A CLINICAL CASE OF GUILLAIN-BARRÉ SYNDROME INDUCED BY COVID-19

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Background: *The coronavirus infection caused by SARS-Cov-2 is characterized by a damage to many organs and systems of the human body. To date, convincing information has been obtained about the involvement of various parts of the nervous system in the pathological process in patients with COVID-19. Among the most frequently described impairments, there are disorders of smell and taste, common disorders of the central nervous system, characterized by general cerebral symptoms, such as headache, asthenization, psychopathological disorders. One of the rare and severe forms of the peripheral nervous system damage in COVID-19 is Guillain-Barré syndrome (GBS), characterized by acute post-infectious inflammatory polyneuropathy with an autoimmune etiology.* ***Clinical case description.*** *We present a clinical case of GBS associated with COVID-19. The disease debuted as a peripheral tetraparesis with a progredient course of up to 21 days. Systemic administration of immunoglobulin stopped the disease progression. The association of GBS with COVID-19 was clarified a month after the disease onset, when bilateral polysegmental pneumonia was diagnosed, and a high level of IgG to the S-protein of SARS-CoV-2 was found, 3 times higher than the level of IgM, which indicated the duration of the disease was not less than three weeks.* ***Conclusion:*** *The GBS development upon infection with SARS-CoV-2 may precede the lung damage. The debut of GBS during the COVID-19 pandemic requires the exclusion of the SARS-CoV-2 etiological role in each case.*

Keywords: *Guillain–Barré syndrome; clinical features; SARS-CoV-2; novel coronavirus infection; COVID-19; thrombotic manifestations; immunoglobulin.*

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BACKGROUND

A novel coronavirus infection caused by the highly transmissible zoonotic beta-coronavirus SARS-CoV-2, as of June 16, 2021, according to official figures, has affected more than 177 million people worldwide, of which more than 3.8 million have died. Data on excess mortality in different countries of the world for 2020 indicate that the real mortality rates from COVID-19 are much higher [1].

Clinically, COVID-19 is characterized primarily by damage to the respiratory system due to the rapid interaction of the virus with angiotensin-converting enzyme 2 (ACE2) on target cells - the pyramidal epithelium of the respiratory tract and alveolocytes [2]. Massive replication of the virus in target cells leads to their death, the development of a systemic inflammatory response, the production of C-reactive protein (CRP) and other acute phase proteins, activation of alveolar macrophages that produce interleukins, which in total triggers a “cytokine storm” and subtotal lung damage [3], hypercoagulation and multiple organ failure [4].

Numerous studies have shown the neurotropicity and neuroinvasiveness of SARS-CoV-2 [5, 6]. The direct neurotoxic effect of the virus is due to the interaction with ACE2-positive neuroepithelial cells and the retrograde axonal spread of the virus; there are other mechanisms providing the neurotropicity of SARS-CoV-2, associated, in particular, with the expression of neuropilin-1 [7]. The most frequent manifestations of the direct neurotoxic effect of SARS-CoV-2 are mononeuropathies of I, II, V, VII and IX pairs of cranial nerves, manifested by impaired smell (anosmia, hypoosmia, paraosmia) and taste (ageusia, hypogeusia, dysgeusia), less often — optic neuropathy, prosoparesis/prosoplegia, bulbar disorders [5].

In addition to the direct neurotoxic effect of SARS-CoV-2, at least two more pathogenetic mechanisms of damage to the central and peripheral nervous system in COVID-19 are distinguished: the first is associated with the development of ischemia due to hypoxemia and impaired blood supply to the central nervous system (CNS) caused by coagulopathy, endothelial dys-

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function and thrombosis. According to this mechanism, encephalopathy of critical conditions, ischemic and hemorrhagic lesions of the central nervous system develop. The second mechanism includes autoimmune damage to the central nervous system, resulting from antigenic mimicry and inflammatory hyperactivation of the cellular and humoral immunity. One of the variants of autoimmune complications of COVID-19 is Guillain-Barré syndrome (GBS) [5, 8-11].

