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Research Article

# Therapeutic Potential of Biocompatible Nanodevices in Multiple Sclerosis: Results of a Clinical Study

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## Abstract

Multiple sclerosis (MS) is a significant neurological disorder due to its high prevalence, chronic progression, frequent development of disability, and predominant onset in young individuals. The pathogenesis of MS is primarily explained by the immunopathogenesis hypothesis. Biocompatible magnetite nanoparticles, known for their selective sorption of cell membrane surface proteins, circulating immune complexes, lymphocytotoxin antibodies, and complement system components, have demonstrated potential for immunoreaction. Additionally, these nanoparticles enhance phagocytic activity and improve the leukocyte phagocytosis index, making them promising candidates for MS therapy. The primary objective of this study is to slow MS progression, improve neurological function and overall patient well-being, and mitigate the expansion of demyelinating lesions in the brain.

**Materials and Methods:** The study included a patient diagnosed with secondary progressive multiple sclerosis in its cerebrospinal form during a phase of clinical exacerbation. Neurological status and disability levels were assessed using the Expanded Disability Status Scale (EDSS), and contrast-enhanced brain MRI was performed. The nanodevice Micromega-B was administered orally as an immunosorbent and immunomodulator, with dosage and treatment regimens tailored to the patient. Assessments of general condition and neurological function were conducted weekly over a six-month period, with a contrast-enhanced MRI scan performed in the fifth month.

**Results:** Treatment with micromega-B resulted in a measurable improvement in neurological status. Patient experienced reduced stiffness and fatigue in the lower limbs, improved gait and coordination, diminished hand tremors, alleviation of depression and concentration difficulties, restoration of appetite, and enhanced speech function. Throughout the treatment, positive trends in neurological normalization were observed. After six months, the total EDSS score decreased from 210 to 45. The most significant improvements were noted in pyramidal function and coordination, with a reduction in the EDSS Disability Scale score from 6.0 to 5.0. Remarkably, by the fourth month of treatment, contrast-enhanced MRI revealed a decrease in the number of newly formed demyelinating foci for the first time. The observed improvements in neurological status corresponded with MRI findings. The recovery of central nervous system function in MS appears to result not only from the immunosuppressive properties of magnetite nanoparticles but also from the activation of remyelination mechanisms and oligodendrocyte differentiation through enzymatic methylation.

**Conclusion:** The integration of biocompatible nanodevices into MS therapy holds significant promise. Further refinement and research into the optimal regimen and administration methods of biocompatible magnetite nanoparticles are warranted to maximize their therapeutic efficacy in MS management.

**Key Words:** multiple sclerosis; treatment; nanodevice; micromega-B; neurological status assessment; remyelination

## Introduction

Multiple sclerosis (MS) is a major neurological disorder characterized by its high prevalence, chronic progression, frequent development of disability, and a tendency to affect young adults, with an average onset age of 30 years. The predominant hypothesis regarding the immunopathogenesis of MS suggests a breakdown in immune tolerance, allowing autoreactive cells sensitized to nervous tissue antigens to cross

the blood-brain barrier and infiltrate the brain. B lymphocytes recognize myelin and activate T cells, triggering an immune response [1-7]. Activated T and B cells release signaling molecules that recruit additional immune cells, leading to inflammation [8,9]. Plasma cells produce antibodies that target myelin and amplify immune cell recruitment [10,11]. This sustained immune activity enables T and B cells to establish a persistent presence in the central nervous system (CNS), perpetuating neural damage [12,13]. There are two primary hypotheses regarding the pathogenesis of multiple sclerosis (MS). The outside-in hypothesis suggests that immunocompetent cells activated in the periphery infiltrate the brain and initiate an immune response. In contrast, the inside-out hypothesis proposes that primary damage to nervous tissue triggers the expression of damage-associated molecular pattern (DAMP) receptors, leading to immune activation and loss of tolerance to myelin antigens [14]. Plasma cells produce antibodies that actively degrade the protective myelin sheath of nerve cells, resulting in inflammation. Over time, this process leads to scarring, disrupting nerve signal conduction. As a result, neural impulses from the brain fail to reach the limbs and organs, ultimately impairing motor control and bodily functions [15]. The most common initial clinical symptoms include limb weakness and sensory disturbances, vision impairment, urinary dysfunction, and cerebellar ataxia. Currently, pathogenetic treatment for MS primarily relies on immunomodulatory and immunosuppressive therapies that modify disease progression. These treatments operate through various mechanisms, including:

