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Clinical Features of Guillain—Barre Syndrome

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Abstract
Guillain—Barre syndrome (GBS) is a disease believed to be a more frequent factor of inflammatory polyneuropathies. The pathogenesis of GBS is based on autoimmune damage to the myelin sheath of peripheral nerves. Probably, both cellular immune mechanisms and humoral ones play an important role. Most often, GBS is manifested by ascending paresis in the extremities and distal paresthesias that develop over many days. GBS is divided into the following types: more frequent — acute inflammatory demyelinating polyradiculoneuropathy (the so—called classic variant), and rarer - acute motor axonal neuropathy (more common in Japan, China and developing countries) and acute sensorimotor axonal neuropathy. A special form of GBS is Miller Fisher syndrome. In the diagnosis of GBS, the leading importance is attached to clinical evaluation, EMG results and examination of cerebrospinal fluid.

Keywords: Guillain—Barre syndrome; clinical features, Miller Fisher syndrome.

Guillain–Barre syndrome is an acute inflammatory demyelinating polyradiculoneuropathy of autoimmune etiology, characterized by peripheral paralysis and in most cases protein-cell dissociation in the cerebrospinal fluid. The incidence of Guillain–Barre syndrome is 0.6–2.4 episodes per 100 thousand. Currently, four key clinical types of Guillain–Barre syndrome are presented: acute inflammatory demyelinating polyradiculoneuropathy, axonal form, acute motor axonal disease and Miller–Fisher syndrome. The formation of the disease is preceded by a connection with the causative agent of a viral or bacterial infection, say Campylobacter jejuni, Mycoplasma pneumonia, cytomegalovirus, Epstein–Barr virus and influenza virus. The pathogenesis of Guillain–Barre syndrome is based on "molecular mimicry" between infectious agents on its plane and structures of peripheral nerves. Elevated titer of antibodies to GM1, GD1a, GD1b and GQ1b gangliosides is shown in the blood serum of patients. Diagnostic aspects of the diagnosis of Guillain–Barre syndrome are examination material, cerebrospinal fluid examination and

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electroneuromyographic examination. There is no seasonal variation in the incidence. GBS can form in all age groups (from 2 months to 95 years), however, among the patients there is a certain dominance of persons aged 15-35 and 50-75 years. Among the patients, men also prevail somewhat (the ratio of men: women is 1.1—1.7: In women, the risk of the origin of this disease will decrease during pregnancy and increases after childbirth. The pathogenesis of GBS 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 noticed, which leads to segmental demyelination, after a few days infiltration by macrophages prevails. Not only peripheral nerves are affected, similar changes are found in spinal roots and cranial nerves. As a result of segmental demyelination, the spread of excitation along the nerve is disrupted, in more severe and rapidly progressing cases; axon damage develops by the mechanism of Wallerian degeneration. Most often, GBS is manifested by ascending paresis in the extremities and distal paresthesia, developing over several days. However, in practice, there are other, atypical, very peculiar types of the course of this syndrome, including a form with predominant axon damage. the differences in these forms are associated not only with clinical and electrophysiological features, but also with etiopathogenetic mechanisms. GBS is divided into the following types: the most common is acute inflammatory demyelinating polyradiculoneuropathy — OVDP (the so—called classic variant) and more rare are acute motor axonal neuropathy (more common in Japan, China and developing countries) and acute sensorimotor axonal neuropathy. a special form of GBS is Miller Fisher syndrome. Among others, there are significantly more rare variants of GBS (in total they account for about 10% of all cases of GBS), the paraparetic variant, the pharyngeal-cervico-brachial variant, facial diplegia with distal paresthesia and acute pandisautonomia should be listed. The presence of a separate sensory type of GBS remains an object of discussion. The connection of these atypical variants with GBS is indicated by the similar nature of changes in the cerebrospinal fluid (CSF) and EMG results certifying the involvement of peripheral nerves in the demyelinating course. Acute inflammatory demyelinating polyradiculoneuropathy (OVDP) despite the fact that this disease is sporadic, in about 2/3 of cases, the phenomenon of neurological disorders is preceded by an infectious process, mainly viral (cytomegalovirus infection, Epstein—Barr virus, influenza, herpes simplex virus, hepatitis A and C, HIV, enteroviruses), less often — mycoplasma or bacterial (Campylobacter jejuni).

