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СИНДРОМ ГИЙЕНА-БАРРЕ У ПАЦИЕНТА С ИНФЕКЦИОННЫМ ЭНДОКАРДИТОМ: КЛИНИЧЕСКОЕ НАБЛЮДЕНИЕ И ОБЗОР ЛИТЕРАТУРЫ

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Guillain-Barre Syndrome in A Patient with Infectious Endocarditis: Clinical Observation and Literature Review

Резюме

Синдром Гийена-Барре (СГБ) — это тяжелое аутоиммунное заболевание периферической нервной системы, представляющее собой наиболее частую причину развития острого вялого тетрапареза, которое может приводить при отсутствии адекватной терапии к жизнеугрожающей дыхательной недостаточности. Взаимосвязь СГБ и инфекционного эндокардита изучена недостаточно.

У пациента 54 лет через 2 недели после перенесенной острой респираторной вирусной инфекции в течение 2 дней наросли онемение и слабость в нижних конечностях, госпитализирован с подозрением на ишемический инсульт. При компьютерной томографии (КТ) головного мозга — данных за острую очаговую патологию не получено, выявлены левосторонний гайморит и этмоидит. В анализе ликвора — белок — 0,8 г/л (норма — до 0,2 г/л), цитоз в пределах нормы. Прозериновая проба отрицательная. Клинико-инструментальная картина расценена как СГБ. При эхокардиографическом исследовании выявлены вегетации на створках митрального клапана (МК), отрыв хорд его передней створки — «молотящая створка», митральная регургитация 3 степени. Несмотря на проводимую антибактериальную, иммуномодулирующую и др. терапию, отмечалось сохранение неврологической симптоматики, развитие тромбоэмболии легочной артерии, внутрибольничной двусторонней полисегментарной пневмонии, сепсиса с летальным исходом. При аутопсии подтвержден инфекционный эндокардит с отрывом хорд МК, обильным ростом Pseudomonas aeruginosa, скудным ростом Klebsiella pneumoniae и Acinetobacter baumannii, тромбоз правого предсердия. Патологических изменений в ткани головного мозга не обнаружено.

Таким образом, синдром Гийена-Барре в редких случаях может сочетаться с инфекционным эндокардитом и оказывать негативное влияние на течение и прогноз заболевания.

Ключевые слова: *синдром Гийена-Барре, инфекционный эндокардит, демиелинизация, нейропатия*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

Источники финансирования

Авторы заявляют об отсутствии финансирования при проведении исследования

Соответствие принципам этики
Информированное согласие пациента не требуется в силу невозможности его получения в виду смерти пациента.

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Abstract
Guillain-Barré Syndrome is a severe autoimmune disease of the peripheral nervous system, representing the most common cause of acute flaccid tetraparesis, which can lead to life-threatening respiratory failure in the absence of adequate therapy. The relationship between GBS and infective endocarditis is not well studied.

A 54-year-old patient experienced increasing numbness and weakness in the lower extremities two weeks after an acute respiratory viral infection, and was hospitalized with suspected ischemic stroke. Brain computed tomography revealed no data for acute focal pathology but showed left-sided sinusitis and ethmoiditis. Cerebrospinal fluid analysis showed protein at 0.8 g/L (normal up to 0.2 g/L), with cytosis within normal limits. The neostigmine test was negative. The clinical and instrumental picture was assessed as Guillain-Barré Syndrome. Echocardiography revealed vegetations on the mitral valve (MV) leaflets, rupture of the anterior leaflet chordae ("flail leaflet" of the MV), and grade 3 mitral regurgitation. Despite ongoing therapy, including antibacterial and immunomodulatory treatment, the patient continued to exhibit neurological symptoms, developed pulmonary artery thromboembolism, nosocomial bilateral polysegmental pneumonia, and sepsis, leading to a fatal outcome. Autopsy confirmed infective endocarditis with rupture of the MV chordae, abundant growth of Pseudomonas aeruginosa, scant growth of Klebsiella pneumoniae and Acinetobacter baumannii, and right atrial thrombosis. No pathological changes were found in the brain substance.