1. Selective immunosuppression.
2. Complete immunosuppression.
3. Inhibition of activated immune cell migration from lymph nodes or into brain tissue.
4. A combination of immunoregulatory, anti-inflammatory, antioxidant, and potentially neurotrophic effects.

Future therapeutic strategies for MS focus on selective local immunocorrection, remyelination, neuroprotection, enhancement of neuroplasticity and functional relocalization, assessment of the efficacy and safety of cell-based therapies, and personalized treatment selection. Advances in molecular and cellular biology are expected to enable more precise predictions of disease progression and individual responses to therapy, paving the way for a more targeted and effective approach to MS management [16-22]. The progression of inflammation and demyelination in multiple sclerosis (MS) is driven by dysregulation of the immune response, an imbalance between regulatory and effector T cells, activation of B-cell immunity, and microglial involvement. All disease-modifying therapies for MS either deplete T or B cells or modulate signaling pathways critical to immune response regulation [23]. Recent research has highlighted the crucial role of B cells in sustaining chronic inflammation within the central nervous system (CNS). Their functions include autoantibody production, antigen presentation, and continuous activation of T cells within the brain parenchyma. Anti-B-cell therapies such as Rituximab, Ocrelizumab, and Ofatumumab have demonstrated efficacy in both relapsing and progressive forms of MS. Efforts to stimulate remyelination have led to the development of novel monoclonal antibodies, including anti-LINGO and human immunoglobulin M (IgM), which promote myelin repair by enhancing oligodendrocyte differentiation and maturation [24]. A key contributor to axonal degeneration in MS is mitochondrial dysfunction. Several compounds, such as Dimethyl fumarate, Idebenone, and Biotin, show promise in addressing mitochondrial deficits and supporting neuronal survival [25]. Additionally, drugs targeting ion channel redistribution in demyelinated axons such as Lamotrigine, Amiloride, and Fampridine - may help mitigate energy deficits in neurons and axons, potentially improving neural function and reducing neurodegeneration [26]. In recent years, biocompatible nanotechnological agents have gained increasing application in medicine. Since 1998, Ukrainian clinics have officially utilized nanodevices developed by Belousov's Applied Nanotechnology Laboratory, including the Micromage-B, MCS-B, and ICNB brands (Figure 1) [27]. These nanodevices are based on biocompatible magnetite nanoparticles, whose unique physicochemical properties enable diverse medical applications. They modulate the quantitative and qualitative composition of bodily fluids, influence metabolic and biochemical processes, and regulate cellular energy balance. Their selective sorption activity targets surface proteins of cell membranes, circulating immune complexes, lymphocytotoxic antibodies, and the complement system. Additionally, they enhance phagocytic activity and improve the leukocyte phagocytosis completion index [28], making them effective for immunocorrection. Beyond immune modulation, these nanopreparations impact glycolysis, ion channel activity in cell membranes, and erythrocyte function, while also improving microcirculation and reducing platelet aggregation (Figure 2) [29-31]. Furthermore, they activate antiradical enzyme systems and inhibit lipid peroxidation, contributing to their broad therapeutic potential [32,33].


