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GUILLAIN-BARRE SYNDROME: ETIOLOGY, CLINICAL MANIFESTATIONS, MODERN TREATMENT OPTIONS

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Abstract: Guillain - Barre syndrome is an acute inflammatory demyelinating polyradiculoneuropathy of autoimmune etiology, characterized by peripheral paralysis and, in most cases, protein-cell secretion in the cerebrospinal fluid. The incidence of Guillain-Barre syndrome is 0.6-2.4 cases per 100,000 population. The development of the disease is preceded by contact with a viral or bacterial infection of the body, for example, Campylobacter jejuni, Mycoplasma pneumoniae, cytomegalovirus, Epstein-Barr virus and influenza virus. The pathogenesis of Guillain-Barre syndrome is based on the "molecular mimicry" between infectious agents on its surface and the structures of the peripheral nerves. High titers of antibodies to GM1, GD1a, GD1b and GQ1b gangliosides are detected in the serum of patients. Diagnostic criteria for the diagnosis of Guillain-Barre syndrome are examination data, analysis of cerebrospinal fluid and electroneuromyographic study. Plasmapheresis and immunoglobulin G therapy are currently the mainstays of treatment for patients with Guillain-Barré syndrome. The favorable prognosis in terms of regression of clinical manifestations of the disease reaches 60-80%.

Keywords: Origin of the disease, diagnosis, prevention, prevention, modern diagnostics

Guillain-Barre syndrome (GBS) is an acute inflammatory demyelinating polyradiculoneuropathy of autoimmune etiology characterized by peripheral paralysis and, in most cases, protein-cell shedding in the cerebrospinal fluid.

The first mention of the disease dates back to the 18th century. In 1859, Landry J. described acute ascending paralysis. In 1916, French physicians Guillain G., Barre J., and Strohl A. described the clinical picture of acute peripheral paralysis with protein-cellular dissociation of cerebrospinal fluid in two French soldiers. In 1949, Haymaker WE and Kemohan JW described the clinical picture and histological changes in the peripheral nervous system in 50 patients with GBS [9].

The incidence of GBS is 0.6-2.4 cases per 100 thousand population. GBS occurs with equal frequency in men and women [3,10,23]. According to data provided by Suponeva NA. et al. (2013) in 7 cities and 2 subjects of the Russian Federation the incidence of GBS ranges from 0.34 to 2.5 per 100 thousand population [4]. In Moscow, about 200 people are infected with GBS annually [6].

The development of the disease is preceded by the body's contact with a viral or bacterial infection. According to the literature, symptoms of this disease appear 10-14 days after a viral respiratory infection [10,33]. Infectious agents can include pathogens such as Campylobacter jejuni, Mycoplasma pneumoniae, cytomegalovirus, Epstein-Barr virus, and influenza virus, which have been described in clinical practice as cases of GBS after surgery and vaccination [9,33].

GBS is characterized by an autoimmune lesion of the peripheral nervous system, which often leads to demyelination, followed by secondary axonal destruction of nerve fibers, sometimes myelin and axonal damage can occur simultaneously. GBS is based on autoimmune mechanisms triggered by viral or bacterial infection. On the surface of infectious agents, structures similar to the structure of peripheral nerves (oligosaccharides) are present, resulting in a "molecular mimicry" [5]. As a result of the "molecular mimicry", autoantibodies are produced to antigens of the peripheral nervous system [5].

In the acute phase of the disease, changes in cellular and humoral immunity occur. In the serum of patients, antibodies to peripheral nerve myelin are detected, the activity of T-cells increases, and the number of T-suppressors decreases. As a result of the activation of humoral and cellular immunity, the concentration of IgM, IgA, and circulating immune complexes (CICs) increases. This leads to the accumulation of CECs along the myelin sheath of peripheral nerves [5,9,10].

One of the evidences for the involvement of infectious agents in the inflammation is the detection of high titers of antibodies to gangliosides GM1, GD1a, GD1b and GQ1b in patients with increased antibody titers to the putative pathogen [20,28]. ,34]. In addition, high levels of antibodies to ganglioside GM1 are a risk factor for the development of acute motor axonal neuropathy, a more severe form of GBS, manifested mainly by sensory impairment and poor recovery [33].

