RISK FACTORS AND DIFFERENTIAL DIAGNOSIS OF VARIOUS VARIANTS OF GUILLAIN-BARRE SYNDROME

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Annotation The lecture discusses the pathogenetic and clinical features of Guillain Barre Syndrome. This disease is considered the most common cause of inflammatory polyneuropathies. The pathogenesis of Guillain Barre Syndrome is based on autoimmune damage to the myelin sheath of peripheral nerves. Probably, both cellular immune mechanisms and humoral ones play a role. Guillain Barre syndrome is most often manifested by ascending paresis in the extremities and distal paresthesias that develop over several days. Guillain Barre syndrome is divided into the following types: the most frequent acute inflammatory demyelinating polyradiculo-neuropathy (the so-called classical variant), and more rare - acute motor axonal neuropathy and acute sensorimotor axonal neuropathy.

Keywords: Guillain-Barre syndrome; diagnosis; differential demyelination.

Guillain Barre syndrome is an inflammatory disease of the peripheral nervous system, which is characterized by the destruction of myelin with lymphocytic and macrocytic infiltration. This disease is considered the most common cause of inflammatory polyneuropathies and occurs with a frequency of 1-3 cases per 100,000 population per year. There is no seasonal variation in morbidity. Guillain Barre syndrome can develop in all age groups (from 2 months to 95 years), although among the patients there is some prevalence of persons aged 15-35 and 50-75 years. Among the patients, men also predominate somewhat (the ratio of men:women 1,1—1,7:1). In women, the risk of this disease decreases during pregnancy and increases after childbirth.

Every year, 0.4-4 new cases of Guillain—Barre syndrome per 100 thousand population are registered in the world [4, 5, 6]. For example, in the Yaroslavl region (as a typical region of our country), the incidence of Guillain—Barre syndrome...
Syndrome accounts for an indicator of 1.8 per 100 thousand inhabitants [7]. Seasonal dynamics of the incidence of Guillain -Barre syndrome was not noted. Guillain—Barre syndrome is most often registered in people aged 15-35 and 50-75 years, although the disease is observed in all age groups (from 2 months to 95 years). Men are more likely to develop Guillain-Barre syndrome (the relative risk for men is 1.1-1.7 compared to women). It is characteristic that the risk of this disease in women decreases during pregnancy, but increases after childbirth [8].

The pathogenesis of Guillain Barre Syndrome is based on autoimmune damage to the myelin sheath of peripheral nerves. Probably, both cellular immune mechanisms and humoral ones play a role. At the onset of the disease, lymphocytic infiltration of the myelin sheath is noted, which leads to segmental demyelination, after a few days infiltration by macrophages prevails. Not only peripheral nerves are affected, similar changes are detected in spinal roots and cranial nerves. As a result of segmental demyelination, the propagation of excitation along the nerve is disrupted, in more severe and rapidly progressing cases, axon damage develops by the mechanism of Wallerian degeneration.

Etiopathogenesis

An immunopathological reaction in the form of autoimmune inflammation leads to demyelination and then to secondary axonal damage to peripheral nerve fibers. Sometimes there is a primary simultaneous lesion of myelin and axons. The autoimmune reaction is triggered after an immuno-activating event. Taking into account a wide range of ganglioses, each of which to a certain extent has its "favorite" localization in the peripheral nervous system, clinical patterns of the disease are determined. In particular, it was found that GM1 gangliosides are located in the myelin sheath of the paranodal locus of the anterior roots, and the detection of antibodies to GM1 ganglioside is associated with the development of an acute motor axonal form of Guillain—Barre syndrome [9].

