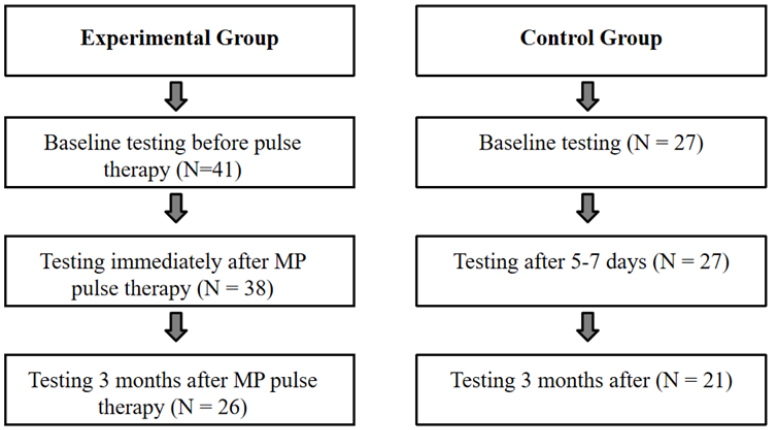

Figure 1 - Study design

Of the 41 MS patients, two withdrew from the study after therapy, and one did not receive MP treatment. Three months later, 14 patients in experimental group and 6 control participants did not respond or declined follow-up participation.

No statistically significant differences were found between groups in sex, age, or education level at any stage (before, immediately after, or 3 months following pulse therapy). Comparison of control and patient groups at each study stage is shown in Table 2.

Table 2 - Comparison of control and patient groups at each study stage

Group	Sex		Age		Education	
	χ2	p	U	p	U	p
Before MP pulse therapy	0.760	0.383	440	0.155	529	0.887
Immediately after MP pulse therapy	0.513	0.474	415	0.194	486	0.853
3 months after MP pulse therapy	0.396	0.529	232	0.380	234	0.407

CSF Analysis

CSF collection was performed prior to MP pulse therapy. Samples (3–5 ml each) were collected into three sequentially labeled sterile tubes and transported to the laboratory within 30 minutes.

Protein concentration was measured by the colorimetric method (pyrogallol red). Glucose was determined by enzymatic glucose oxidase assay. Lymphocytes, neutrophils, and total cell count were assessed by microscopy after centrifugation (azure–eosin staining) using a Fuchs–Rosenthal chamber. The pattern of oligoclonal IgG synthesis was determined by isoelectric focusing.

Cognitive Assessment

For the integral assessment of cognitive functions, the Montreal Cognitive Assessment (MoCA test) was used [29]. To evaluate the rate of cognitive processing and attention, the Symbol Digit Modalities Test (SDMT) was applied [30]. To assess short-term and long-term visuospatial memory, the Brief Visuospatial Memory Test–Revised (BVMT-R) was used [31]. To assess the rate of psychomotor processing and semantic memory, a variant of the Controlled Oral Word Association Test (COWAT) was used [32]. To assess executive functions, the Stroop Color–Word Test was administered [33].

Statistical Analysis

Statistical processing included descriptive statistics, Mann–Whitney U-test, Wilcoxon W-test, Friedman χ^2 with Durbin–Conover post-hoc tests, and General Linear Models (GLM). All analyses were performed using Jamovi v 2.7.7.0.

This observational study was conducted in accordance with the STROBE reporting guidelines.

3. Results

Table 3 presents the results of within-group changes in cognitive functions among patients with demyelinating diseases. Only statistically significant differences are shown.

Significant effects were observed in the BVMT-R (first and second trials and total score for three trials), Stroop test (reading time for the third card), and the visuoconstructive subtest of the MoCA test. Pairwise were conducted using the Durbin–Conover method.

Table 3 - Within-group changes in the patient group across cognitive tests (only significant results)

Groups	Durbin–Conover (Statistic)	p	M
BVMT-R (first trial) $\chi^2 = 6.33$, p = 0.042			
Before-After	0.335	0.739	Before = 5
Before – 3 months after	2.430	0.019	After = 4.88
After- 3 months after	2.094	0.041	3 months after = 6.62
BVMT-R (second trial) $\chi^2 = 9.19$, p = 0.010			
Before-After	0.509	0.613	Before = 7.92
Before – 3 months after	2.547	0.014	After = 7.46
After- 3 months after	3.057	0.004	3 months after = 9.27
BVMT-R (sum of three trials) $\chi^2 = 10.9$, p = 0.004			
Before-After	0.520	0.520	Before = 22.5
Before – 3 months after	2.859	0.006	After = 21.6
After- 3 months after	3.379	0.001	3 months after= 26.1
Stroop Test (reading time for 3rd card with color–word interference, seconds) $\chi^2 = 10.5$, p = 0.005			
Before-After	3.443	0.001	Before = 34.4
Before – 3 months after	2.525	0.015	After = 30.5
After- 3 months after	0.918	0.363	3 months after= 31.8
Visuoconstructive abilities (MoCA test) $\chi^2 = 8.07$, p = 0.005			
Before-After	3.18	0.003	Before = 4.36, After = 4.72