Analysis of the available data on the relationship between SARS-CoV-2 and the development of GBS made it possible to establish that the prevalence of this disease was significantly higher among patients with COVID-19 than in the general population [8]. As shown by the results of two systematic reviews, clinical manifestations of GBS in patients with COVID-19 appear on the 14th day (11–16th day) [9, 10]. The authors noted that although patients with COVID-19 and GBS more often required treatment in intensive care units, the development of GBS against the background of COVID-19 was not accompanied by a significant increase in mortality [9]. In most patients, GBS occurs as a parainfectious rather than postinfectious complication [11].

Despite the descriptions of clinical cases of GBS in patients with COVID-19 available in the literature, each such case is of scientific and practical interest both in terms of clarifying the mechanisms of nerve tissue damage in coronavirus infection and for developing standards for the treatment of such complications. The clinical case described below is interesting in that the patient's neurological disorders preceded the classic symptoms of COVID-19 with bilateral polysegmental pneumonia and respiratory failure.

CLINICAL CASE

About the patient

The patient, a 72-year-old male, accordion player by profession, was admitted to the neurological department of the Federal Research Center of the Federal Medical and Biological Agency of Russia on February 18, 2021.

Complaints at admission for weakness in the legs, arms; numbness of feet and hands; severe pain in the buttocks, thighs, aggravated by the slightest movement, with an intensity on the visual analogue scale (VAS) up to 9-10 points ("hellish"); retention of urination and stool.

Anamnesis morbi: fell ill on 02/07/2021, when he noted numbness of the fingers, first of the feet, then of the hands. From 12.02.2021, he began to notice weakness in the legs, difficulty walking, a feeling of heaviness in the legs. The patient denies fever, catarrhal, dyspeptic symptoms before illness.

On the morning of 02/13/2021, immediately after sleep, he noted intense pain in the thoracic spine (up to 8 points on the VAS), an increase in weakness in the legs (he could not get out of bed on his own). On 13.02.2021 and 14.02.2021 he called the ambulance teams three times: medications were administered for the purpose of pain relief.

02/15/2021 the patient was admitted to the hospital at the place of residence, where he was until 02/18/2021: Despite vascular, neurometabolic therapy and therapy with non-steroidal anti-inflammatory drugs, there was an increase in muscle weakness in the lower extremities, the appearance of weakness in the upper extremities, increased numbness, pain intensity in limbs with a gradual complete loss of the possibility of self-care.

On February 18, 2021, with the help of relatives, I turned to the CDC FNKTs FMBA for a consultation with a neurologist. From an outpatient appointment, he was sent for emergency hospitalization with a diagnosis of Guillain – Barré syndrome. Due to the high risk of developing acute respiratory failure, the patient was admitted to the intensive care unit.

Anamnesis vitae. Past diseases: permanent form of atrial fibrillation for 2.5 years. In 2019, according to electrocardiography, a diagnosis of postinfarction cardiosclerosis of unknown age was made.

Epidemiological anamnesis: according to the patient, he did not have a coronavirus infection, he did not come into contact with patients. On admission, a polymerase chain reaction (PCR) test for SARS-Cov2 is negative. Denies other infectious diseases.

Physical diagnostics

General condition of moderate severity. The physique is normosthenic, the skin and visible mucous membranes are unremarkable. Body temperature 36.7°C. Respiratory system without features, arterial blood oxygen saturation ($SatO_2$) 99% in atmospheric air, respiratory rate 18/min. Muffled heart sounds, blood pressure (BP) 120/75 mm Hg, pulse 80 beats/min, arrhythmic. The abdomen was normal, peristalsis was heard. Violation of urination by the type of retention.

Neurological status. Consciousness is clear. Oriented in place, time and self. There are no meningeal symptoms. Cranial nerves without signs of focal pathology.