Two thirds of patients with Guillain-Barre syndrome suffered various infectious diseases on the eve of the development of neurological symptoms. Most often these are acute respiratory tract infections or gastroenteritis [4, 6]. Campylobacterjejuni is the predominant etiological factor identified in 25-50% of patients from Asian countries. Other infectious agents associated with the development of Guillain—Barre syndrome are cytomegalovirus, Epstein—Barr viruses, Varicellazoster, Mycoplasmapneumoniae, ECHO, Coxsackie, influenza A [10, 11]. Numerous cases of Guillain -Barre syndrome have been described after surgical treatment, epidural anesthesia. Also in the literature there are such provoking factors as taking heroin and even snake bite. There is a lot of data on post-vaccination Guillain-Barre syndrome, which arose after immunization against swine flu, rabies, diphtheria, whooping cough and tetanus, polio, hepatitis A.

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Contradictory data of iatrogenic provoking factors are described in the literature. Thus, it was believed that patients receiving penicillin were at high risk for Guillain-Barre syndrome, but subsequent scientific work did not confirm this [8]. Recently, research has been actively conducted to establish a predisposition to Guillain—Barre syndrome, as well as to identify immunogenetic factors that increase the likelihood of developing this disease in each individual. Numerous studies have been devoted to establishing a link between the occurrence of Guillain—Barre syndrome and a certain type of HLA (human leukocyte antigens) of the main histocompatibility complex, but reliable data have not been obtained in any of them [12, 13, 14].

**Classification**

Depending on the location of the immunopathological process in the structures of the nerve fiber (myelin sheath or axonal rod), there are three main forms of Guillain—Barre syndrome: acute inflammatory demyelinating polyneuropathy (OVDP) (70-80% of cases worldwide, in Europe and North America before 95%) [6, 15, 16]; acute motor axonal neuropathy (OMAN) (10-15%) and acute motor-sensory axonal neuropathy acute motor axonal neuropathy (up to 5%). Acute motor axonal neuropathy and OMSAN are often considered as a single axonal form, since they have a similar pathomorphological substrate, albeit with varying degrees of severity [17]. In Asia, Central and South America, the proportion of axonal forms is significantly higher than in European countries (up to 30-47% of all cases of the disease) [4, 17]. Other variants of Guillain—Barre syndrome are extremely rare (Miller Fisher syndrome, acute pandisautonomia, pharyngocervico-brachial form, paraparetic, sensory) [4, 15, 18]. The representation of different clinical variants of Guillain-Barre syndrome in the Russian population corresponds to that in Western countries [19].

Depending on the severity of clinical manifestations of Guillain—Barre syndrome, several degrees of severity of the condition are distinguished. There are no or minimally pronounced paresis in the adjacent form, which does not cause significant violations of walking and self-service. With a moderate course, a violation of the function of walking with limited movement or requiring outside help, support is characteristic. In severe cases, the patient is immobilized, inert, needs daily qualified care, and swallowing disorders are often observed. Every fourth to fifth patient with Guillain—Barre syndrome develops an extremely severe degree of the disease, which entails the transfer of the patient to artificial lung ventilation (ventilator) [1, 19, 20]. In clinical practice, there are several scales for assessing the severity of the condition of patients with Guillain-Barre Syndrome. One of such generally recognized international scales is Guillain - Barre Syndrome Disability Score (R. Hughes, 2010), which in Russia is called the
North American Scale of severity of motor deficiency in Guillain-Barre Syndrome (Table 1).

Table 1. Guillain — Barré Syndrome Disability Score, North-American scale (R. Hughes, 2010)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal - no movement disorders</td>
</tr>
<tr>
<td>1</td>
<td>Minimal movement disorders that do not affect performance</td>
</tr>
<tr>
<td>2</td>
<td>Measured movement disorders, in which it is possible to walk 10 m or more without support or support, there are significant restrictions on fine motor skills of the hands</td>
</tr>
<tr>
<td>3</td>
<td>Moderate movement disorders that require support or support to walk and move</td>
</tr>
<tr>
<td>4</td>
<td>Severe movement disorders, tetraplegia, inability to walk with support or support, immobility, &quot;confinement&quot; to a bed or wheelchair</td>
</tr>
<tr>
<td>5</td>
<td>The need for mechanical ventilation</td>
</tr>
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</table>

Symptoms of damage to the facial, oculomotor nerves, as well as the bulbar group of cranial nerves are often associated. In addition to peripheral paresis, patients may initially have sensory disturbances, ataxia, and symptoms of autonomic dysfunction. In the advanced stage of the disease, there is a sharp decrease or absence of deep reflexes. With a severe form of the disease, a gross violation of walking and movement function occurs. In the event of respiratory muscle weakness, 25% of patients develop respiratory failure with the need for mechanical ventilation (ALV) [1, 5].