The role of "molecular mimicry" in patients with Miller-Fisher syndrome (MFS) is clear. "Molecular mimicry" between infection (Campylobacter jejuni) and peripheral nerve structures plays a key role in axon terminal damage [5,32]. Antibodies to GQ1b and GT1a gangliosides, which target the oculomotor and bulbar nerves, are detected in the blood of patients with MFS [18,25,26,32].

The final step in the pathogenesis of GBS is the entry of T cells and CECs into the endoneurium along with macrophages. This leads to severe tissue damage, which is accompanied by active phagocytosis carried out by cells of the monocyte-macrophage lineage [3,20].

GBS usually begins with muscle weakness and/or sensory disturbances in the lower extremities, which then spread to the upper extremities. According to the literature, in 50% of patients, the disease progresses within 2 weeks from the onset of the first clinical symptoms. By 4 weeks, GBS is detected in 90% of patients. Approximately 80-90% of patients require hospitalization [9,10,16].

Clinical symptoms in the advanced stages of SGB are usually caused by motor, sensory and vegetative disorders; sukhojilnoy hypo- ili areflexii. Dvigatelnye narusheniya (paresis of varying degrees of expression, often vplot do paralichey) nablyudayutsya practically in all patients. V tyajelyx sluchayax u bolshinstva otmechaetsya takje porazhenie myshts tulovishcha, vklyuchaya myshtsy shei, spiny, jivota. Myshechnaya nauskaty v nechnostyakh, kak rule, symetricna i bolshe vyrajena v nagax, odnako vozmojno small preobladanie ee na odnoy strone tela.

Neurological examination allows to reveal motor disorders, which have a symmetrical character. Pri SGB mogut porajatsya cranial nerves (VII, IX X), v rezultate chego narushaetsya glotanie, phonatsiya i voznikayut glazodvigatelnye rasstroystva.

Neobkhodimo pomnit, chto krome neurological manifestations of the disease, and the patient may be driven to the side of other organs and the system. According to Shio A. et al (2003), approximately one third of patients develop respiratory insufficiency, leading to paresis of the diaphragm and respiratory muscles [3,14,15,33]. For patients with SGB, there is a characteristic connection with the side of the cardiovascular system, which manifests itself in the form of arterial hypertension, tachycardia, and bradycardia [15]. V svyazi s narusheniem nervno-musechnoy providomosti u odnoy treti bolnyx nblyudaetsya dysfunction chevevogo zyrya, proyavlyayushchayasya v vide zaderjki mochi. Rasstroystva zhudochno-kishechnogo tract obnarujivayutsya u 15% bolnyx SGB, vklyuchaya takoe groznoe oslojnenie, kak kishechnaya neprokhodimost [9].

V nastoyashchee vremya opisany four basic clinical variants of SGB. Naibolee chastym (classic) variantom yavlyaetsya ostraya vospolitelnaya demyeliniziruyushchaya polyradiculoneuropathy (85-

90%) [14,23]. Full axonal forms of SGB and acute motor axonal neuropathy occur in 10-15% of all SGB cases [27]. Dannye varianty SGB znachitelno chashche vstrechayutsya v stranax Asia i Yuzhnoy Ameriki (30-47%) po sravneniyu so stranami Evropy i Severnoy Ameriki [7]. Miller-Fisher syndrome occurs in 3% of cases and is characterized by ophthalmoplegia, cerebellar ataxia, and paresis [7,21,23].

Obnovlennymi diagnosticheskimi kriteriyami SGB yavlyayutsya sleduyushchie priznaki: progressive motor weakness with vovlecheniem more than one limb, areflexia or expressed hyporeflexia. Dlya podverjdeniya diagnoza SGB ispolzuyut analyz spinomozgovoy jidkosti i elektroneuromiograficheskoe issledovanie. At the analysis of the spinal cord, there are several diagnostic criteria, confirmatory SGB, the relative increase in the concentration of the spinal cord and the firing of cytosis. According to Ropper AH (1992), characteristic changes in spinal fluid are diagnosed more than 90% of patients in the period of acute pain [27,28]. Electroneuromyographic research reveals slowing down of the speed of nerve conduction, late responses of F-waves [9].