Motor sphere: flaccid tetraparesis; in the upper extremities, the strength is reduced to 2 points, in

the lower extremities — to 1.5 points. Muscle tone is diffusely reduced in the upper and lower extremities. Tendon reflexes from the hands: carporadial - not triggered; reflexes from the biceps muscle are reduced. Knee and Achilles reflexes are not triggered. There are no pathological signs. Superficial sensitivity is preserved, muscular-articular feeling in the distal parts of the feet is sharply reduced. Symptoms of tension are positive on both sides — 25°. Paravertebral points, spinous processes are painless. Movements, turns in bed cause sharp pain in the spine and limbs. He does not perform coordination tests due to paresis.

Autonomic disorders: severe diffuse hyperhidrosis; periodically there was a decrease in blood pressure to 100/60–90/50 mm Hg; tachycardia — pulse 100–110 beats/min.

Laboratory and instrumental diagnostics

Complete blood count, urinalysis, biochemical blood assay, coagulogram are without pathological changes. Antineuronal antibodies (IgG), line blot [separately: amphiphysin (AMRH), CV2.1, PNMA2 (Ma-2/Ta), Ri (ANN A2), Hu (ANN A1)] in blood/cerebrospinal fluid - negative. Antibodies (IgG) to Yo-1 (PCA1) — borderline result (+/-).

Electrocardiography: atrial fibrillation with a ventricular rate of 71–165 beats/min.

The electrical axis of the heart is type SI – SII – SIII. Incomplete right bundle branch block.

Echocardiography: the study was carried out against the background of rhythm disturbances with a ventricular rate of 103–169 per minute. Mitral regurgitation of the 2nd degree. Tricuspid regurgitation of the 1st degree. The global contractility of the myocardium is reduced. Ejection fraction 48%.

Preliminary diagnosis

G61.9 Acute inflammatory motor axonal polyneuropathy (Guillain – Barré syndrome).

Dynamics and outcomes

In the period from 20.02.2021 to 27.02.2021, negative dynamics was noted in the neurological status — the appearance of focal disorders of the cranial nerves in the form of half-ptosis on the left; Difficulty swallowing solid food an increase in weakness in the extremities (in the fingers, strength up to 0.5 points proximally, distally up to 1–1.5 points, in the lower extremities — proximally up to 0.5–1 points, distally up to 1–1.5 points); the appearance of painful paresthesias in the lower and upper extremities; sleep disturbance due to pain in the limbs. In addition, confabulations were observed in the patient: he claimed that he was walking around the ward at night, asking to take off the “chains from his hands.”

On February 20, 2021, electroneuromyography (ENMG) was performed: all the examined nerves showed signs of severe axonal demyelinating lesions.

On February 20, 2021, a lumbar puncture was performed: protein-cell dissociation was revealed: cytosis 1 1/μL, protein 2.7 g/L (N 0.66-0.9).

On February 24, 2021, a course of intravenous drip injection of normal human immunoglobulin Octagam (0.4 g/kg 30 g 600.0 ml) 1 time/day, No. 5 was started.

From February 27, 2021, the patient’s condition stabilized (Fig. 1). In the period from 03.03.2021 to 07.03.2021, positive dynamics was noted in the neurological status in the form of a decrease in pain syndrome (occurs only with movements), a decrease in ptosis on the left, an improvement in swallowing, an increase in strength in the hands up to 2–2.5 points, in the legs up to 1.5 points.