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The forms of OVDP and OMSAN in the acute period of the disease can be clinically indistinguishable. Only with the help of ENMG it is possible to differentiate between demyelinating and axonal forms of Guillain-Barré Syndrome. Acute motor axonal neuropathy is characterized by minimal or no sensory impairment. The paraparetic form of Guillain-Barré Syndrome is characterized only by the lower flaccid paraparesis without the involvement of the upper limbs in the pathological process. In Miller Fisher syndrome, bilateral relatively symmetric ophthalmoparesis, cerebellar ataxia, and tendon areflexia are observed, which appear by the end of the first week of the disease. Sensory disturbances in the arms and legs are weak or absent; weakness in the oropharyngeal muscles rarely occurs. Cerebellar dysarthria and pyramidal symptoms are absent. During the course of the disease, a moderate severity of flaccid tetraparesis can join these symptoms. Sensory form Guillain-Barré syndrome is manifested only by disorders of superficial and deep sensitivity and tendon areflexia, sometimes accompanied by neuropathic pain syndrome. With this form of weakness in the limbs and bulbar disorders do not occur. With the pharyngo-cervic-brachial form of Guillain-Barré Syndrome, at the onset of the disease, bulbar syndrome, weakness of the muscles of the neck, upper shoulder girdle and arms are observed. Sensory disturbances, the absence of tendon reflexes can be observed only on the upper limbs, while no changes in the lower limbs are observed [1, 8, 21].

**Diagnostics**

For the diagnosis of Guillain-Barré Syndrome, in addition to a thorough analysis of clinical and anamnestic data, additional paraclinical signs are required. The clinical criteria for Guillain-Barré Syndrome include acute or subacute developing progressive muscle weakness in more than one limb, suppression of reflexes of varying degrees, relative symmetry of symptoms, absence of gross sensory disturbances, weakness of the cranial muscles (due to the involvement of the facial nerves and nerves of the bulbar group), signs of autonomic dysfunction.
Febrile syndrome at the onset of the disease is not characteristic of Guillain-Barré Syndrome [21, 22].

In order to verify the diagnosis of Guillain-Barré Syndrome, cerebrospinal fluid analysis and electroneuromyographic study (ENMG) are performed. By the end of the 1st - the beginning of the 2nd week of the disease, the key CSF syndrome is detected in the cerebrospinal fluid - albumin-cytological dissociation: the protein level exceeds 0.55 g / l with normal or slightly increased cytosis, but usually no more than 10 cells per 1 μl. In the first days of the disease, the amount of protein in the majority of patients remains within the reference values. From 10-14 days of illness, albumin-cytological dissociation is found in more than 80% of patients. Cytosis, accompanied by an increase in cells of more than 50 per 1 μL and / or the presence of polymorphonuclear leukocytes in the cerebrospinal fluid, suggests another cause, for example, neuroborreliosis, HIV infection, etc. [8, 21].

More information about this source For more information, enter the source to send the comment Side Panels Serum immunoblot analysis detects antibodies to GM1 and GD1b gangliosides. The presence of antibodies to gangliosides in the blood is more often found in patients with axonal forms of Guillain-Barré Syndrome, which can serve as a laboratory differential diagnostic criterion [13].

