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IN THE EARLY STAGES OF GUILLAIN-BARRE SYNDROME, GANGLIOSIDES (GM1 AUTOANTIBODIES AND GM 1) IN THE BLOOD SERUM

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THESIS

In the last two decades, patients with GBS have shown variability in the course of the disease and its outcomes, despite timely specific therapy (plasmapheresis or class G immunoglobulins) In this regard, there is a need to create a system for predicting the condition of patients for a year from the moment of the appearance of the first clinical symptoms. The percentage of delayed diagnosis of the disease remains quite high, and the ambiguity of pathogenetic mechanisms makes it difficult to choose an adequate therapy. Mortality in GBS is 5-10% and a number of patients (16%) have severe residual manifestations of the disease that limit their medical and social rehabilitation.

It is known that some infectious agents are specifically associated with the development of axonal or demyelinating polyneuropathy (Mori M. et al., 2000, Vittorio G., 2001). The question of the role of previous factors (infectious and non-infectious) for the course and formation of individual clinical and immunological variants of GBS has not been sufficiently studied.

Gangliosides are a group of glycosphingolipids that make up 6% of all lipids in the nervous system. They are the main structural part of the myelin of Schwann cells, are part of the structures of synaptic membranes and neuromuscular endings. The structure of gangliosides is based on a ceramide molecule combined with molecules of carbohydrates and sialic acids. The spectrum of gangliosides is different in the central nervous system and peripheral nerves. Gangliosides GM1 and GD1a are expressed on motor nerves and axons. Cranial nerves have a lot of ganglioside GQ1b, sensory nerves are rich in GD1b.

As the main cause of sensitization of the immune system to ganglioside antigens in acute polyneuropathies, a cross-immune reaction with lipopolysaccharides of gastrointestinal bacteria is assumed. Guillain-Barre syndrome is often preceded by enteritis caused by Campylobacter jejuni. C. jejuni lipopolysaccharide contains a ganglioside-like structure similar in structure to the GM1 ganglioside molecule, which is capable of stimulating autoimmune responses. Other known inducers of Guillain-Barre syndrome

are cytomegalovirus, Epstein-Barr virus, Mycoplasma pneumoniae and Haemophilus influenzae. Antibodies to gangliosides occur against the background of a number of acute and chronic polyneuropathies.

A common feature of both acute and chronic autoimmune polyneuropathies is protein-cell dissociation in the cerebrospinal fluid, manifested by a significant increase in the protein content in the cerebrospinal fluid with normal or minimally increased cellularity. Guillain-Barre syndrome and its varieties belong to the group of acute inflammatory demyelinating polyradiculoneuropathies, often resolved spontaneously. This disease often develops against or after a respiratory or intestinal infection within 10-14 days from its onset, so the formation of autoantibodies is often considered as a component of the primary immune response against the causative agent of infection. After achieving remission of the disease, antibody titers gradually decrease and increase in case of repeated exacerbation. One of the forms of acute inflammatory demyelinating polyneuropathy is Miller-Fisher syndrome. Characteristic clinical manifestations are lesions of the cranial nerves, as a result of which ophthalmoplegia develops, in addition, areflexia and ataxia are expressed. Chronic inflammatory demyelinating polyneuropathy is a chronic progressive disease with damage to sensory and motor nerves. The disease can occur with exacerbations alternating with prolonged periods of remission. In chronic polyneuritis, antibodies to gangliosides can be represented by monoclonal immunoglobulin and require immunofixation of serum immunoglobulins (see the description of the test).

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