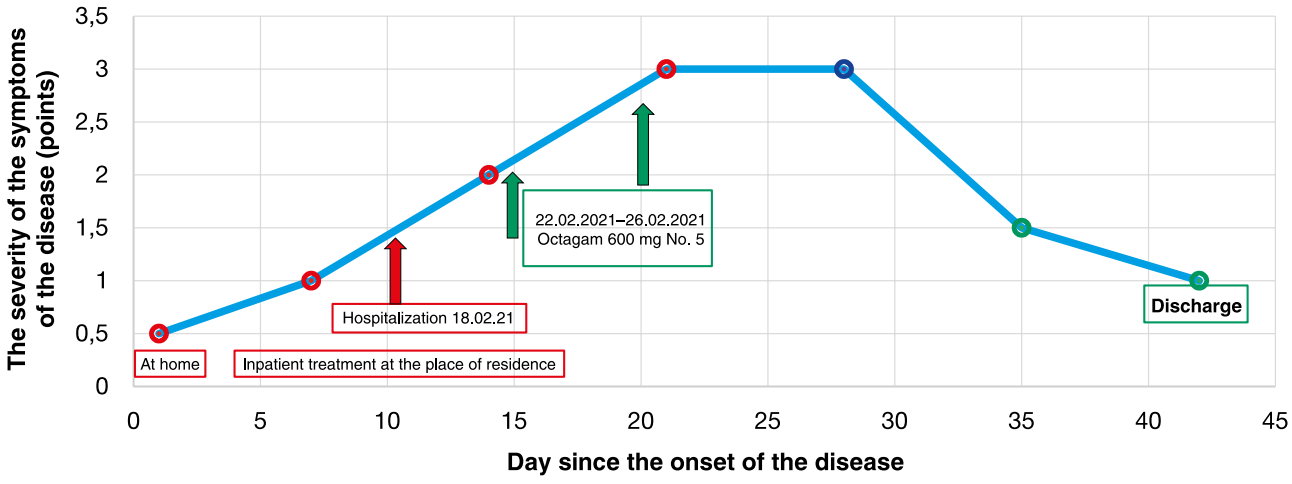

Fig. 1. Patient, male, 72 years old: the time course of Guillain–Barré Syndrome.

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On 03/07/2021, the patient's general condition deteriorated again: general weakness, shortness of breath when speaking, a rare cough, a decrease in SpO_2 saturation to 74% appeared; body temperature remained normal. In laboratory blood counts, there was an increase in leukocytes up to 17.16 × 10^9/l, CRP up to 155.8 mg/l, procalcitoninin up to 4.48 ng/ml, D-dimer up to 1.114 μg/ml, ESR up to 40 mm/h, as well as a decrease in lymphocytes to 0.46 × 10^9/l. Computed tomography (CT) of the chest organs was performed: bilateral polysegmental pneumonia was revealed (Fig. 2, *a-c*).

03/08/2021 repeated PCR test for SARS-CoV-2 RNA was performed — the result was negative; enzyme immunoassay of IgG and IgM to the S-protein SARS-CoV-2 in the blood — the result is positive: the coefficients of positivity were found to be 15.2 and 3.4, respectively.

The next day, CT scan of the chest cavity vessels with contrast enhancement was performed, which revealed thromboembolism of the branches of the pulmonary artery (Fig. 2d).

Duplex scanning of the veins of the lower extremities was performed: deep vein thrombosis of both legs was revealed at the stage of initial (mild) recanalization without signs of flotation.

From 03/10/2021 to 03/20/2021, against the background of therapy, there is a gradual stabilization of the condition, pain in the lower extremities bothers only when activated, swallowing improved, there is an increase in strength in the upper extremities due to the proximal sections. An increase in the range of active movements (can compress and unclench, rotate the hands, bend the legs at the knee joints), confabulations disappeared. According to laboratory parameters, a decrease in leukocytosis to 6.9 × 10^9/l, CRP to 7.2 mg/l, an increase in the number of lymphocytes to 1.49 × 10^9/l, a decrease in ESR to 20 mm/h, positive dynamics on CT of the chest organs were noted.

In the period from 03/20/2021 to 03/26/2021, a significant improvement in the patient's condition is noted: shortness of breath disappeared; general weakness has significantly decreased; pains, paresthesias in the limbs, the spine do not bother; sleep has returned to normal. In the neurological status: the ptosis on the left significantly decreased, swallowing was restored, the range of motion increased when shrugging the shoulders, as well as in the upper and lower extremities (he can raise his arms to the level of the face, the strength in the hands increased to 3.5 points, in the legs up to 3 points. The patient sits on his own, works out in the gym on simulators.

The prognosis of the disease is favorable.