ENMG serves not only to diagnose Guillain-Barré Syndrome and determine the subtype of the disease, but also to exclude other causes of acute muscle weakness [3, 23]. The electrophysiological patterns of Guillain-Barré Syndrome are the disappearance of the F-wave, a decrease in the magnitude and change in the latency of the M-response. The sensitivity of this paraclinical method is 85% [1]. The scope of ENMG examination in patients with Guillain-Barré Syndrome should include examination of at least 5 motor and 5 sensory nerves in the upper and lower extremities. Assessment of the parameters of late neurographic responses (4w and Fw) is mandatory, especially in the early stages of the disease In the case of proximal paresis, it is necessary to examine at least one or two short nerves (axillary, musculocutaneous, femoral). Currently, the neurophysiological criteria for Guillain-Barré Syndrome proposed by R. Haddenetal are used. in 1998 (Table 2) [24]. It should be emphasized that ENMG certainly helps in the diagnosis of Guillain-Barré Syndrome, but it is still an additional research method. Clinical and anamnestic data remain leading in the diagnosis of Guillain-Barré Syndrome [25].

As auxiliary diagnostic methods, it is possible to use various methods for assessing the vegetative sphere, for example, the study of heart rate variability, blink reflex [8].

In accordance with the international criteria of the WHO (from 1993), there are signs necessary for the diagnosis of Guillain-Barré syndrome, signs that
support the diagnosis, and signs that raise doubts about the diagnosis. The above criteria can be used to verify such forms of Guillain-Barré Syndrome as AIDP, acute motor axonal neuropathy, OMSAN, paraparetic and pharyngo-cervico-brachial forms. Due to the significant clinical features of Miller Fisher syndrome and acute pandizautonomy, the diagnosis of these forms is based mainly on clinical and anamnestic data, and not on the generally accepted international criteria for Guillain – Barré Syndrome [14].

Most often, Guillain-Barré Syndrome is manifested by ascending paresis in the limbs and distal paresthesias that develop over several days. However, in practice, there are other, atypical, very peculiar variants of the course of this syndrome, including a form with predominant damage to axons. The differences between these forms are associated not only with clinical and electrophysiological features, but also with etiopathogenetic mechanisms. Guillain Barré syndrome is divided into the following types: the most common - acute inflammatory demyelinating polyradiculoneuropathy - (the so-called classic variant) and more rare - acute axonal motor neuropathy (more common in Japan, China and developing countries) and acute sensorimotor axonal neuropathy. A special form of Guillain-Barré Syndrome is Miller Fisher Syndrome.

Among other, significantly rarer variants of Guillain-Barré Syndrome (in total, they account for about 10% of all cases of Guillain-Barré Syndrome), the paraparetic variant, pharyngeal-cervico-brachial variant, facial diplegia with distal paresthesias and acute pandisautonomy should be mentioned. The presence of an isolated sensory variant of Guillain-Barré Syndrome remains a matter of debate. The relationship of these atypical variants with Guillain’s Syndrome is indicated by a similar nature of changes in cerebrospinal fluid and EMG results, indicating the involvement of peripheral nerves in the demyelinating process.

**Differential diagnosis of Guillain Barré Syndrome**

In addition to acute transverse myelitis, difficulties in the differential diagnosis of Guillain-Barré Syndrome may arise during myasthenic crisis (it is necessary to take into account anamnestic data, the presence of transient ptosis and other oculomotor disorders, the severity of which depends on physical activity) and botulism (which is characterized by the presence of dilated pupils that do not respond to light, constipation and the appearance of neurological symptoms within 12-36 hours after eating infected foods). The development of paresis of the facial muscles, including facial diplegia, is possible with neuroborreliosis. Among other diseases that may be accompanied by symptoms similar to Guillain-Barré's Syndrome, polyneuropathies of a different genesis (with vasculitis, porphyria, paraneoplastic processes), acute polymyositis and acute steroid myopathy, and in rare cases, poisoning with heavy metals and organophosphorus compounds, should
be mentioned. (with parenteral nutrition, withdrawal symptoms in alcoholics and some other conditions, tetraparesis, characterized by hyporeflexia, can rapidly develop, which also quickly regresses when phosphate levels are restored). In resuscitation patients with sepsis and multi-organ failure, it is possible to develop axonal polyneuropathy (polyneuropathy in critical conditions).