Three months after methylprednisolone pulse therapy, patients demonstrated improvement in short-term visuospatial memory compared with results obtained before and immediately after treatment. Improvement was also observed in executive functions, reflected by reduced reading time on the third Stroop card, as well as higher scores in the visuoconstructive subtest of the MoCA test immediately after therapy.

In the control group, differences were observed for the following cognitive tests: SDMT (total correct responses and errors), BVMT-R (first trial), and Stroop test (third card). Results are shown in Table 4.

Table 4 - Within-group changes in the control group across cognitive tests (only significant results)

Groups	Durbin–Conover (Statistic)	p	M
SDMT (number of correct symbol–digit matches) $\chi^2 = 11.6$, p < 0.001			
Before - After	4.41	<.001	Before = 49.1, After = 53.3
SDMT (errors) $\chi^2 = 7.20$, p = 0.007			
Before - After	3.07	0.005	Before = 1.9, After = 0.6
BVMT-R (first trial) $\chi^2 = 6.11$, p = 0.047			
Before - After	0.089	0.930	Before = 5.9
Before – 3 months after	2.311	0.026	After = 5.7
After – 3 months after	2.222	0.032	3 months after = 7.15
Stroop test (reading time for 1st card with black words) $\chi^2 = 6.02$, p = 0.049			

Table 4 (Continuation) - Within-group changes in the control group across cognitive tests (only significant results)

Groups	Durbin–Conover (Statistic)	p	M
Before - After	1.647	0.107	Before = 13.5
Before – 3 months after	0.906	0.371	After = 12.8
After – 3 months after	2.552	0.015	3 months after = 13.9
Stroop test (reading time for 3rd card with color–word interference, seconds) $\chi^2 = 12.2$, p = 0.002			
Before - After	3.888	<.001	Before = 34.9
Before – 3 months after	2.894	0.006	After = 29.2
After – 3 months after	0.995	0.326	3 months after = 30.2

In the control group, after only five days, improvements were observed in processing speed (increased SDMT scores and decreased errors) and executive functions. Similar to the experimental group, visuospatial memory improved after three months compared with both earlier measurements.

Further analysis compared results between the control and experimental groups before, immediately after, and three months after therapy.

At baseline, both groups differed only in the visuoconstructive subtest of the MoCA test ($M_{experimental}$ = 4.3, $M_{control}$ = 4.93, U=322, p<0.001) and in the total MoCA score ($M_{experimental}$ = 25.52, $M_{control}$ = 27.19, U=380, p=0.040).

At the second time point (five days for the control group and 5–7 days after pulse therapy for the experimental group), differences were observed in the SDMT test ($M_{experimental}$ = 45.14, $M_{control}$ = 53.26, U=267, p=0.002); the control group showed faster cognitive processing.

At the third time point (3 months after), there were no significant differences between the groups in any cognitive tests. It should be noted that the SDMT and the visuoconstructive MoCA subtest were not conducted at this stage because the follow-up assessment was performed remotely (online).

General Linear Models (GLM) were used to identify predictors of changes in cognitive performance. Predictors included: CSF protein concentration (g/L), total cell count (cells/mm³), glucose concentration (mmol/L), lymphocytes (cells/mm³), neutrophils (cells/mm³), number of previous pulse therapies, and oligoclonal IgG synthesis type. Dependent variables were calculated as the difference in cognitive test results before and immediately after pulse therapy. Table 5 shows the GLM results for BVMT-R scores as dependent variables.