In 40% of cases, it is a respiratory infection, in 20% — gastrointestinal disorders (mainly diarrhea), in addition of a contagious nature. As a rule, these are more young patients, and in the clinical picture they often have sensory disturbances and damage to cranial nerves. in this group of patients, the disease is more severe, with respiratory disorders (in 65%), and recovery is more protracted and sometimes not so significant. In patients who have undergone an infectious process caused by Campylobacter jejuni, acute motor axonal neuropathy appears more often, and antibodies to Gm1

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gangliosides are detected during examination. However, antibodies to Gm1- and other gangliosides are found in 20-40% of patients with GBS. Since Campylobacter jejuni lipopolysaccharides have a structure similar to Gm1 gangliosides, it is possible that the pathogenesis of the disease in these cases is based on the mechanism of molecular mimicry between the neural tissue and the infectious agent. In children, there was a link between GBS and infection with Mycoplasma pneumoniae. The occurrence of GBS can also be preceded by vaccination against rabies, influenza, measles, mumps, rubella, oral vaccination against polio, surgical interventions, kidney transplantation, epidural anesthesia, thrombolytic therapy, the use of heroin and even a snake bite. In extremely rare cases, the origin of GBS has been recorded in patients with toxoplasmosis and malaria. As for iatrogenies in the property of activating factors, the available initial ones are contradictory. previously, it was reported that among patients with GBS there were more people who received penicillin and fewer women who took oral contraceptives, however, further studies did not confirm this. the key clinical manifestations of OVDP are contained in muscular depression in the extremities, as a rule, invariant (at the very beginning of the disease, paresis may be asymmetrical), predetermined by peripheral nerve damage, which develops within 5-10 days, as well as sensory symptoms, usually not rough. Sensory disturbances in the form of mild paresthesia in the distal extremities or pain in the extremities or back may precede the phenomenon of motor disorders for 1-2 days. At the same time, in patients, these disorders can be misinterpreted as "hysterical", and only then the phenomena of other (motor, bulbar) symptoms are diagnosed with OVDP. Paresthesia as an early sign of the disease is observed in 50% of patients, and as it progresses — in 70-90% of patients. In children at their debut, symptoms of damage to the central nervous system (headache, mental disorders) may occur. Tetraparesis of varying severity develops in 30-60% of patients with OVDP. It should be noted that despite the fact that patients often complain only of weakness in the legs, a thorough clinical examination reveals weakness in the hands. the increase in muscle weakness stops after 2 weeks of the disease in 50% of patients, after 3 weeks — in 80% and after 4 weeks — in 90% of patients. next comes the plateau stage. the resumption in the form of muscle strength growth occurs after 1-4 weeks after the plateau period. Usually, motor symptoms at the beginning of the disease are noted in the legs, however, a debut from the hands or face is likely. The severity of paresis is very variable — from mild weakness with minimal walking disorders to plegia and damage to the respiratory muscles, which can lead to death due to respiratory insufficiency. the performance of artificial lung ventilation (ventilator) is required in the acute period by almost a third of patients. Paresis is both proximal and distal in nature, however, the proximal parts usually suffer more in the hands. almost half of the patients have some degree of facial muscle weakness, and facial diplegia is more characteristic for the early period of the disease, and unilateral facial nerve damage is more characteristic for the late period. In addition to the lesion of the facial nerve, the oculomotor nerves may be affected in about 5% of cases. In rare cases (also about 5%), in which a separated lesion of the facial nerve is present at