It is not typical for Guillain-Barré Syndrome to maintain asymmetry in muscle weakness with a significant degree of paresis (in these cases, vasculitis should be assumed first of all), clear boundaries of sensory disturbances, or pronounced pelvic disorders at the onset of the disease, which is characteristic of spinal lesions.

**Paraclinical studies in Guillain-Barré Syndrome**

For the diagnosis of Guillain-Barré Syndrome, in addition to a thorough analysis of anamnestic and clinical data, the results of CSF examination and data from electrophysiological research methods are important. In CSF, pleocytosis is usually absent, however, in about 10% of patients, an increase in the number of cells up to 11-50 in 1 μl can be detected. If 50 cells / μL are exceeded, it is necessary to look for another cause of the disease. In these cases, differential diagnosis of Guillain-Barré Syndrome with neuroborreliosis, neurosarcoidosis, poliomyelitis, neuro AIDS and a number of other diseases is required.

More significant for the diagnosis of Guillain-Barré Syndrome is an increase in protein levels (more than 0.55 g / l), which develops within 1 week from the onset of the disease and reaches a peak at 3-4 weeks. In the first 2 days, in 85% of patients, the protein level is within the normal range, by the end of the 1st week of the disease, an increase in the protein level is detected in 2/3 of patients, and in the 2nd week - in 80% of patients. The protein level can be an order of magnitude higher than normal values, but the degree of its increase does not correspond to the severity of the clinical picture of the disease, but correlates with the degree of decrease in the rate of propagation of excitation along peripheral nerves according to EMG data. In 10-30% of patients, oligoclonal antibodies are detected in the cerebrospinal fluid.

In blood and urine tests, there are no specific changes, however, a slight increase in ESR, an increase in liver enzymes (in about 10-38% of patients) and mild proteinuria (in 25% of patients) can be detected. Among patients in whom an increase in liver enzymes is detected, an indication of a transferred cytomegalovirus infection is often found.

Electrophysiological diagnostics are especially important in case of suspicion of Guillain-Barré Syndrome. According to EMG data, multiple conduction blocks are revealed, a decrease in the speed of propagation of excitation along peripheral nerves with an increase in latency (more than 150% of
the norm) and varying degrees of severity of denervation. At the onset of the disease, the rate of propagation of excitation can be within the normal range, but its marked decrease at various stages of the disease is noted in 80-90% of patients. Fibrillation potentials and positive acute waves appear at 2–4 weeks of illness and reach a peak at 2–3 months of illness. It should be noted that a pronounced decrease in the rate of propagation of excitation (32 m / s and below) in combination with a recurrent or chronic course is characteristic not of Guillain-Barré’s syndrome, but of chronic inflammatory demyelinating polyradiculoneuropathy. Recurrences in Guillain-Barré Syndrome occur in about 3% of patients. In addition, in 8-16% of cases, a new deterioration develops immediately after improvement or stabilization of the condition.

Guillain-Barré syndrome in most cases is characterized by a monophasic course. In 98% of patients, the onset of the plateau phase is noted within 4 weeks from the onset of the disease, the average duration of this phase is 12 days, and then there is a slow recovery. The progression of the disease beyond these time boundaries indicates either an exacerbation of the disease or the presence of chronic inflammatory demyelinating polyradiculoneuropathy. Recurrences in Guillain-Barré Syndrome occur in about 3% of patients. In addition, in 8-16% of cases, a new deterioration develops immediately after improvement or stabilization of the condition.

The clinical manifestations of the recurrent variant of Guillain-Barré Syndrome are no different from the clinical manifestations of the monophasic form of the disease. Each of the episodes of exacerbation is manifested by the rapid development of a neurological defect over several days, followed by complete or almost complete recovery. In patients who have had several episodes of exacerbations, peripheral nerves become palpably enlarged.

In principle, it is difficult to draw a clear line between the recurrent course of Guillain-Barré Syndrome and chronic inflammatory demyelinating polyradiculoneuropathy, however, the diagnosis of Guillain-Barré Syndrome becomes problematic if the patient starts to deteriorate again after 8 weeks of illness or 3 or more exacerbations are noted.