Thus, Guillain-Barré Syndrome in rare cases can be associated with infective endocarditis and negatively impact the course and prognosis of the disease.

Key words: *Guillain-Barré syndrome, infective endocarditis, demyelination, neuropathy*

Conflict of interests
The authors declare no conflict of interests

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Conformity with the principles of ethics
Informed consent of the patient is not required due to the impossibility of obtaining it due to the death of the patient

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BP — blood pressure, ALT — alanine aminotransferase, AST — aspartate aminotransferase, IV immunoglobulin — intravenous immunoglobulin, ALV — artificial lung ventilation, CT — computer tomography, MV — mitral valve, GBS — Guillain — Barre syndrome, US — ultrasound examination

Introduction

Guillain-Barre syndrome (GBS) is a severe autoimmune disease of the peripheral neural system, the most common cause of acute flaccid quadriparesis, which, if left untreated, can result in life-threatening respiratory distress [1].

GBS is the most common form of acute polyneuropathy. Annually, the global incidence of this disease varies from 0.6 to 2.4 per 100,000 people. GBS is diagnosed more often in men than in women; the incidence grows with the age [2].

There are the following clinical forms of GBS (Figure 1) [2]:

1) Classical sensorimotor (30–85%);
2) Isolated motor (10–70%);
3) Paraparetic (5–10%);
4) Oesophageal-cervicobrachial (< 5%);
5) Facial diplegia with paresthesia (< 5%);
6) Isolated sensor (< 1%);
7) Miller Fisher syndrome (5–25%);
8) Bickerstaff's brainstem encephalitis (< 5%).

Aetiology and Pathogenesis

The aetiology of GBS is still unclear; however, its development is assumed to be related to a past infection, primarily to Campylobacter jejuni, zika virus, flu, and SARS-CoV-2 virus (after 2020) [3, 4].

Patients with GBS caused by an infectious disease produce anti-gangliosides antibodies by molecular mimicry. Bacterial cross-reactive antigen identified by macrophages and T cells induces B cells for the development of anti-gangliosides response. These antibodies bind both to nerve fibre gangliosides and microbial antigens. Activated endoneural macrophages release cytokines and free radicals, penetrate compact myelin, periaxonal

space and sometimes block nervous conductivity or cause axonal degeneration. Activated T cells release pro-inflammatory cytokines, fix complement, damage Schwann cells, and cause myelin dissolution.

In acute inflammatory demyelinating polyradiculoneuropathy, immune damage occurs mostly in the myelin sheath and associated components of Schwann cells, whereas in acute motor axonal neuropathy, the key target is neural axon membranes (axilemma).

The clinical signs of GBS are caused by interruption of ascending and descending neuroanatomic pathways in the transverse plane of the spinal cord. Despite the proven relationship between GBS and infectious agents, in a number of cases the disease is identified as idiopathic [5].

Clinical presentation of Guillain-Barre syndrome

Patients with the classical sensomotor form of GBS have distal paresthesia or loss of sensation with weakness, which starts from lower limbs and progresses to the upper limbs and cranial nerves. A typical sign is weakening or diminishment of tendon reflexes in the majority of patients during the first days of the disease. Vegetative nervous system impairments are a common sign and can include unstable blood pressure, arrhythmias, pupil dysfunction, and gut or urinary bladder dysfunction. Often patients report pain syndrome, which can be muscular, radicular or neuropathic. The disease can have acute or subacute onset; patients can become disabled within two weeks. However, if the peak clinical signs appear within the first 24 hours or four weeks after disease onset, GBS is unlikely. GBS is characterised by monophasic clinical progression, however, some patients can have recurrences [6].

Atypical clinical presentation

GBS can have atypical clinical presentation. In the majority of cases, weakness and sensor signs are bilateral; however, they can be asymmetrical, mostly proximal or distal, they can start from lower, upper limbs or simultaneously in all limbs. Weakness can be preceded by severe diffuse pain or isolated dysfunction of cranial nerves. Children under six years of age often have non-specific or atypical clinical manifestations, such as pain without clean localisation, irritability, meningism or unsteady gait. Inability to identify these signs as early onset of GBS can result in delayed diagnosis. In some patients with atypical GBS, especially with isolated motor form, electromyographic examination can show normal or even enhanced reflexes over the entire period of disease duration [7].