Table 5 - General linear models (GLM) for BVMT-R changes before and after methylprednisolone pulse therapy

Dependent vari-able	BVMT-R (2nd trial)			BVMT-R (3rd trial)			BVMT-R (sum of 1st three trials)			BVMT-R (25 minutes after)		
Predictor	**F**	**η^2p**	**β**	**F**	**η^2p**	**β**	**F**	**η^2p**	**β**	**F**	**η^2p**	**β**
Model	4,6*	0,45	-	4,5*	0,46	-	4,0*	0,42	-	3,1*	0,45	-
Protein (g/L)	3,6	0,17	-0,35	7,1*	0,31	-0,51	5,7*	0,25	-0,46	6,1*	0,29	-0,50
Total cell count (cells/mm³)	-	-	-	3,8	0,19	0,39	-	-	-	3,7	0,20	0,40
Number of previous pulse therapies	5,2*	0,23	-0,44	-	-	-	1,5	0,08	-0,24	1,6	0,10	-0,27
Oligoclonal IgG synthesis type (2-1)	7,8*	0,31	-1,11	4,6*	0,23	-0,87	5,1*	0,23	-0,92	4,5	0,23	-0,92

*Note. * – p < 0.05, ** – p < 0.001*

Short-term visuospatial memory was predicted by approximately 50% of the variance explained by the number of previous pulse therapies, the type of oligoclonal IgG synthesis, and total CSF protein concentration. A greater number of previous pulse therapies (equal to the number of relapses) was negatively associated with improvement in short-term visuospatial memory following the new pulse therapy.

The second (intrathecal) type of oligoclonal IgG synthesis was also negatively associated with short-term visuospatial memory changes. For long-term visuospatial memory, total CSF protein concentration emerged as a significant predictor: higher protein levels were negatively associated with changes in long-term visuospatial memory.

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No model could be established for the difference between BVMT-R first-trial scores before and after therapy. Glucose, lymphocytes, and neutrophils in CSF were not significant predictors in any model.

Table 6 presents the GLM results for Stroop test (2nd card reading time), COWAT scores, and total MoCA scores as dependent variables.

Table 6 - General linear models (GLM) for Stroop Test and MoCA Test differences before and after methylprednisolone pulse therapy

Dependent vari-able	Stroop Test (2nd card, seconds)			MoCA Test		
Predictor	F	η^2p	β	F	η^2p	β
Model	3,6*	0,71	-	5,6*	0,35	-
Protein (g/L)	2,9	0,23	0,34	10,4*	0,33	-0,57
Total cell count (cells/mm³)	11,0*	0,53	-6,72	-	-	-
Glucose (mmol/L)	1,0	0,09	-0,23	-	-	-
Lymphocytes (cells/mm³)	12,6*	0,56	6,81	1,2	0,06	-0,20
Neutrophils (cells/mm³)	17,8*	0,64	1,56	-	-	-
Number of previous pulse therapies	0,0	0,00	-0,03	-	-	-
Oligoclonal IgG synthesis type (2-1)	0,5	0,05	-0,34	-	-	-

*Note. * – $p < 0.05$, ** – $p < 0.001$*

More than 70% of the variance in Stroop test second-card performance was explained by CSF cytosis, lymphocyte, and neutrophil counts. Lymphocyte and neutrophil concentrations showed positive associations with Stroop completion time, indicating poorer executive functioning with higher counts. In contrast, pleocytosis was negatively associated with Stroop time differences, meaning higher pleocytosis corresponded to reduced completion time after therapy. The total MoCA score was negatively associated with CSF protein concentration.

No significant models were obtained for SDMT, COWAT, or other Stroop test cards.

4. Discussion

This study examined changes in cognitive functioning in patients with multiple sclerosis after methylprednisolone pulse therapy compared with a control group. It also investigated the predictive role of cerebrospinal fluid components in relation to cognitive function dynamics following MP therapy.

Significant differences in the experimental group were observed in BVMT-R scores for the first and second trials, as well as in the total BVMT-R score. These differences appeared in the results obtained three months after therapy compared with both pre- and post-therapy data. No significant differences were found between the pre- and post-therapy stages. This finding is consistent with the German study by Naber et al., which, using the Benton Visual Retention Test, also reported no significant differences before and immediately after methylprednisolone pulse therapy [34]. This effect may be explained by the counteracting influence of two factors that potentially reduce visuospatial ability—the relapse state itself and the impact of pulse therapy.

It is also noteworthy that the control group showed a significant improvement in BVMT-R first-trial scores after three months, which may indicate a general practice or learning effect, although the specific stimulus materials differed across all three stages in both groups. The decrease in reading time for the third Stroop card was similar in both groups, suggesting a nonspecific improvement due to task familiarity rather than treatment effects.