**Figure 1:** New official nanomedical devices based on biocompatible magnetite nanoparticles.

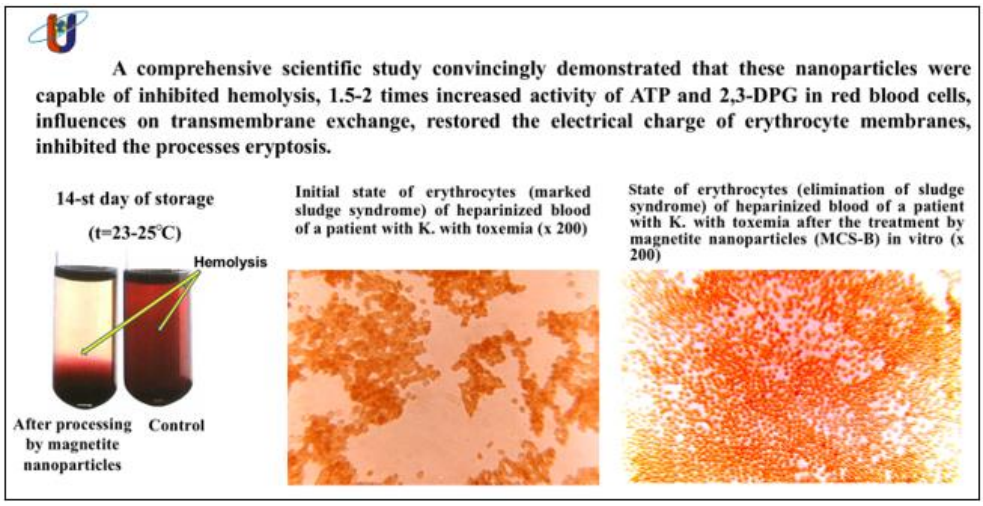


**Figure 2:** Effect of biocompatible magnetite nanoparticles on functional status of erythrocytes and hemorheology.

The findings of these studies provide a solid foundation for developing innovative approaches to the safe and effective use of biocompatible magnetite nanoparticles in the treatment of severe autoimmune diseases, such as multiple sclerosis (MS). The main goal of this research is to slow the progression of MS, enhance the patient's neurological function and overall health, and limit the spread of demyelinating lesions in the brain.

## Materials and methods

Patient K. was diagnosed with secondary progressive multiple sclerosis (MS), cerebrospinal form, at the stage of clinical exacerbation. The patient exhibited severe spastic tetraparesis, with greater involvement of the lower limbs, resulting in impaired mobility. Additionally, the patient presented with significant urinary-ataxic syndrome, as well as sphincter and sensory dysfunction. MRI scans revealed multifocal diffuse demyelinating brain lesions (more than 30), indicative of the active disease phase, along with a diffuse atrophic process in the cerebral cortex. Over the year preceding study inclusion, the patient experienced an average of 1.0 relapse, with an Expanded Disability Status Scale (EDSS) score of 6.0. The disease had been progressing for 24 years since the onset of initial symptoms. For 14 years, the patient underwent regular treatment with vascular and metabolic agents, in conjunction with hormonal therapy. Despite this, the disease transitioned to a secondary progressive form after 6 years, prompting the introduction of the immunosuppressive drug Teriflunomide into the treatment regimen.

However, despite ongoing treatment efforts, the patient's general condition continued to deteriorate, and the neurological status remained unstable. MRI scans over the past four years showed a progressive increase in the number of new demyelinating lesions in the brain. In light of the above, the patient's treatment regimen was augmented with the addition of Micromage-B [34]. Micromage-B is an oral nanodevice officially registered by the Ministry of Health of Ukraine. It consists of a powdered form of magnetite ($Fe_3O_4$) nanoparticles, developed for the prevention and treatment of various conditions, as well as to enhance the body's resilience to adverse environmental factors. As a nanotechnology-based product, Micromage-B contains magnetite nanoparticles ranging in size from 6 to 12 nm. The therapeutic effect of Micromage-B is primarily driven by the sorption mechanism and the influence of a constant magnetic field on cellular and subcellular structures induced by the magnetite nanoparticles. These nanoparticles have a sorption surface area of 800 to 1200 $m^2/g$ and generate a magnetic field strength of 300-400 kA/m. The main target of Micromage-B is the microenvironment of the cell's aqueous spaces and surface membrane proteins. Through selective sorption, the magnetite nanoparticles alter the quantitative and qualitative composition of cell surface proteins. Additionally, the constant magnetic field affects the mobility and orientation of hydrogen protons within the cell's aqueous microenvironment (Figure 3).