Dlya otsenki neurologicheskogo status u bolnyx SGB ispolzuetsya Severomarikensaya skala tyajesti dvigatelnogo defitsita (SASh). SASh allows you to estimate the condition of the patient and his engine capacity from 0 (norm) to 5 stages (need for IVL) (Table 1).

Necessary provision IVL

V nastoyashchee vremya v povsednevnoy praktike primenyaetsya prognostic scale erasmus GBR outcome score (EGBR) and eyo modification mEGBR (table. 2) [1]. The prognostic scale EGBR pozvolyaet v techenie pervyx 1-2 nedel prognozirovat vozmojnoe vosstanovlenie hodby s oporoy k 6-mu mesyatsu zabolevaniya. The prognostic scale EGBR, which was presented by Van Koningsveld R. et.al (2007), shows the age of the patient, diarrhea and the degree of motor impairment. The higher the number of points in the patient's SGB on the EGBR scale, the higher the probability that the patient will not undergo self-care in 6 months (Table 2) [31].

Prognostic Scale Modified Erasmus Guillain-Barré Syndrome Outcome Scores (mEGOS)

The treatment of patients with SGB has two main goals: intensive therapy, aimed at preventing and stopping the clinical manifestations of respiratory insufficiency, preventing thrombosis, adding secondary infectious complications, and suppressing the autoimmune process, which leads to damage to the peripheral nerves of the myelin sheath, before carrying out specific therapy.

In the nastoyashchee vremya k spesificheskoy therapy, including plasmapheresis (PF) and therapy with immunoglobulin class G (IgG), which is carried out in the first week of the disease [7,8,34].

Plasmapheresis (PF) is one of the methods of efferent therapy, in which the volume of removed plasma is mixed with solutions of crystalloids, albumin and donor plasma. PF primenyaetsya u bolnyx s SGB s 1985 goda. Soglasno rekomendatsiyam Amerikanskogo Obshchestva Afereza (2010) PF yavlyaetsya standardnoy protseduroy pri SGB [29]. The scheme of implementation of PF zaklyuchaetsya v udalenii 200-250 ml/kg plasma v techenie 7-14 dney. Zameshchenie udalyaemogo volume plazmy proizvoditsya 5% albuminom. Pokazaniyami dlya provedeniya PF yavlyaetsya narastyushchaya neurological symptoms, trebuyushchaya iskusstvennoy ventilyatsii legkix, nesposobnost proyti bolee 5 m s oporoy ili podderjkoy ili nesposobnost vstat i proyti 5 m self-standing [2,8,17,19,20,30]. In the results of a series of control studies, it was established that the inclusion of PF allows to speed up the healing process and reduce the time required for artificial ventilation [7,14,21,22,25]. As an example, it is possible to present the results obtained by the French United Group, which followed the time of occurrence of patients with SGB and IVL. The results obtained in the data analysis showed that the average time of treatment of patients with SGB and IVL in the group where PF sessions were conducted was 18 days. In the control group, the data showed 31 days [29].

Hughes RA et al. (2003). According to the results obtained in the group of patients, PF vosstanovlenie full muscle strength was observed in 135 of 199 patients. In the control group, there are 112 and 205 patients [15]. According to the data analysis, conducted by the American Neurological Academy in

2003, it was noted that PF allows faster healing of patients with SGB, which has a duration of 4 weeks of pain (level A recommendation) [27].

And the method of efferent therapy - immunosorption (IS) - is a method of efferent therapy in order to achieve a wide spread of PF. IS pozvolyaet svyazyvat i izvlekat trace blood antibodies ili antigeny s moshchyu immunosorbentov. Reaction svyazyvaniya opredelennyx molecule is based on reaction antigen — antibody. Method IS allows to remove circulating plasma Ig without the need to use a buffering solution, such as albumin solution or fresh frozen plasma, which reduces the risk of allergic and infectious complications [8,13]. Immunosorbtsionnye kolonki soderjat gel-sorbent, sostoyashchii trace of tryptophan, covalentnosvyazannogo s polyvinylovym spirtom. In literature predstavleny dannye o primenenii IS u bolnyx s SGB [19]. The results of comparative analysis of the effectiveness of IS and PF do not demonstrate the superiority of the first method over PF [8,19,24].