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the onset of the disease, weakness in the extremities develops in the future. from case to case, the bulbar group of cranial nerves is clinically clearly involved in OVDP, while the risk of progression with the development of respiratory disorders is high. It should be noted that an isolated violation of the respiratory muscles without involving the muscles of the extremities and trunk, with OVDP does not occur. Among other symptoms, it is worth mentioning the pains observed in 30-50% of patients with OVDP, and in 15% they are pronounced. The intense nature of the pain syndrome, especially in the onset of the disease, often makes it difficult to diagnose correctly, since it forces us to look for a different cause of the disease, different from the OVDP. With OVDP, pains have different origins — neuropathic, muscular and radiculopathic. The loss of sensitivity of all modalities is characterized by a significant variation in its severity, in some cases they lead to ataxia, which can be mistakenly regarded as cerebellar in origin. At the beginning of the disease, loss of sensitivity is rare, symptoms of irritation — dysesthesia and paresthesia, which are ascending in nature as the disease progresses, are much more common. However, a subsequent decrease in sensitivity develops in almost 75% of patients. Usually, in the acute period of the disease, sensory disturbances are more diffuse, but later (during the period of convalescence) they are detected in the distal parts of the extremities, their proximal localization is characteristic of severe variants of the course of the disease. Deep reflexes are usually reduced or absent, preserved reflexes occur in extremely rare cases. However, at the onset of the disease in about 30% of patients, deep reflexes can be preserved. During the 1st week of the disease and in this category of patients, their decrease or disappearance is noted. The occurrence of areflexia is more correlated with a decrease in sensitivity, rather than with muscle weakness. The preservation of deep reflexes throughout the entire period of the disease suggests a diagnosis other than GBS. It should be noted that hyperreflexia may occur in GBS — mainly in people with acute motor axonal form of the disease, especially those with GM1 antibodies - during the recovery period, although isolated cases of hyperreflexia in the acute progressive phase of the disease have been noted. At the same time, the increase in deep reflexes is confirmed by the data of electrophysiological research — an increase in the amplitude of the H-reflex with m.soleus and the appearance of the H-reflex with small muscles of the hands and feet. In 2/3 of cases with OVDP, vegetative disorders are noted, both sympathetic and parasympathetic in nature. These disorders are especially characteristic for patients who are undergoing a ventilator. More than 50% of patients develop sinus tachycardia. Among other autonomic disorders, bradycardia (mainly in intubated patients), orthostatic hypotension (and syncopal states associated with it), fluctuations in blood pressure (more often arterial hypertension, less often arterial hypotension), sweating disorders (despite their frequent occurrence, patients usually do not complain about these disorders) should be mentioned. Approximately 10-20% of patients develop pelvic disorders of a transient nature (more often delay, less often urinary incontinence) due to sphincter disorders. In these cases, difficulties may arise in the differential diagnosis of OVDP and acute transverse myelitis, since both diseases have motor

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disorders, but the latter is characterized by the development of pronounced pelvic disorders in the onset, as well as the conductive nature of sensory disorders. With OVDP, patients with transient episodes of arterial hypertension are very characterized by hypersensitivity to antihypertensive drugs, which must be taken into account when prescribing them. Acute motor axonal neuropathy Acute motor axonal neuropathy occurs in 10-20% of cases of sporadic GBS. Clinically, this variant resembles OVDP, but it is more severe and more often leads to disability. This condition is characterized by a prodromal period with gastrointestinal symptoms (mainly diarrhea), while 2/3 of patients have elevated titers of antibodies to Campylobacter jejuni. In 13% of patients (mainly from Japan) in the prodromal period — 4-12 days before the development of acute motor axonal neuropathy — there is a respiratory infection caused by Haemophilus influenzae. The clinical picture of this variant of GBS is the development of symmetrical muscle weakness, mainly in the distal extremities, in a quarter of cases combined with cranial neuropathy. Sensory disturbances, both clinically obvious (paresthesia, pain) and paraclinical (according to EMG data) are absent. The decrease in deep reflexes is all the more significant the more pronounced motor disorders are, and in some patients, as mentioned above, hyperreflexia may even be detected. The revival of reflexes is detected in a third of patients, usually in the early recovery period and less often in the acute phase of the disease. Hyperreflexia is noted in patients with the presence of Gm1 antibodies and a less severe course of the disease. Symptoms of acute motor axonal neuropathy increase within a few days, reaching its maximum by day 6, and recovery proceeds fairly quickly - a significant recovery of muscle strength is noted after 1-2 months, and it occurs even faster in patients who had an indication of an infection caused by Haemophilus influenzae in the prodromal period. Electrophysiological examination confirms the predominantly axonal nature of peripheral nerve damage with minimal pronounced demyelination. The pathogenesis of this condition is based on the blockade of anti-Gm1- and other antibodies of excitation along peripheral nerves, degeneration of motor terminals without involving the muscles of the limbs and trunk, does not occur with OVDP. Among other symptoms, it is worth mentioning the pains observed in 30-50% of patients with OVDP, and in 15% they are pronounced. The intense nature of the pain syndrome, especially in the onset of the disease, often makes it difficult to diagnose correctly, since it forces us to look for a different cause of the disease, different from the OVDP. With OVDP, pains have different origins — neuropathic, muscular and radiculopathic. The loss of sensitivity of all modalities is characterized by a significant variation in its severity, in some cases they lead to ataxia, which can be mistakenly regarded as cerebellar in origin. At the beginning of the disease, loss of sensitivity is rare, symptoms of irritation — dysesthesia and paresthesia, which are ascending in nature as the disease progresses, are much more common. However, a subsequent decrease in sensitivity develops in almost 75% of patients. Usually, in the acute period of the disease, sensory disturbances are more diffuse, but later (during the period of convalescence) they are detected in the distal parts of the extremities, their proximal localization is