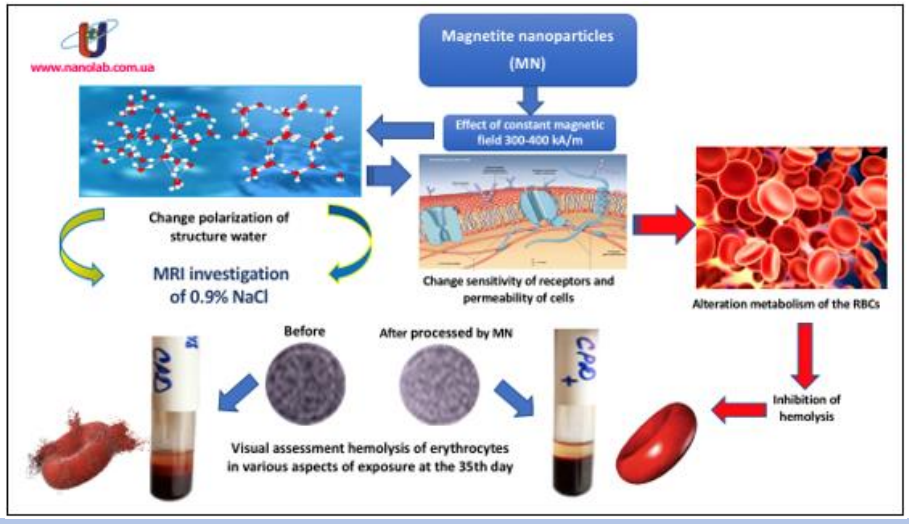


**Figure 3:** The effect of magnetite nanoparticles on hemolysis processes by changing the polarization structure of the aqueous sector of the erythrocyte microenvironment.

This mechanism triggers the hydrolysis of the phosphate residue of ATP, leading to alterations in the cell's transmembrane exchange and metabolism, and modifying its susceptibility to various stimuli. The nanodevice enhances the cell's adaptive mechanisms and optimizes the function of cellular organelles. It accelerates repair processes at the membrane and macromolecular levels, significantly boosts intracellular synthetic reactions, and enhances the compensatory capabilities of organelles within the renal glomerulus cells and the epithelial cells of the proximal and distal nephron segments. This structural improvement is reflected in enhanced bioenergetic support for synthetic intracellular processes, as well as an increase in the repair and adaptive functions of nephrons. By enhancing redox phosphorylation processes, which meet the energy demands of synthetic reactions in the membrane and macromolecular components of hepatic cells, Micromage-B acts as a direct activator of reparative intracellular processes in hepatocytes, promoting glycogen synthesis. This property makes it an effective hepatoprotective agent for treating both acute and chronic liver conditions. Micromage-B acts as a potent erythropoiesis stimulator, rapidly restoring hemoglobin levels in the blood. It also stimulates the synthesis of pulmonary surfactant, improving lung tissue stretchability and elasticity, thereby enhancing lung stability against both internal and external stressors. This effect is mediated by increased activity of alveolar macrophages. Clinically and in laboratory settings, Micromage-B enhances microcirculation and blood rheological properties by stabilizing erythrocyte membrane bioelectric charge, exhibiting a significant immunomodulatory effect, and selectively exerting bacteriostatic effects against pathogenic microflora while preserving normoflora. It impedes the growth and proliferation of various fungi and promotes the growth and activity of lactic acid bacteria in the intestines, thereby enhancing the efficacy of antibacterial and antifungal agents by 2-3 times. These attributes enable Micromage-B's effective application in dysbacteriosis and candidiasis treatment. Micromage-B nanoparticles adsorb toxic substances and circulating immune complexes, significantly enhancing the treatment efficacy of various allergic diseases, autoimmune processes (such as rheumatoid arthritis, acute and chronic polyarthritis, eczema, etc.), and acute poisoning. Micromage-B moderately improves renal blood flow, exerting a mild diuretic effect.