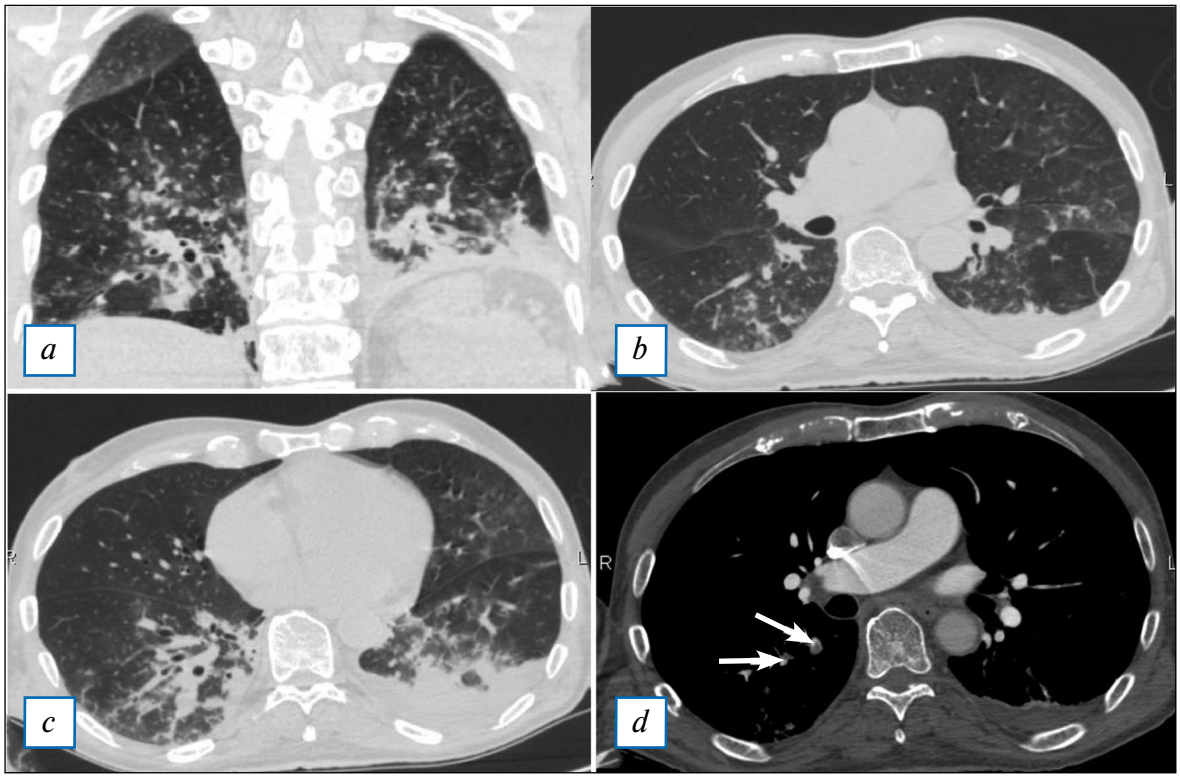

Fig. 2. The same patient. CT-scan of the chest: (*a–c*) — In both lungs (in the upper lobe of the left lung, in S2, S4, segments of the lower lobe of the right lung), areas of «ground glass» and consolidation are determined; (*d*) — Pulmonary embolism of segmental branches on the right (white arrows).

Clinical diagnosis

Acute inflammatory motor axonal polyneuropathy (Guillain-Barré syndrome) caused by SARS-CoV-2 coronavirus infection. Flaccid tetraparesis with predominance in the legs, severe pain syndrome and dysfunction of the pelvic organs, severe course. Bilateral polysegmental pneumonia in the stage of resolution. Complication: thromboembolism of small branches of the pulmonary artery. Deep vein thrombosis of the lower extremities at the stage of initial recanalization.

A characteristic clinical and radiological picture (interstitial lung lesion with frosted glass and consolidation phenomena, lymphopenia and increased CRP), as well as the presence of specific IgG and IgM to the SARS-CoV-2 S-protein made it possible to diagnose COVID-19, despite a negative PCR result on SARS-CoV2.