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dysesthesia in the distal extremities and face, a slight decrease in sensitivity), a decrease in muscle strength and pelvic disorders. Among other (besides oculomotor and cranial) nerves, the bulbar nerves (in a quarter of patients) and the facial nerve (in a third of patients) may be involved in the pathological process. According to MRI data, no pathology of the brainstem is detected with this syndrome, during a study with contrast enhancement (gadolinium), cranial nerves accumulating contrast may be visualized in some patients. In CSF, an increase in protein levels is detected with an unchanged number of cells. In most patients with Miller Fisher syndrome, antibodies to ganglioside GQ1b are detected. According to EMG data, damage to the axons of sensitive nerves is noted with the preservation of motor nerves. The progressive increase in symptoms occurs for several days, less often for weeks, and in some patients may be accompanied by the appearance of a pronounced motor defect. Recovery usually takes from 2 weeks to 2 months and in most cases is complete. Acute sensorimotor axonal neuropathy Acute sensorimotor axonal neuropathy is characterized by the onset of the disease with the appearance of pain, paresthesia and dysesthesia in the distal extremities. In the future, a decrease in all types of sensitivity develops in the distal and proximal parts. This symptomatology may be accompanied by non-rough motor and vegetative disorders. Deep reflexes are usually absent. Axonal degeneration is confirmed by both EMG results and nerve biopsy data. Recovery is often incomplete and lasts for a long time. It is possible that in some cases this disease is associated with Campylobacter jejuni and Epstein—Barr virus. The course and prognosis of GBS in most cases is characterized by a monophasic course. In 98% of patients, the onset of the plateau phase is noted after 4 weeks from the onset of the disease, the average duration of this phase is 12 days, and then a slow recovery occurs. The progression of the disease beyond these time limits indicates either an exacerbation of the disease or the presence of CVD. Relapses in GBS occur in about 3% of patients. In addition, in 8-16% of cases, a new deterioration develops immediately after the improvement or stabilization of the condition. The clinical manifestations of the recurrent variant of GBS 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 within a few days, followed by a complete or almost complete recovery. In patients who have had several episodes of exacerbations, peripheral nerves become palpationally enlarged. In principle, it is difficult to draw a clear line between the recurrent variant of the course of GBS and CVD, however, the diagnosis of GBS becomes problematic if the patient begins to deteriorate again after 8 weeks of the disease or 3 exacerbations or more are noted. The prognosis for GBS is very variable — from complete and rapid recovery to slow, with pronounced 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 probability of improving 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 persists. The restoration of

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vegetative disorders occurs in parallel with improvements in the motor and sensory spheres, while residual vegetative symptoms are usually not noted. With increasing age, the recovery process is worse (especially for people in the age group from 40 to 60 years). Predictors of incomplete recovery, in addition to ventilation in the acute phase, are rapid progression of the disease, significant motor defect, and total areflexia in the acute phase, diarrhea in the prodromal period, as well as signs of pronounced axonal damage according to EMG data. In children, recovery occurs at a faster pace than in adults, but residual symptoms are noted in about a third of cases. Basically, this is weakness in the feet, deformation of the feet according to the type of pes cavus, as well as tremor. The mortality rate in GBS is from 3 to 10-15%, and the remaining patients (mainly those patients who needed a ventilator) have residual neurological symptoms in the form of pronounced dysesthesia or moderate / pronounced weakness in the distal parts of the legs, manifested by walking disorders. Electromyographic predictors of an unfavorable 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, vegetative insufficiency, as well as a sharp drop in blood pressure provoked by iatrogenic effects.

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