Magnetite nanoparticles in Micromage-B, with their intrinsic magnetic properties, aid in the breakdown and dissolution of kidney and bile duct stones, allowing them to be eliminated from the body as magnetically responsive crystalline forms through the excretory system. In addition to this, Micromage-B plays a key role in normalizing blood lipid and protein levels. It also impacts factors involved in atherogenesis, helping to slow the progression of atherosclerotic processes. The constant magnetic field generated by the magnetite nanoparticles reduces the release of excess aggressive mediators from macrophage cells into the bloodstream, leading to notable anti-inflammatory and mild analgesic effects. Furthermore, Micromage-B regulates antioxidant enzyme activity, absorbs products of lipid peroxidation, and helps restore the balance between antiradical and pro-radical substances. Under its influence, monocytes actively produce tumor necrosis factor (cachexin), which exerts cytotoxic and cytostatic effects on tumor cells. When applied topically (as powders, ointments, or aqueous colloidal solutions), Micromage-B accelerates the healing of mucous membranes and skin wounds, promoting the transition from wet tissue necrosis to dry. Micromage-B is non-toxic and does not interfere with organ or systemic function [35,36].

The dosing regimen for Micromage-B entails 500 mg daily for the first month, 500 mg every other day for the second month, and subsequently, 500 mg once every three days. The selection of Micromage-B dosage and regimen is tailored individually, considering the patient's rate of improvement and neurological recovery.

The study monitored changes in neurological status using a modified version of the Multiple Sclerosis Patient Assessment Scale [37,38], which evaluates the severity of motor impairments alongside other nervous system damage indicators. Disability was quantitatively assessed using Kurtzke's online EDSS calculator [39]. Manifestations of cerebral demyelination foci were examined through contrast-enhanced MRI.

The patient's overall condition and neurological status were assessed every 7 days over a 6-month period. As per the protocol, a contrast-enhanced MRI of the brain was conducted annually, aligning with the 5th month of Micromage-B usage.

## Results

The progression of neurological changes was evaluated using a modified scale. One week after starting Micromage-B, a significant improvement in the patient's neurological condition was observed. The patient reported a substantial reduction in lower limb stiffness and fatigue. Objectively, there were notable improvements in gait and coordination, a decrease in hand tremors, complete resolution of depression and concentration issues, restoration of appetite, and enhanced speech. Positive trends in the normalization of neurological status were maintained throughout the entire duration of Micromage-B treatment. Table 1 shows the evaluation of the neurological status of a multiple sclerosis patient before and after six months of Micromage-B therapy.

| 1. Movements (pyramidal system) | | | | |
|---|---|---|---|---|
| Input data | | Six months after application Micromage-B | | Clinical manifestations |
| Arm | Leg | Arm | Leg | |
| 0 | 0 | 0 | 0 | Norma |
| 5 | 10 | **5*** | **10*** | Absence of loss symptoms, revival of tendon reflexes, enlargement of reflexogenic zones, clonus, presence of pathological signs, anisoreflexia, (absence of paresis) |
| 10 | 20 | 10 | 20 | Raises a limb independently, full volume of active movements, signs of pyramidal lesion, overcomes not only the gravity of his own limb, but also an additional obstacle of moderate strength, positive Barre-Rusetsky's symptom |
| 15 | 40 | 15 | 40 | Raises a limb independently, the volume of active movements is full, cannot hold a limb in a given position for a long time, and cannot overcome an additional obstacle. |
| **20*** | **60*** | 20 | 60 | Can pull a limb off the plane, the amount of active movement is limited, cannot hold a limb in a given position. |
| 40 | 80 | 40 | 80 | Cannot detach a limb from the plane, active movements in the joints of the fingers, ankle and wrist, elbow, knee joints are possible only on the plane. |
| 50 | 100 | 50 | 100 | Complete absence of movement (paralysis) |
| 2. Sensitivity | | | | |