Visuoconstructive abilities improved immediately after pulse therapy. This result aligns with the findings of Castagna et al., who reported significant improvement in MoCA visuoconstructive subtest scores following corticosteroid therapy [35]. This effect can be explained by the high concentration of corticosteroid receptors in the prefrontal cortex, which is responsible for executive functions [36,37].

An interesting finding was the improvement in SDMT performance and reduction in errors observed in the control group. The experimental group showed no significant changes. Moreover, after pulse therapy, the MS group had significantly lower SDMT scores than the control group. It is possible that patients with MS exhibit reduced learning ability and slowed cognitive processing [38], which may explain the absence of improvement within 5–7 days compared with healthy participants in the control group.

CSF components emerged as significant predictors of visuospatial memory changes in MS patients after pulse therapy. High CSF protein levels, an intrathecal IgG synthesis pattern, and a high number of previous pulse therapies explained up to 46% of the variance in BVMT-R score dynamics. The

combination of intrathecal IgG synthesis and elevated CSF protein concentration indicates active inflammatory processes in the CNS [39]. A high number of prior pulse therapies reflects more frequent relapses and a more aggressive disease course. Together, these three factors point to the severity of inflammation and serve as unfavorable predictors of visuospatial memory recovery after new pulse therapy.

Neutrophil and lymphocyte counts in CSF were significant predictors of poorer performance on the second Stroop card (color naming) after MP therapy. Conversely, total cytosis showed a positive relationship—higher cytosis predicted shorter completion time after therapy. This may be explained by the idea that lymphocytes and neutrophils indicate deeper or more specific CNS damage [39] that is not mitigated by methylprednisolone therapy, while cytosis, as a more general inflammatory marker, decreases after treatment and is associated with improved cognitive processing speed.

5. Conclusion

Three months after pulse therapy, patients with multiple sclerosis demonstrated improvement in short-term visuospatial memory and executive functions. However, similar findings in the control group indicate that these changes are not specific to methylprednisolone pulse therapy. The only effect characteristic of MS patients was observed in the visuospatial subtest of the MoCA test: a significant improvement appeared immediately after treatment.

It is important to note that, unlike the control group, MS patients did not show improvement in processing speed (SDMT scores) following pulse therapy.

The study demonstrated the pattern of cognitive function changes in MS patients over time compared with the control group.

1. Before pulse therapy, MS patients exhibited lower total MoCA scores and lower visuoconstructive subtest scores.
2. After pulse therapy, MS patients demonstrated slower cognitive processing speed.
3. Three months after therapy, differences in cognitive functions between groups were no longer present.

The study also demonstrated the predictive potential of CSF biochemical parameters for various cognitive tests, explaining 35–71% of the variance. Negative predictors of cognitive functioning included CSF protein concentration, lymphocyte and neutrophil counts, type 2 (intrathecal) pattern of oligoclonal IgG synthesis, and the number of previous pulse therapies (relapses). Pleocytosis was identified as a positive predictor.

Conflicts of Interest. The authors declare no conflict of interest.

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Ethical Approval Statement. The research protocol was approved by the Local Ethics Committee of Sechenov University (Approval No. 19-24 of 24.07.2024). All participants provided written informed consent.

AI Declaration. The authors confirm that no generative AI or AI-assisted technologies were used in the preparation of this manuscript.

Author Contributions. Conceptualization, V.G.; Methodology, V.G.; Formal analysis, V.G., A.K., E.I., N.B.; Writing – original draft, V.G., K.K.; Writing – review & editing, V.G., N.B.; Supervision, V.G.; Project Administration, V.G.; Investigation, A.K., E.I., K.K., N.B.; Visualization, A.K.; Data curation, E.I., N.B.

All authors have read and approved the final version of the manuscript and have signed the copyright transfer form.

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Ремиттивтік шашыраңқы склероз кезіндегі метилпреднизолонмен пульс-терапиядан кейінгі когнитивті өзгерістер: Жұлын-ми сұйықтығының биохимиялық көрсеткіштерімен байланысы

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Түйіндеме

Зерттеудің мақсаты: ремиттивтік шашыраңқы склерозы бар науқастарда метилпреднизолонмен пульс-терапиядан кейінгі когнитивті өзгерістерді бағалау және осы өзгерістерді болжауда жұлын-ми сұйықтығының биохимиялық көрсеткіштерінің маңызын анықтау.