Specific Forms of Guillain-Barre Syndrome Depending on the Symptoms

Some patients demonstrate a clear and persistent clinical variant of GBS, which does not progress to the classical pattern of loss of sensation and weakness. These variants include: weakness without sensor signs (isolated motor variant); weakness restricted to the cerebral nerves (bilateral facial nerve palsy with paresthesia), upper limbs (oesophageal-cervicobrachial form) or

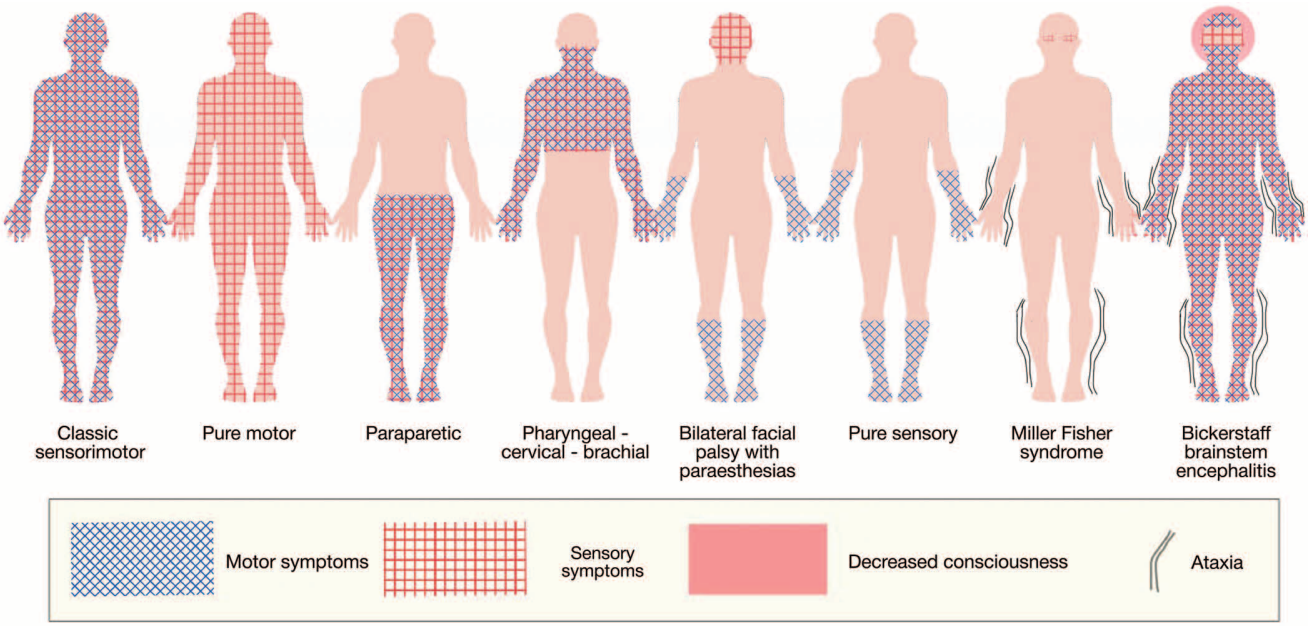

Fig.1. *Clinical forms of Guillain-Barré syndrome [2]*

lower limbs (paraparetic variant); and Miller Fisher syndrome, which in its full manifestation includes ophthalmoplegia, areflexia and ataxia [8]. GBS variants are rarely straightforward, they partially coincide with the classical syndrome or demonstrate characteristics typical for other forms. In addition to the mentioned variants, GBS often includes Bickerstaff's brainstem encephalitis and isolated sensor variant, since they have common clinical or pathophysiological signs. However, inclusion of these clinical variants is a subject of discussions, because they do not meet the diagnostic criteria for GBS [9].

