FEATURES OF ELECTROPHYSIOLOGICAL METHODS FOR GUILLAIN–BARRÉ SYNDROME

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Abstract. *2/3 of patients have symptoms of acute respiratory viral infections or gastrointestinal disorders. The first symptoms of GBS are paresthesia of the toes, followed by progressive weakness of the muscles of the lower extremities and impaired walking. The disease progresses for several hours or days, weakness of the upper limbs and cranial nerve palsy develop. Paralysis is usually symmetrical and, of course, peripheral in nature. Pain may be the initial complaint in half of patients, which makes diagnosis difficult. Ataxia and pain are more common in children than in adults. Urinary retention is observed in 10-15% of patients. Damage to the autonomic nerves is manifested by dizziness, hypertension, excessive sweating and tachycardia.*
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Complications of Guillain-Barre syndrome. Patients with GBS are at risk of life-threatening respiratory complications and autonomic disorders.

Instructions for transfer to the intensive care unit are as follows:

- Rapid development of motor weakness with damage to the respiratory muscles;
- Ventilation respiratory failure;
- Bulbar disorders;
- Severe autonomic failure.

Treatment complications requiring resuscitation include fluid overload, intravenous immunoglobulin anaphylaxis, or hemodynamic disturbances during plasmapheresis. 15-25% of children with GBS develop decompensated respiratory failure, requiring mechanical ventilation. Respiratory failure often occurs in children with rapid disease progression, upper limb weakness, autonomic dysfunction, and cranial nerve involvement. Tracheal intubation may be required in patients to protect the airways and mechanical ventilation. In GBS, rapid progression, bilateral facial palsy, and autonomic dysfunction increase the likelihood of intubation. Early intubation planning is essential to minimize the risk of complications and the need for emergency intubation. Autonomic dysfunction increases the risk of endotracheal intubation. On the other hand, dysautonomia may increase the risk of hemodynamic reactions to drugs used to induce anesthesia during intubation.

- Signs indicating the need for mechanical ventilation:
- Sentinel respiratory failure;
- Increased oxygen demand to maintain SpO2 above 92%;
- Signs of alveolar hypoventilation (PCO2 above 50 mm Hg);
- Rapid decrease in vitality by 50% compared to the initial level;
- Inability to cough;

Autonomic dysfunction is a major cause of death in GBS. Fatal cardiovascular collapse due to autonomic dysfunction occurs in 2%-10% of critically ill patients. Heart rate, blood pressure, and electrocardiogram monitoring should continue as long as patients require respiratory support. Transcutaneous pacing may be required for severe bradycardia. Hypotension is corrected by replenishing circulating blood volume (CBV), and if the patient does not respond to CBV replacement, α-agonists such as norepinephrine, mesa tone, epinephrine are used.

In unstable hemodynamics, it is necessary to continuously record arterial and central venous pressure to control the volume of infusion therapy. Arterial hypertension may occur, but this complication does not require special treatment unless it is complicated by pulmonary edema, encephalopathy, or subarachnoid hemorrhage.

- Diagnosis of Guillain-Barré syndrome
- Instrumental diagnostics
- Lumbar puncture
- Neurofunctional diagnosis

ENMG (Electroneuromyography) is the only instrumental diagnostic method that allows confirming the diagnosis of GBS and determining the nature of pathological changes (demyelinating or axonal) and their distribution. Needle electromyography is characterized by the presence of signs of the current denervation-reinnervation process in polyneuropathy. Check the distal muscles of the upper and lower extremities (e.g., tibialis anterior, extensor digitorum general) and, if necessary, the proximal muscles (e.g., quadriceps femoris).

ENMG research in patients with GBS depends on clinical manifestations:

with distal paresis, the long nerves in the arms and legs are examined: at least four motor and four sensory (motor and sensory parts of the median and ulnar nerves; peroneal, tibial, superficial peroneal and sural nerves on the one hand).

Evaluation of the main parameters of ENMG:

motor responses (distal latency, amplitude, shape and duration), the presence of conduction blocks and the spread of responses; the speed of propagation of the excitation along the motor fibers in the distal and proximal areas is analyzed. Sensory responses: amplitude and speed of excitation along sensory fibers in distal regions.

Late ENMG events (F-waves): latency, shape and amplitude of responses, chronodispersion value, dropout percentage are analyzed.

With proximal paresis, it is mandatory to study two short nerves (axillary, musculo-cutaneous, femoral, etc.) with evaluation of motor response parameters (delay, amplitude, form). The first signs of the denervation process appear two to three weeks after the onset of the disease, and the signs of the reinnervation process appear after a month.

Treatment of Guillain-Barre syndrome

Patients requiring intensive care require serious general care. Constipation occurs in more than 50% of patients with GBS as a result of dynamic bowel obstruction.

Paracetamol is used for pain. Catadolone and tramadol are used for severe pain syndrome. Carbamazepine and gabapentin are effective for neuropathic pain.

Various immunomodulatory therapies are used in the treatment of GBS.

Intravenous immunoglobulin is administered at a dose of 0.4 g/kg per day for 5 days during the first 2 weeks of the disease. A second course of immunoglobulin may be required in 5%-10% of patients, with a negative trend after initial improvement. The mechanism of action of

intravenous immunoglobulin is probably multifactorial and includes modulation of complement activation, neutralization of idiotypic antibodies, suppression of inflammatory mediators (cytokines, chemokines).

Side effects of immunoglobulin include headache, myalgia and arthralgia, flu-like symptoms, and fever. Patients with IgA deficiency may develop anaphylaxis after the first course of intravenous immunoglobulin. The combination of plasmapheresis and immunoglobulin did not provide clinical benefit. Corticosteroids should not be used in the treatment of GBS because they do not accelerate recovery, reduce the likelihood of mechanical ventilation, and do not affect long-term outcome.