The prognosis for Guillain-Barré Syndrome is very variable - from complete and rapid recovery to slow, with severe residual symptoms and disability. It is determined by the degree of segmental demyelination and axonal damage. In general, the recovery period takes up to 1.5-2 years, after this period, the likelihood of improvement in the lost functions is extremely low. However, in most patients - in more than 75-80% of cases within these time limits, recovery occurs in full, or a minimal motor or sensory neurological defect remains. Restoration of autonomic disorders occurs in parallel with improvements in the motor and sensory spheres,

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while residual autonomic symptoms are usually not observed.

With increasing age, the recovery process is worse (this is especially true for people in the age group from 40 to 60 years). Predictors of incomplete recovery, in addition to mechanical ventilation in the acute phase, are the rapid progression of the disease, a significant movement defect, total areflexia in the acute phase, diarrhea in the prodromal period, as well as signs of pronounced axonal damage according to EMG data. Children recover more rapidly than adults, but residual symptoms are observed in about a third of cases. This is mainly weakness in the feet, pescavus deformity of the feet, as well as tremors.

Mortality in Guillain-Barré Syndrome ranges from 3 to 10-15%, while the rest of the patients (mainly those patients who required mechanical ventilation) retain residual neurological symptoms in the form of severe dysesthesias or moderate/severe weakness in the distal legs, manifested by gait disorders... Electromyographic predictors of poor prognosis are signs of severe axonal damage and the presence of pronounced spontaneous activity. The cause of death may be cardiac arrest, pulmonary embolism, sepsis, pneumonia, bronchospasm, pneumothorax, acute respiratory distress syndrome, autonomic failure, as well as a sharp drop in blood pressure provoked by iatrogenic effects.

**Treatment**

Guillain-Barré syndrome in the progression phase, even with a mild course, is considered an emergency requiring emergency hospitalization. Within a few hours, a formidable complication is possible - decompensation of the state with the development of severe respiratory failure requiring mechanical ventilation [21, 26].

Specific and non-specific therapeutic measures for Guillain-Barré syndrome are distinguished. Specific treatment methods, which include high-volume programmed plasmapheresis (PF) operations and high-dose intravenous immunotherapy with class G immunoglobulins (intravenous immunoglobulin), are used primarily to interrupt autoimmune inflammation. In this case, it is supposed to achieve inhibition of the further development of the disease, reduce the duration of the period of increase in symptoms, accelerate the beginning of the recovery period and achieve the most complete regression of neurological deficit. Non-specific measures include symptomatic therapy and prevention of complications specific to Guillain-Barré Syndrome and characteristic of immobilized patients [1, 21, 26].

Specific therapy for Guillain-Barré Syndrome is carried out with the progression of neurological symptoms (especially during the first two weeks from the onset of the disease), the presence of a motor deficit corresponding to 3-5 stages of the Guillain-Barre Syndrome severity scale (Guillain - Barre Syndrome Disability Score (R. Hughes, 2010)), as well as in an episode of repeated deterioration after a temporary improvement or stabilization of the condition (regardless of previous
PF is carried out according to the following scheme: 3-5 sessions with plasma exchange of approximately one volume of plasma (at least 35-50 ml / kg) every 1-2 days for 7-14 days. Removal and replacement of the patient's plasma should be 200-250 ml / kg for the entire course. The removed plasma volume is replaced with 5% albumin. Contraindications to PF are anemia, thrombocytopenia, hypofibrinogenemia, erosive and ulcerative lesions of the gastrointestinal tract, recurrent hemorrhoids, menstruation, coagulopathy, as well as any other conditions that contribute to the development of hemorrhagic complications.

Treatment with intravenous immunoglobulins of class G is carried out according to the same indications as when performing sessions of high-volume programmed PF. The standard course of therapy is intravenous administration of a human immunoglobulin preparation at a dose of 0.4 g / kg of the patient's body weight per day every day for 5 days (2 g / kg of body weight per course). The effectiveness of intravenous immunotherapy in Guillain-Barré Syndrome is comparable to the action of programmed PF, which can serve as an alternative to the latter. There are categories of patients for whom immunotherapy is preferable to programmed PF sessions - these are elderly people, children, as well as patients who have contraindications for its implementation (erosive and ulcerative process in the stomach and duodenum, exacerbation of hemorrhoids, etc.)