Diagnostics of Guillain-Barre Syndrome

In the absence of adequately sensitive and specific biomarkers of the disease, the diagnosis of GBS is based on the clinical manifestation and test results and is confirmed with additional data, such as cerebrospinal fluid examination and electromyographic study. The two most commonly used sets of diagnostic criteria of GBS developed by the National Institute of Neurological Disorders and Stroke in 1978 (revised in 1990) andBrighton Collaboration in 2011 [10] are still relevant today.

Cerebrospinal fluid examinations are used mostly to rule out other causes of weakness, other than GBS, and should be performed during initial patient examination. A classical sign of GBS is a combination of higher protein levels and normal cell count in cerebrospinal fluid (known as albuminocytologic dissociation). However, protein levels can remain within the normal range in 30% to 50% of patients during the first week after disease onset and in 10% to 30% of patient during the second week. Marked pleocytosis (> 50 cells/μL) is not typical of GBS and is possible, e.g., in leptomeningeal malignancies, infectious or inflammatory conditions of the spinal cord or nerve roots. Moderate pleocytosis (10–50 cells/μL) is likely to be observed in GBS patients, but clinicians should consider alternative diagnoses, e.g., infectious causes of polyradiculitis [11].

Diagnostic Criteria of Guillain-Barre Syndrome [2, 12]

Criteria essential for the diagnosis:

1. Progressive bilateral weakness in the upper and lower limbs (initially only lower limbs can be affected).
2. Reduced or absent tendon reflexes in affected limbs (at a certain time point during disease progression).

Symptoms confirming the diagnosis:

- Progressive phase lasts for a period of up to four weeks (usually less than two weeks).
- Relatively symmetrical symptoms and signs.
- Relatively mild sensor symptoms and signs (absent in isolated motor variant).
- Cranial nerve involvement, especially facial nerve palsy.
- Vegetative dysfunction.
- Muscle or root pain in the back or limbs.
- Increased protein levels in cerebrospinal fluid. Normal protein levels do not rule out the diagnosis.
- Electromyographic signs of motor or sensorimotor neuropathy. Normal electrophysiological characteristics at early stages of the disease do not rule out the diagnosis.

Symptoms placing the diagnosis in question:

- Increased levels of mononuclear cells or polymorphonuclear cells in spinal fluid (> 50×10^6/L).
- Marked, persistent asymmetric weakness.
- Gut or urinary bladder dysfunction at early stages of the disease, or persistent dysfunction over the disease duration.
- Severe respiratory dysfunction with limited limb weakness at the onset of the disease.
- Sensor symptoms with limited weakness during the course of the disease.
- Fever at the onset of the disease.
- Significantly increased sensitivity, pointing to a spinal cord trauma.
- Hyperreflexia or clonic seizures.
- Extension plantar reflexes.
- Abdominal pain.
- Slow progression with limited weakness, no damage to breathing muscles.
- Continuous progression for over four weeks after symptom manifestation.
- Alteration of consciousness (except for Bickerstaff's brainstem encephalitis).

Therapy of Guillain-Barre Syndrome

The therapy should be initiated if a patient is unable to walk ten metres. Evidence of therapy efficacy in ambulatory patients is limited; however, treatment should be considered, especially if patients experience rapidly progressive weakness or other serious symptoms, such as vegetative dysfunction, bulbar or respiratory distress. Clinical trials demonstrated efficacy of intravenous (IV) immunoglobulin, if started within two weeks after the onset of weakness, and of plasma separation, if started within four weeks. No evidence of the efficacy outside these windows has been reported [13].

IV immunoglobulin (0.4 g/kg of body weight daily for five days) and plasma separation (200–250 mL of plasma/kg of body weight, five sessions) are equally efficient

in the management of GBS. IV immunoglobulin and plasma separation have similar risks of side effects. Since IV immunoglobulin is more accessible and easy to use than plasma separation, it is used more often. It is worth noting that plasma separation followed by IV immunoglobulin did not demonstrate superior efficacy vs. both methods used separately. In clinical settings, where resources are limited, small-scale plasma separation can be a cost-effective and relatively safe alternative to traditional plasma separation; however, this approach cannot be recommended for wide use until its efficacy is proven in future studies [14].