Әдістері. Ремиттивтік шашыраңқы склерозы бар 41 науқас және бақылау тобындағы 27 дені сау адам зерттелді. Науқастар метилпреднизолонмен пульс-терапияға дейін, ем аяқталғаннан кейін бірден және үш айдан кейін тексерілді. Бақылау тобы ем қабылдамай, сәйкес уақыт аралықтарында тексерілді. Когнитивті бағалау жалпы когнитивті жағдайды, ақпаратты өңдеу жылдамдығын, көру-кеңістіктік жадты, вербалды еркін сөйлеуді және атқарушы функцияларды бағалауды қамтыды. Жұлын-ми сұйықтығында ақуыз, глюкоза, лимфоциттер, нейтрофилдер, жасушалардың жалпы саны және G иммуноглобулинінің олигоклоналды синтезінің типі анықталды.

Нәтижелері. Науқастарда көру-конструктивтік қабілеттердің (χ^2 = 8,07; p = 0,005) және атқарушы функциялардың (χ^2 = 10,5; p = 0,005) емнен кейін бірден жақсаруы, ал 3 айдан кейін көру-кеңістіктік жадтың жақсаруы (χ^2 = 10,9; p = 0,004) байқалды. Көру-конструктивтік қабілеттердің жақсаруы және ақпаратты өңдеу жылдамдығында оң динамиканың болмауынауқастарды бақылау тобынан ерекшелендірді (χ^2 = 11,6; p < 0,001). Жұлын-ми сұйықтығының көрсеткіштері және метилпреднизолонмен бұрын жүргізілген ем курстарының саны көру-кеңістіктік жад вариабельділігінің 42-46%-ын, атқарушы функциялар вариабельділігінің 71%-ын және жалпы когнитивті көрсеткіштер вариабельділігінің 35%-ын түсіндірді.

Қорытынды. Метилпреднизолонмен пульс-терапия ремиттивтік шашыраңқы склероз кезінде көру-конструктивтік және атқарушы функциялардың жақсаруымен байланысты болды. Жұлын-ми сұйықтығының биохимиялық көрсеткіштері емнен кейінгі когнитивті динамиканы статистикалық мәнді түрде болжады.

Түйін сөздер: ремиттивтік шашыраңқы склероз, метилпреднизолон, жұлын-ми сұйықтығы, когнитивті бұзылулар, биомаркерлер, нейропсихология.

Когнитивные изменения после пульс-терапии метилпреднизолоном при ремиттирующем рассеянном склерозе: Связь с биохимическими показателями спинномозговой жидкости

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Резюме

Цель исследования: оценить изменения когнитивных функций после пульс-терапии метилпреднизолоном у пациентов с ремиттирующим рассеянным склерозом и определить прогностическую значимость биохимических показателей спинномозговой жидкости в отношении этих изменений.

Методы. Обследованы 41 пациент с ремиттирующим рассеянным склерозом и 27 здоровых лиц контрольной группы. Пациентов обследовали до пульс-терапии метилпреднизолоном, непосредственно после нее и через три месяца. Контрольную группу обследовали через сопоставимые интервалы времени без проведения терапии. Когнитивное обследование включало оценку общего когнитивного статуса, скорости обработки информации, зрительно-пространственной памяти, вербальной беглости и исполнительных функций. В спинномозговой жидкости определяли содержание белка и глюкозы, количество лимфоцитов, нейтрофилов и общее количество клеток, а также тип олигоклонального синтеза иммуноглобулина G.

Результаты. У пациентов отмечалось непосредственное улучшение зрительно-конструктивных способностей ($\chi^2 = 8,07$; $p = 0,005$) и исполнительных функций ($\chi^2 = 10,5$; $p = 0,005$), а через три месяца — улучшение зрительно-пространственной памяти ($\chi^2 = 10,9$; $p = 0,004$). Улучшение зрительно-конструктивных способностей и отсутствие положительной динамики скорости обработки информации отличали пациентов от контрольной группы ($\chi^2 = 11,6$; $p < 0,001$). Показатели спинномозговой жидкости и количество предшествующих курсов метилпреднизолона объясняли 42–46% вариабельности зрительно-пространственной памяти, 71% вариабельности исполнительных функций и 35% вариабельности общего когнитивного статуса.

Выводы. Пульс-терапия метилпреднизолоном была ассоциирована с улучшением зрительно-конструктивных и исполнительных функций при ремиттирующем рассеянном склерозе. Биохимические показатели спинномозговой жидкости значимо прогнозировали когнитивную динамику после лечения.

Ключевые слова: ремиттирующий рассеянный склероз, метилпреднизолон, спинномозговая жидкость, когнитивная дисфункция, биомаркеры, нейропсихология.