Nachinaya s 1988 goda, v lechenii dannogo zabolevaniya ispolzuetsya IgG. Intravenous introduction of high-dose IgG was recognized as an effective method of treatment of SGB, capable of reducing prolongation and prolongation of the disease [12]. Kak i v sluchae s PF, the mechanism of therapeutic action of IgG ostaetsya okonchatelno ne izuchennym. It is believed that immunoglobulin removes pathogenic antibodies, blocks Fc-component antibodies and cell-targeting, and also inhibits complement activation, dissolves immune complexes, strengthens the function of lymphocytes, increases production and prevents the growth of functional cytokines [11,28].

Dlya vzroslyx and detey s SGB IgG ispolzuetsya v doze 2g/kg v techenie 2-5 days. The American Neurological Academy (2003) recommended the use of IgG in patients with prolonged illness that does not exceed 2 weeks (uroven A recommendation) [11].

An international randomized controlled trial comparing the effectiveness of PF, IgG and the combination of PF and IgG was conducted in 383 patients with severe GBS. The average time for the development of muscle strength and ability without postoronney assistant in the group where PF was used was 49 days, in the group where IgG was used - 51 days, and in the group with combined use of PF and IgG - 40 days. Dannoe issledovanie prodemonstrirovalo odinakovuyu effektivnost dvukh variantov lecheniya SGB [11,29].

Primenenie corticosteroidov v nastoyashchee vremya schitaetsya neeffektivnym i ne ispolzuetsya v therapyi bolnyx s GB [15]. Obzor six issledovaniy, provedyonnyx and 587 bolnyx, demonstrated the absence of positive influence of corticosteroids and technical pain [17,22].

According to literature data, approximately 20-30% of patients develop respiratory insufficiency, requiring artificial lung ventilation (IVL) [10,27]. Indications for the transfer of a patient to IVL include the growth of respiratory insufficiency: tachypnea, use of auxiliary muscles, tachycardia, blood volume of the lung up to 20 ml/kg [27]. Bolnye, nakhodyashchiesya na IVL podvergayutsya vysokomu risku razvitiya oslojneniy, takikh kak pneumonia, tracheobronkhitis ili sepsis. The average duration of IVL is 2 to 6 weeks [9].

Paresis i paralichi, voznikayushchie u bolnyx SGB, uvelichivayut risk razvitiya tromboembolicheskikh oslojneniy. For the prevention of deep vein thrombosis and pulmonary embolism, low-molecular heparin and prophylactic dosage are prescribed.

Na bol ukazyvayut bolshinstvo bolnyx SGB. After careful research, about 47% of patients perceive it as "scary", "unbearable". V dannom issledovanii 75% bolnym dlya kupirovaniya bolevogo syndrome trebovalos primenenie narcoticheskikh analgekiv [9]. V nastoyashchee vremya preparatami vybora yavlyayutsya gabapentin, carbamazepine, pregabalin.

Approximately 40% of SGB patients need to be rehabilitated. The main group consists of patients who are undergoing IVF for a long time with more severe pain. V kompleks reabilitatsionnyx meropriyatiy khodit massage, lechebnaya fizkultura [9].

Na fone providomoy therapy, vklyuchayushchey PF ili IgG, polnoe vosstanovlenie dvigatelnyx funktsiy nblyudaetsya u bolshinstva bolnyx (60-80%). However, it is often painful, especially in the

axonal form of SGB, which has a good motor function recovery [3,7]. Lethality in SGB averages 5% and can reach 20% in patients who do not have IVF [27,28]. The most common cause of death in patients with GBS may be respiratory failure, aspiration pneumonia, sepsis, thromboembolism of the pulmonary artery. Letalnost znachitelno povyshaetsya s vozrastom: u detey do 15 let ona ne prevyshaet 0.7%. v to vremya kak u lits oldershe 65 let dostigaet 8.6%. Other neblagopriyatnye prognostic factors for full-term recovery include prolonged period IVL (more than 1 month), nalichie somaticheskoy pathology. Persistent residual symptoms are preserved in approximately 7-15% of cases [14,21,23,25]. Predictors of unfavorable functional outcome - age older than 60 years, rapidly progressive course of the disease, low amplitude M-response during stimulation in the distal tochke (underlying traumatic axonal damage). The frequency of recurrence of SGB is 3-5% [3,27].

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