DISCUSSION

The appearance of neurological symptoms in our patient preceded the development of bilateral polysegmental pneumonia. At the same time, the detection of a high level of IgG to the S-protein SARS-CoV-2 as well as the fact that the level of IgG was almost 3 times higher than the level of IgM (positivity coefficients of 15.2 and 3.4, respectively), unambiguously indicated that the moment of the examination, at least 3 weeks have passed from the onset of the disease, i.e. the onset of neurological symptoms almost coincides with infection with SARS-CoV-2. Thus, in this clinical case, we are dealing with GBS associated with COVID-19.

Guillain-Barré Syndrome (GBS) is the most common cause of peripheral paralysis. The incidence averages 1.7 per 100,000 population per year, is approximately equal in men and women, has no seasonal fluctuations, and is more common in old age [12]. In about 1/3 of cases, patients with GBS require resuscitation benefits up to mechanical ventilation [13]. The mortality rate ranges from 2 to 12%.

In the 17th century, the first mentions of peripheral neuropathies appeared. In 1859, the French neurologist J.B. Landry observed in 10 patients ascending paralysis, including the muscles of the face and tongue, with minor sensory disorders. Severe symptoms grew rapidly, two patients died. Later, the disease was defined as “Landry’s ascending paralysis”. In 1916 G. Guillain, J.A. Barre and A. Strol described a special form of primary polyradiculoneuritis in two soldiers of the French army. The disease had a characteristic clinical picture, and in less than 2 months they recovered. Later it was concluded that the diseases under consideration are variants of the same pathological process, despite the different severity of the course.

The demyelinating nature of the disease was proven in the 1950s, but in some cases axonal damage took place, which made it possible to distinguish two main forms of the disease — demyelinating (the most common) and axonal (a rarer variant) [14]. With the demyelinating variant of the disease, the myelin sheaths of the axons suffer, demyelination is observed without the involvement of the axonal cylinders of the axons, and therefore the speed of conduction along the nerve fiber decreases with the development of paresis. The demyelinating variant is characteristic of the classic Guillain – Barré syndrome.

The main clinical manifestation of the disease is a relatively symmetrical flaccid tetraparesis, growing over several days or weeks (on average 7–15 days): weakness in the arms and legs with low muscle tone and decreased/loss of tendon reflexes, impaired deep sensitivity. In the beginning, the proximal parts of the legs are more often involved, which is manifested by difficulty in climbing stairs or getting up from a chair, only after a few hours or days the hands are involved — “ascending paralysis”. Muscle hypotension and hypotrophy develop (in the late period).

The disease can quickly (within a few hours) lead to paralysis of the respiratory muscles. Vegetative disorders in the acute period occur in more than half of cases of the disease and are often the cause of death; observed violation of sweating, intestinal paresis, increase or decrease in blood pressure, orthostatic hypotension, tachycardia or bradycardia, supraventricular, ventricular arrhythmias, cardiac arrest [14]. Pain in the limbs and spine often occurs. Pain can be both the first symptom and be observed in the late period of the disease.

Much less often, an axonal variant of the lesion is observed, which is more severe, in which degeneration of axial cylinders of Wallerian type axons develops, as a rule, with the development of gross paresis or paralysis. In the axonal variant, the antigens of the axons of the peripheral nerves are primarily subjected to autoimmune attack, and a high titer of GM1 antibodies is often found in the blood [13].

In addition to the typical forms of GBS, there are atypical forms of the disease. One of them is Miller-Fisher syndrome, which is manifested by ophthalmoplegia with the involvement of the external, less often internal muscles of the eye, motor ataxia — a violation of gait and ataxia of the trunk muscles. It occurs in 5% of cases of Guillain – Barré syndrome.

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The sensory form of GBS is manifested only by sensory disorders and loss of tendon reflexes, often with pain. Acute motor-sensory axonal polyneuropathy is characterized by predominantly motor and minimal sensory disturbances. Acute pandizautonomy is an isolated autonomic dysfunction with no other GBS symptoms. The pharyngo-cervico-brachial variant of GBS is manifested by bulbar syndrome, weakness of the neck muscles; decreased tendon reflexes and impaired sensitivity are usually detected only on the upper extremities [14]. GBS with lesions of the cranial nerves is more often manifested by paresis of the muscles of the eyeball or facial nerve.

