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Clinical and Laboratory Changes in Guillain-Barre Syndrome

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Abstract: Guillain-Barré syndrome is a severe autoimmune disease affecting the peripheral nervous system. The most common manifestation is acute tetraparesis, in which the movement of the four limbs becomes almost impossible. Other movements also cease, including swallowing, the ability to lift the eyelids, and spontaneous breathing. Nevertheless, the course of the disease is good, and in most cases it ends with recovery. Transition to a chronic course or relapses are less common. Guillain-Barre syndrome occurs with the same frequency in all countries, regardless of their level of development - about 2 cases per 100,000 people, regardless of gender. The disease can affect patients of any age.
Key words: Epstein-Barr virus or human herpes type, mycoplasma; Campylobacter, which causes infectious diarrhea; cytomegalovirus. Guillain-Barré syndrome

The leading mechanism of development is autoimmune. In most cases, the onset of the disease occurs within the first three weeks after an acute respiratory or intestinal infection. Since enough time has passed since the onset of the disease and there is time for the symptoms characteristic of the infectious process to pass, the patients themselves, as a rule, do not associate these conditions with each other. The reason for this may be pathogens, for example:

Epstein-Barr virus or human herpes type 4;
mycoplasma;
Campylobacter, which causes infectious diarrhea;
cytomegalovirus.
Guillain-Barré syndrome
Researchers have found that the "cap" of these pathogens is similar to the myelin sheath of the axon of peripheral nerves. This similarity causes the nerves to be attacked by antibodies, which are initially produced and circulate in the blood in response to the appearance of an infectious agent. This phenomenon is called "molecular mimicry" and explains why immune complexes attack the body's own tissues.

Cases of the syndrome after vaccination, surgery and abortion, hypothermia and stress have been described. In some cases, the cause cannot be found.

HOW DOES THE SYNDROME APPEAR?
For several days, up to a maximum of 1 month, muscle weakness in the legs increases, difficulties appear when walking. Later, the hands become weak, the last time the facial muscles suffer. Such

symptoms have a special name - elevated Landry paralysis.

But sometimes the paralysis starts from above, from the hands, spreads downwards, but all the limbs are always affected.

Every fifth case is accompanied by paralysis of the trunk muscles, that is, the diaphragm and intercostal muscles. With such paralysis, breathing becomes impossible and artificial ventilation is required.

A common manifestation is bulbar syndrome or bilateral paralysis of soft palate muscles, inability to swallow and speak clearly.

Along with motor fibers, sensory fibers are sometimes affected. Emotional disorders develop, tendon reflexes decrease, pain occurs in the limbs. The pain is clearly "neuropathic" in nature - burning, streaming, tingling. Disorders of the pelvic organs are rare, but often there is retention of urine, which in some cases is combined with excessive urine production.

Autonomic dysfunction, manifested by changes in blood pressure, palpitations, other heart rhythm disturbances, sweating and lack of bowel movement, is added.

CLASSIFICATION

Depending on the severity and prognosis, there are several forms depending on whether the myelin sheath or axon is damaged:

acute inflammatory demyelinating polyneuropathy or AIDP, when the myelin sheath is destroyed;
acute motor or sensorimotor axonal neuropathy, when axons are destroyed;
rare forms - Miller-Fisher syndrome, acute pandysautonomia, etc., the frequency of which does not exceed 3%.

DIAGNOSTIC MEASURES

Diagnosis is based on WHO recommendations. There are two main diagnostic criteria:

muscle weakness develops in the limbs;
decrease or absence of tendon reflexes from the first days of the disease.
WHO also identifies additional signs that confirm the diagnosis, including:

lesion symmetry;
symptoms increase in no more than 4 weeks;
emotional disorders of the "glove and socks" type;
involvement of the cranial nerves, especially the facial nerve;
after stopping the development of the disease, the functions can be restored by themselves (the so-called "plateau");
presence of vegetative diseases;
absence of hyperthermia (if there is a fever, it is caused by other infections);
an increase in the amount of protein in the cerebrospinal fluid, its cellular composition does not change (protein-cellular separation).
It is impossible to make a definite diagnosis without electroneuromyography or ENMG. This test determines which part of the nerve - the myelin sheath or the axon - is damaged. ENMG also accurately

determines the level of the lesion, its severity and the possibility of recovery.

Since there are a number of acute, subacute and chronic polyneuropathy in addition to Guillain-Barre syndrome, electroneuromyography allows for differential diagnosis between them and helps to develop the correct treatment tactics.

Diagnosis often requires a lumbar puncture, followed by a study of the cerebrospinal fluid and the following tests:

blood for autoantibodies to neuronal structures;
blood for gamma globulins of class A (especially if immunoglobulin therapy is planned);
biomarkers of neurofilament (a part of neuronal cytoplasm);
markers of tau protein (a special protein that destroys neurons).
Guillain-Barré syndrome

TREATMENT

Plasmapheresis is a blood purification operation. Often, hardware plasmapheresis is used in continuous separators, during which the blood taken from the body is divided into formed elements (or blood cells) and plasma (or serum). All toxic substances are in the plasma, so it is removed. A person's own blood cells are diluted with plasma replacement solutions or donor plasma, if needed. The duration of the procedure is about an hour and a half, the whole course consists of 3 or 5 sessions. At the same time, plasma is removed in an amount not exceeding 50 ml / kg of body weight.

Blood parameters are monitored during treatment: electrolytes, hematocrit, clotting time, etc.

Intravenous immunotherapy is the administration of human immunoglobulin G. These immunoglobulins stop the production of antibodies to their nerves, while at the same time reducing the production of pro-inflammatory substances. These drugs are indicated for the pathogenetic treatment of Guillain-Barre syndrome in adults and children.

At the same time as specific treatment, the patient is carefully cared for, including the prevention of bedsores, pneumonia, and contractures. Treatment of co-infections is often required. Venous thrombosis is prevented, tube feeding is performed, and excretory function is monitored. Bedridden patients undergo passive exercises, as well as early verticalization to avoid circulatory compromise. If there is a risk of developing contact (joint immobility), paraffin procedures are possible. If necessary, motor simulators based on biofeedback are used.

Patients with damage to the myelin sheath recover faster, while axonal damage requires a longer rehabilitation period. Axonal lesions often leave neurological deficits that are difficult to repair.

PREVENTION

The main method is the complete treatment of infections that we consider common and common. Guillain-Barre syndrome often develops with a slight weakening of the immune system, which is possible for everyone.
The easiest way to protect yourself is to check your current immune status. It only takes a few days, and detected abnormalities can be treated in time.

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