Causes of the disease

Scientists from different countries have been studying the syndrome for more than 100 years, but they still cannot determine the exact causes that caused the onset of the disease.

It is believed that the appearance and development of the anomaly is caused by the malfunctioning of the patient's immune system. When a person is completely healthy, when foreign cells enter the body, the immune system begins to fight the infection, rejecting all its dangerous elements. The patient is recovering. With GBS, the body begins to confuse "friends and enemies": the patient's neurons are perceived as alien and are "attacked". There is a disorder of the nervous system - a syndrome appears. It is not fully known what disorders there are in the work of the immune system itself. The most common reasons include:

Traumatic brain injury. A strong blow to the head, any damage to it, as well as swelling, swelling or bleeding in the brain can become the main factor in the development of the syndrome. Therefore, when the patient contacts a specialist, first of all, the doctor should determine the presence of any craniocerebral injuries.

Infections. Recent viral infections significantly weaken a person's immune system, thereby increasing the likelihood of GBS. The body's defense mechanism perceives the neurons as an infection and proceeds to kill them with the help of white blood cells. In this case, the syndrome manifests itself one to three weeks after the infectious disease.

Allergy. The disease often develops in allergic people, for example, after chemotherapy, vaccination against poliomyelitis and diphtheria, or after major surgery.

Genetic predisposition. Most diseases are hereditary, and Guillain-Barre syndrome is no exception. If someone in the family has already suffered from the pathology, then it is likely that it will also appear in the offspring. In this case, you should especially monitor your health: take care of your head and try not to start infectious diseases.

The following studies should also be conducted:

- General analysis of blood and urine;
- blood chemistry;
- Serological and virological tests;
- Examination of cerebrospinal fluid;
- Magnetic resonance imaging;
- electrocardiography;
- Registration of electrical activity of muscles;
- x-ray or ultrasound examination of the affected area;
- Examination of external breathing;

Study of basic vital signs.

Muscle weakness and tendon areflexia in several limbs at the same time can be another obvious symptom of Guillain-Barré syndrome. It also includes various pelvic disorders, polymorphonuclear leukocytes, paresis asymmetries, and sensory disturbances.

Differential diagnosis

Although the symptoms of GBS are similar to many other diseases (diphtheria, porphyria, transverse myelitis, botulism, and myasthenia gravis), they still need to be differentiated for proper treatment. The following factors should be taken into account in the differential diagnosis:

If poliomyelitis is suspected, it is necessary to collect data from an epidemiological study, take into account the symptoms of the gastrointestinal tract, high cytosis in the cerebrospinal fluid, the asymmetry of the lesion and the absence of sensory disturbances. The diagnosis can be confirmed by serological or virological analyses. Polyneuropathy is characterized by the appearance of psychopathological symptoms, as well as pain in the pelvis and abdomen. Abnormalities of the main indicators in the urine also indicate the development of the disease. Transverse myelitis is accompanied by dysfunction of the pelvic organs, without damage to the cranial nerves. Abnormal symptoms can be confused with cerebral infarction. But in this case, the pathology affects the body in a few minutes and often leads to coma. MRI helps to determine the exact cause of dysfunction of body systems. Botulism is characterized by the absence of sensory disturbances and any changes in the cerebrospinal fluid.

Treatment

Patients diagnosed with GBS should be hospitalized. In about 30% of cases, mechanical ventilation should be performed. Pathological therapy is carried out in the following stages:

- resuscitation;
- symptomatic;
- Cleansing the blood;
- Preparation;
- Muscle recovery;
- Prophylactic.
- Resuscitation therapy

If the anomaly is in an acute form, resuscitation treatment aimed at relieving symptoms is carried out:

- The patient is connected to an artificial respiration system;
- Use a catheter to remove urine;
- Position the trachea and probe if there are problems with swallowing.
- Symptomatic therapy
- This type of treatment is carried out using different drugs:
- Antihypertensive drugs: Anaprilin, Metaprolol;
- Antibiotic therapy: "Norfloxacin";
- Medicines that help stabilize heart rate and pressure: Propranolol, Anaprilin (with tachycardia), Piracetam (with bradycardia);
- Low molecular weight heparin: "Hemapaxan", "Certoparin";
- Painkillers - NSAIDs or Gabapentin, Pregabalin;
- Antipyretic, when the temperature exceeds 38 degrees: "Ibuklin", "Next";
- Laxatives: Bisacodyl, Laxatin.

Plasmapheresis

One of the most effective procedures for the treatment of GBS is plasmapheresis. It helps stop autoimmune processes in the body. It is indicated for severe and moderate course of the disease. Usually, about 4-6 operations are performed with a one-day break. Instead of plasma, a special isotonic solution of sodium or albumin is injected into the blood, which cleans the blood and normalizes the work of all body systems.

Surgical treatment of the syndrome

If mechanical ventilation was carried out for more than 7-10 days, a tracheostomy should be used - a throat for artificial respiration. In severe cases, a gastrostomy may also be required - a surgically created opening in the stomach to feed the patient.

In conclusion, from what has been analyzed from the abovementioned details it can be inferred that the first symptoms of GBS are paresthesia of the toes, followed by progressive weakness of the muscles of the lower extremities and impaired walking. The disease progresses for several hours or days, weakness of the upper limbs and cranial nerve palsy develop. Paralysis is usually symmetrical and, of course, peripheral in nature. Pain may be the initial complaint in half of patients, which makes diagnosis difficult. Ataxia and pain are more common in children than in adults. Urinary retention is observed in 10-15% of patients. Damage to the autonomic nerves is manifested by dizziness, hypertension, excessive sweating and tachycardia.

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