The main diagnostic method that confirms the diagnosis of GBS is ENMG, which allows to identify the peripheral nature of the lesion, as well as to differentiate the demyelinating and axonal variants of the disease. In the demyelinating variant, the disease is characterized by a decrease in the amplitude of the M-response against the background of signs of demyelination of nerve fibers — a decrease in the conduction speed along the motor fibers by more than 10% of the normal value, lengthening of the distal latency, and partial conduction blocks. In the axonal variant, a decrease in the amplitude of the M-response is detected against the background of a normal conduction velocity along motor fibers (or a decrease in velocity, but not more than 10%), a normal value of the distal latency and F-response [15]. In addition to ENMG, the study of cerebrospinal fluid is of diagnostic value: starting from the 2nd week. protein-cell dissociation is detected with normal or slightly increased cytosis (no more than 50 cells/μl) [13, 16]. Understanding of the autoimmune nature gave impetus to the use of specific therapy for GBS — plasmapheresis and immunotherapy. The results of multicenter studies have shown that immunotherapy with class G immunoglobulins is effective for the treatment of GBS [9].

Data on the features of the course of COVID-19 show a wide prevalence of neurological symptoms: more than 30% of patients have dizziness, headaches, impaired sensitivity to odors and myalgia [8]. During the COVID-19 pandemic, there have been reports of patients with GBS in the literature. In April 2020, the Lancet described the first case of Guillain-Barré syndrome associated with COVID-19 in a 61-year-old woman who consulted a doctor complaining of increasing weakness in her legs and arms. After 8 days, her temperature increased, CT of the lungs revealed pneumonia, and PCR diagnostics revealed SARS-CoV-2 RNA [17]. There are also descriptions of GBS variants with lesions of the oculomotor innervation — Miller-Fisher syndrome associated with COVID-19 [5, 18].

GBS associated with COVID-19 does not have any characteristic clinical features compared to GBS of other etiology [19]. According to modern concepts, the pathogenesis of GBS is due to the cross-reaction of immune cells activated by inflammation with components of peripheral nerves by the mechanism of molecular mimicry [20]. The immune response can target myelin antigens or other axonal antigens of peripheral nerves, which leads to the development of demyelinating or axonal GBS [21]. Probably, with SARS-CoV-2-induced GBS, the same pathogenesis takes place, given the timing of the development of this condition (usually after the 10th day from the onset of COVID-19) and the absence of the pathogen RNA in the CSF of patients [22]. The rapid development of the disease in our patient's case, coinciding with the onset of viral replication, probably indicates its premorbid features and the presence of latent autosensitization, in which infection with SARS-CoV-2 was a trigger that triggered the symptomatic phase of the disease.

Limitations

The limitation of this clinical case is the lack of PCR confirmation of infection with SARS-CoV-2. The lack of examination of our patient can also be attributed to the lack of tests for antibodies to gangliosides and the study of cerebrospinal fluid for SARS-CoV-2 RNA.

CONCLUSION

In the above clinical observation, Guillain-Barré syndrome occurred, which developed as a result of infection with the COVID-19 virus. This is confirmed by both clinical data characteristic of GBS and laboratory instrumental (protein-cell dissociation in the cerebrospinal fluid, ENMG signs of axonal demyelinating lesion) studies. COVID-19 was confirmed radiographically (polysegmental pneumonia) and clinical and laboratory (positive IgG and IgM to the S-protein SARS-CoV-2, lymphopenia, increased C-reactive protein, coagulopathy, the development of thromboembolic complications). The IgG / IgM ratio allowed us to conclude that the development of neurological complications practically coincided with infection and the beginning of viral replication, which may indicate the presence of premorbid features in the patient, which caused the rapid development of inflammatory demyelination.

ADDITIONAL INFORMATION

Author contribution. Shirshova E.V., Knaub V.V. — patient management, manuscript writing; Baklaushev V.P. — general concept, manuscript editing. The authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work.

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Competing interests. The authors declare that they have no competing interests.

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