| Before | After | (a) superficial sensitivity |
|---|---|---|
| 0 | **0*** | Norma |
| 5 | 0 | Paresthesias, burning sensation, numbness, coldness of a limb (no objective disorders) |
| **10*** | 10 | Hyperesthesia or hypoesthesia |
| 15 | 15 | Anesthesia phenomena |
| | | b) deep sensitivity |
| **0*** | **0*** | Norma |
| 10 | 10 | Disorder of joint and muscle feeling in small joints |
| 20 | 20 | Disorder of joint and muscle feeling up to the level of the middle joints (wrist, ankle) |
| 40 | 40 | Disorder of joint and muscle feeling up to the level of large joints (elbow, shoulder, knee, hip) |
| | | |
| 3. Coordination | | |
| 0 | 0 | Norma |
| 10 | **10*** | Unsteadiness, swaying in the sensitized Romberg's test while standing on one leg, mild intensional trembling (in the complicated test), slight ataxia in the heel-knee test, deviation when walking with eyes closed. |
| **40*** | 10 | Unsteadiness in simple Romberg's pose, atactic gait with open eyes and legs spread wide apart, "drunkenness," moderately pronounced intensional tremor and ataxia in the heel-knee test. |
| 100 | 100 | Because of ataxia, the patient cannot move without assistance, sharp hypotonia of muscles, intensional shaking of the head, trunk, coarse - upper extremities, coarse ataxia during heel-knee test, trembling of upper extremities when trying to perform purposeful movement, chanted speech. |
| 4. Psycho-emotional sphere | | |
| 0 | **0*** | Norma |
| 10 | 10 | Mild impairment of the intellect in combination with euphoria, rapid change of mood, neurasthenic syndrome. |
| **20*** | 0 | Euphoria, depression, decreased criticism of one's condition, decreased memory. |
| 100 | 100 | Severe mental disorder, complete intellectual disintegration, Korsak's syndrome, etc. |
| 5. Nystagmus | | |
| 0 | **0*** | Norma |
| 5 | 5 | Nystagmus is detected only in the extreme leads (degree 1) |
| **10*** | 10 | Nystagmus when looking straight ahead (degree 2) |
| 15 | 15 | Sharp beating nystagmus, nystagmus in both directions, even toward the slow component (3rd degree) |
| | | 6. State of the sphincters |
| 0 | **0*** | Norma |
| **10*** | 10 | Impulsive urges, inability to hold urine for a long time, difficulty urinating |
| 20 | 20 | Urinary incontinence, urinary retention, intermittent urination disorders, persistent constipation |
| | | 7. Sexual function |
| 0 | **0*** | Norma |