The choice between PF and immunotherapy is influenced by many factors, among which two main ones should be highlighted: the possible risk associated with the occurrence of side effects when using a particular method of treatment for Guillain-Barré Syndrome, and the technical equipment of the hospital. The latter assumes the presence in the intensive care and intensive care units of equipment for the programmed PF, as well as the use of intravenous human immunoglobulin preparations with a high concentration (> 95% IgG).

Currently, in many countries of the world, glucocorticosteroids are excluded from the treatment protocols for Guillain-Barré Syndrome. The lack of a positive effect of glucocorticosteroids on the course of the disease was shown in a meta-analysis of six large studies, which involved 587 patients. In many countries of the world, glucocorticosteroid therapy for Guillain-Barré Syndrome is recognized as a serious medical error. This statement applies equally to cytostatic drugs. You should not simultaneously combine PF with glucocorticosteroids, since the effectiveness of therapy for Guillain-Barré Syndrome in this case decreases. Also, to date, there is no evidence of the effectiveness of the combined use of class G immunoglobulins and glucocorticosteroids.

Non-specific methods include a program of qualified care for severe patients: prevention of pressure sores, care of the skin and mucous membranes, hygienic treatment of the mouth and nasopharynx; drug and non-drug prevention of venous
thromboembolic complications, sanitation of the tracheobronchial tree (during mechanical ventilation); prevention and timely adequate correction of secondary infectious complications, control of water balance with the introduction of a sufficient volume of fluid and nutritional support for patients (a nasogastric tube can be inserted); monitoring the state of the functions of the pelvic organs; passive gymnastics and daily massage; correction of pain syndrome, as well as psychological support [1, 14, 21, 26].

**Course and prognosis**

Guillain-Barré syndrome in most cases is characterized by a monophasic course. The duration of the development of symptoms of the disease for more than 4 weeks indicates either an exacerbation of the disease, or the presence of chronic inflammatory demyelinating polyradiculoneuropathy.

The severity of segmental demyelination and axonal degeneration is directly related to the outcome of the Guillain-Barré Syndrome. It is possible both complete and quick recovery, and protracted, with pronounced residual neurological defect and disability. In general, the stage of reverse development of symptoms takes up to 1.5-2 years, after which the prospects for the restoration of lost functions, rehabilitation potential are minimal. In most patients, complete recovery occurs during these periods, or mild residual motor and / or sensory manifestations remain. The restoration of autonomic functions is complete and usually proceeds simultaneously with improvement in the motor and sensory areas. Persistent residual neurological symptoms are observed in 7-15% of patients [18, 33, 34]. Prognostically unfavorable functional prognosis is more common in patients over 60 years of age, with a rapidly progressive course of the disease, as well as with a detected ENMG pattern - a decrease in the amplitude of the distal M-response <1 mV (which indicates axonal damage).

Despite the monophasic nature of Guillain-Barré Syndrome, a recurrent course is observed in 3-5% of cases [1, 35].

In 2007 R. VanKoningsveldetal. developed a prognostic scale for the outcome of the course of Guillain – Barré Syndrome Erasmus Guillain – Barresyndrome outcomescore (EGOS) and its modified version mEGOS [36]. The EGOS scale already at an early stage of the disease with a high degree of probability allows predicting the restoration of the function of independent walking by 4 weeks, 3rd and 6th months. The EGOS scale takes into account the patient's age, the presence / absence of previous diarrhea, and the total muscle strength score. A high final score calculated on this scale indicates a significant likelihood

**REFERENCES**

1. Bakhodirovna, Azizova Rano. "Interrelation of P300 cognitive potentials and..."
neuro-immunologic values of patients with idiopathic and symptomatic epilepsy." European science review 7-8 (2014).


