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## Differential Diagnosis and Treatment of Multiple Sclerosis

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ABSTRACT	It is known that multiple sclerosis is one of the most common chronic autoimmune diseases of the central nervous system. Although "sclerosis" is often colloquially referred to as memory impairment in old age, the term "multiple sclerosis" does not refer to either senile "sclerosis" or distraction. "Sclerosis" in this case means "scar", and "scattered" means "multiple", since the distinguishing feature of the disease in pathological anatomical examination is the presence of scattered foci of sclerosis throughout the central nervous system without a specific localization - replacement of normal nervous tissue with connective.	
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Multiple sclerosis (MS) is a chronic progressive disease of the nervous system of unknown etiology that can occur at any age, but is most often observed in young and middle (15-40 years old). The disease proceeds with exacerbations (exacerbations) and remissions, or has a primary or secondary progressive course, invariably leading to permanent disability, impaired professional functioning and loss of social activity [1].

MS is one of the most common diseases: today there are about 3 million patients in the world. To a greater extent, the disease is recorded among white people with a certain zonality: MS is rare in Japan, Korea, China, and is most typical for Europe. Women are more often ill, and their disease begins on average 1-2 years earlier than in men. However, in men, a more unfavorable, progressive form of the course predominates [2, 3].

Treatment of MS can be conditionally divided into two groups: treatment of exacerbations and prevention of exacerbations by immunomodulatory therapy.

At the moment, а number of immunomodulatory drugs that change the natural course of the disease have also been approved as a pathogenetic treatment for MS. There are several groups of modulating action: interferon-b preparations, glatiramer acetate (Copaxone), drugs cytotoxic with immunosuppressive properties (mitoxantrone) and preparations of monoclonal antibodies to various immunocyte epitopes (natalizumab, alemtuzumab, rituximab and daclizumab) [5, 6.71.

Immunomodulatory therapy with interferons helps to slow down the progression

of atrophic changes in the brain by 45-55% compared with placebo.

All three interferon-b preparations can be used to treat relapsing-remitting MS: betaferon (interferon-P-1b), rebif (interferon-P-1a), and Avonex (interferon-P-1a). Betaferon is used at a dose of 250 mcg (8 MME) subcutaneously, every other day, rebif - in two doses - 22 mcg (6 MME) and 44 mcg (12 MME) 3 times a week, subcutaneously. Avonex administered is intramuscularly once a week at a dose of 30 µg (6 MME), which is an advantage from the patient's point of view [8]. For the treatment of secondary progressive MS, betaferon and rebif at a dose of 44  $\mu$ g are mainly used [9].

At present,  $\beta$ -interferons (betaferon, rebif) and Copaxone remain the first-line drugs for pathogenetic preventive therapy of MS in the world. Multicenter placebo-controlled studies have proven the effectiveness of long-term use of such treatment, a significant drawback of which is its high cost, which reduces its availability [10,11].

Natalizumab, a selective adhesion molecule inhibitor and  $\alpha$ -4 integrin antagonist, is a new drug used to treat patients with active relapsing-remitting MS. By binding to a-4integrins, which are highly expressed on the surface of leukocytes (with the exception of neutrophils), the drug prevents their ability to interact with adhesion molecules and osteopontin, which is a necessary condition for the attachment of leukocytes to the endothelial surface [12].

Reparative agents include neuroprotectors, specific inhibitors of metalloproteinases, potassium channel blockers, however, none of them has proven clinical efficacy, therefore they are used as additional therapeutic agents for MS.

Drugs not registered for the treatment of MS (in clinical trials):

• immunosuppressive agents (teriflunomide, cladribine);

• anti-inflammatory drugs (laquinimod, fingolimod, dimethyl fumarate or BG-00012);

• combinations of registered agents for the treatment of MS.

These drugs allow you to choose not only the method of administration, but also the mechanism of action of the drug. *Cladribine* is a purine nucleoside analog that acts against certain populations of lymphocytes, which reduces their amount in the blood. According to studies of cladribine for parenteral administration at doses of 0.7–2.8 mg/kg in relapsing-remitting MS and secondarily progressive

MS, treatment leads to a decrease in the number and volume of new lesions, the frequency of exacerbations and the rate of progression of disability.

*Teriflunomide* is a modified form of leflunomide that is in phase III clinical trials for remitting MS and secondary progressive MS with exacerbations. Its efficacy and safety have been demonstrated in a Phase II study [7].

*Fingolimod* (FTY720) is the first sphingosine-1-phosphate receptor modulator that was shown to be effective in reducing the frequency of exacerbations and disease activity by 50% in a 6-month phase II study, according to neuroimaging data [13,14].

Of great importance in MS is symptomatic therapy, the purpose of which is to influence various manifestations of the disease (spasticity, urination disorders, and others). For this, medications, electrical stimulation and surgical methods of treatment are used.

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