| | | |
|---|---|---|
| **5*** | 5 | Decreased sexual activity in men (intermittent impotence), sexual coldness in women |
| 10 | 10 | Total impotence, menstrual disorders in women |
| | | 8. The ocular fundus |
| 0 | 0 | Norma |
| 5 | 5 | Disturbance of vascular pattern, narrowing of arteries, dilation of veins, changes on fluorescence ophthalmoscopy |
| **10*** | **10*** | Partial optic atrophy (bitemporal pallor), optic neuritis |
| 15 | 15 | Complete atrophy of the optic nerve |
| | | 9. Visual acuity |
| 0 | 0 | Norma (vision within 1.0 or myopia) |
| 5 | 5 | Occasional blurring of vision, rapid fatigue when reading and performing work without impaired visual acuity |
| **10*** | **10*** | Visual acuity from 0.9 to 0.7 |
| 15 | 15 | Visual acuity from 0.6 to 0.4 |
| 20 | 20 | Visual acuity from 0.3 to 0.1 |
| 25 | 25 | Visual acuity 0.1 and below |
| 100 | 100 | Blindness in one or both eyes |
| | | 10. Oculomotor nerves |
| 0 | **0*** | Norma (absence of subjective and objective symptoms) |
| **5*** | 0 | Concealed insufficiency, without visible dysfunction of one of the oculomotor nerves, inter-nuclear ophthalmoplegia syndrome |
| 10 | 10 | Mild visible impairment, visible insufficiency of one or more nerves, diplopia, ptosis, anisocoria |
| 15 | 15 | Convergent or divergent strabismus |
| 20 | 20 | Complete ophthalmoplegia (in one or both eyes) |
| | | 11. The trigeminal nerve |
| **0*** | **0*** | Norma (absence of subjective and objective symptoms) |
| 5 | 5 | Subjective sensations in the form of pain, numbness, sense of "creeping chills", oppression in the face. |
| 10 | 10 | Objective signs of lesions, hypoesthesia, loss or decrease in the corneal reflex. |
| 20 | 20 | Severe anomalies with loss of trigeminal nervous functions, with or without neuralgic disorders. |
| | | 12. The facial nerve |
| 0 | **0*** | Norma |
| **5*** | 0 | Moderate weakness of facial muscles (eye closes completely, but cannot actively close it), asymmetry of frontal and nasolabial folds |
| 10 | 10 | Moderate weakness of mimic muscles (lagophthalmus, positive Bell's symptom, facial asymmetry in grinning), with preservation, to some extent, of mimic movements |
| 20 | 20 | Complete paralysis of facial muscles |
| | | 13. Bulbar group of cranial nerves (nerves 9,10,11,12) |
| 0 | **0*** | Norma |

| | | |
|---|---|---|
| **5*** | 5 | Mildly pronounced bulbar phenomena (gagging when taking liquid food, change in speech, without gross organic symptoms of prolapse) |
| 10 | 10 | Severe dysphagia, dysarthria, decreased soft palate and posterior pharyngeal wall reflexes |
| 100 | 100 | Complete bulbar paralysis |
| | | 14. Auditory nerve |
| **0*** | **0*** | Norma |
| 5 | 5 | Conversational speech at a distance of 4 to 6 m, whispered speech at a distance of 1 to 3 m. |
| 10 | 10 | Speech - from 2 to 4 m, whisper - from 0.5 to 1 m. |
| 15 | 15 | Spoken speech 2 m or less, whispered speech 0 to 0.5 m |
| 20 | 20 | Complete deafness in one or both ears |
| Total: | Total: | |
| **210*** | **45*** | |

**Table 1:** Assessment of Neurological Status Scale for Patient K. with Multiple Sclerosis.

**Note: *** - estimated scores of the neurological status before and after using the Micromage-B.

The data presented in Table 1 illustrate a positive trend in normalizing neurological status following 6 months of Micromage-B usage. Initially, the total points amounted to 210, which decreased to 45 after the 6-month period, indicating a reduction of 165 points. The most significant improvement was observed in the assessment of the pyramidal system and coordination. Additionally, the EDSS disability scale score decreased from 6.0 to 5.0. A contrast-enhanced MRI of the brain conducted after 4 months of Micromage-B usage revealed a decrease in the number of new demyelination foci in the brain for the first time. The favorable progression of neurological status correlated with the brain MRI findings.

Upon analyzing the collected data, particular attention should be given to the observed positive clinical effects attributed to immunosorption and the active contribution of Micromage-B's magnetite nanoparticles to neurological status restoration. This effect may be attributed to remyelination processes and oligodendrocyte differentiation. Oligodendrocytes, a type of neuroglial cell, form the myelin sheath around neurons in the central nervous system (CNS). The molecular mechanisms underlying cell differentiation and specialization remain complex and poorly understood, presenting a challenging area in cell and developmental biology. The development and maturation of various cell types continue to pose significant research challenges.

A recent study has shown that one of the key mechanisms of oligodendrocyte maturation involves enzymatic methylation, specifically the addition of a methyl group (-CH3) to the N6 nitrogen atom in the adenosine base, a process known as m6A-methylation. Although this modification may seem subtle, it can profoundly influence subsequent stages of protein biosynthesis. The role of m6A-methylation has been demonstrated in various processes associated with oligodendrocyte maturation [40]. The universal donor of methyl groups in the body is S-adenosylmethionine, which is synthesized through the interaction of the amino acid methionine and ATP. Micromage-B activates glycolysis, leading to a significant increase in ATP production [41,42], and promotes the formation of the reduced form of the coenzyme NADPH2, aiding in the reduction of oxidized glutathione [43]. These conditions create an environment conducive to the initiation of enzymatic methylation processes, likely enhancing the action of magnetite nanoparticles (Micromage-B). This, in turn, supports oligodendrocyte differentiation and the remyelination process. Additionally, it is important to note that these nanoparticles alter the aqueous environment of the cellular microenvironment [44], triggering ATP hydrolysis, energy release, and the formation of ADP. For a more comprehensive understanding of how biocompatible magnetite nanoparticles (Micromage-B) influence remyelination, refer to Figure 4.

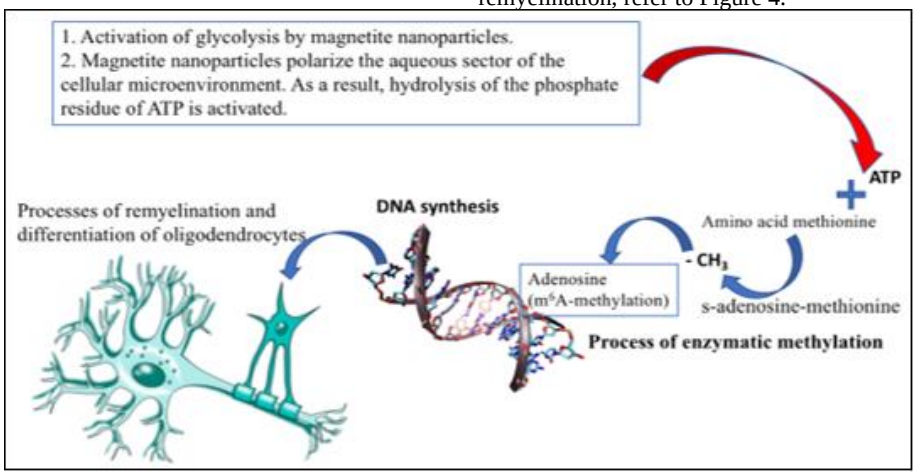


**Figure 4:** Mechanism of the effect of magnetite nanoparticles on oligodendrocyte maturation.

Due to the positive changes in the neurological status, it was determined that continuing the administration of Micromage-B at the prescribed dosage was appropriate. The therapy was further supplemented with a comprehensive rehabilitation exercise program aimed at accelerating the recovery of physical, cognitive, and psychosocial functions in the patient with MS.

## Conclusion

The results of the study expanded the clinical applicability of biocompatible magnetic nanoparticles in the treatment of severe autoimmune diseases [45-48]. The use of the Micromage-B nanopreparation in the treatment of multiple sclerosis (MS) showed significant positive clinical effects. Throughout the treatment period, notable improvements were observed in the normalization of the neurological condition. After six months of treatment, the overall score dropped from 210 to 45, with the most significant improvements seen in the pyramidal system and coordination assessments. The EDSS Disability Scale score decreased from 6.0 to 5.0. For the first time, contrast-enhanced brain MRI revealed a reduction in the number of new demyelination foci by the fourth month of Micromage-B administration. The improved neurological condition correlated with positive results from brain MRI scans. The restoration of central nervous system function in MS is attributed not only to the immunosuppressive effects of magnetite nanoparticles but also likely to the activation of remyelination mechanisms and oligodendrocyte differentiation through enzymatic methylation. The design and use of biocompatible magnetite nanoparticles to enhance MS treatment efficacy require further refinement and study. The incorporation of biocompatible nanodevices in the comprehensive treatment of MS is a promising innovation, with plans to integrate magnetite nanoparticles into treatment protocols for MS in the near future [49-51].

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