Besides IV immunoglobulin and plasma separation, other therapies did not demonstrate any efficacy in the management of GBS. Eight randomised, controlled clinical trials did not identify any significant benefits of using corticosteroids in GBS patients, and oral corticosteroids even had unfavourable impact on the prognosis [14].

In the presence of clinical signs of an infection, antibacterial or antiviral therapy can be initiated in patients with GBS [15].

The relationship between GBS and infective endocarditis has not been sufficiently studied.

Case Study

A 54-year-old patient suddenly developed weakness and numbness in his lower limbs, more marked on the left side. Two days later, he fell on the stairs because of increasing weakness in his lower limbs. The patient was hospitalised with suspected ischaemic stroke in the bed of the right medial cerebral artery. His past medical history shows that he did not control his BP; two weeks before the incident, he had acute respiratory virus infection, his body temperature rose to 37.3 °C, but he did not call for medical assistance. The patient denies chronic conditions, nicotine and drug addiction.

Upon admission, he was in serious condition. The skin was unremarkable, with bruises all over his lower and upper limbs, sustained at various times. Auscultation findings: harsh breathing, large bubbling rales over the whole surface of the lungs. Respiratory rate: 25 respirations per minute. BP: 170/105 mm Hg. Auscultation findings: muffled heart tones, systolic murmurs at the apex, irradiating to the left axillary region. Heart rate: 78 beats per minute. No signs of congestion in the central circulatory system. Neurological status upon admission: lucid, Glasgow coma scale: 15 points. Meningeal signs are not observed. Fields of vision are normal; no gaze palsy; pupils are round; OD=OS; photoreactions are preserved. Horizontal end-position nystagmus when looking to the left; asymmetric nasolabial folds because of the left part of the face (preclinical sings); the tongue is on the midline; swallowing, voice set and articulation are preserved. Motor functions: quadriparesis up to four points; Barre test on the upper and lower limbs show more quick lowering movement of the left limbs. Weaker tendon reflexes, D=S. No pathological foot reactions. Sensitivity is normal. Coordination tests: the patient misses targets because of weakness in his limbs.

Brain CT: no signs of acute cerebrovascular accident; left-sided acute maxillary sinusitis and ethmoiditis were diagnosed.

During the 12 hours spent in the inpatient clinic, the patient's neurological status changed negatively: quadriparesis reached three points in the upper limbs and two points in the lower limbs. Mild dysphagia and dysarthria appeared. The patient was consulted by a neurologist once again. To rule out ischaemic stroke in the vertebrobasilar system, *brain CT* was performed; no focal pathology was observed. *CT angiography* of brachiocephalic arteries, intracranial section of brachiocephalic arteries, and the circle of Willis did not show any hemodynamically significant stenosis and acute pathologies.

A *clinical analysis of spinal fluid* showed an increase in protein concentrations to 0.8 g/L (normal range: 0.22–0.33 g/L). Electroneuromyography was not performed due to technical difficulties. Taking into account past medical history, clinical presentation, no CT signs of ischaemia, no CT angiography signs of occlusion, GBS or generalised myasthenia were suspected. To rule out myasthenia, a proserine test was performed, which came back negative.

Echocardiography showed signs of mucoid degeneration and detached anterior mitral leaflet chords — flail leaflet. Hypoechogenic and isoechoic overlaps along the leaflet edge, most likely vegetations, were observed. Stage 3 mitral insufficiency. *Transesophageal echocardiography* confirmed infectious endocarditis, once vegetation on the anterior leaflet of MV were found (Figure 2). Also, a blood clot was found in the right atrial cavity, which was attached to the wall (Figure 3).

A comprehensive examination was undertaken: *ultrasound examination (US) of brachiocephalic arteries and radial arteries* with rotating tests*; ultrasound examination of lower limb veins; ultrasound examination of kidneys, adrenals, retroperitoneal space; no pathologies were identified. Abdominal ultrasound:* signs of chronic calculous cholecystitis, moderate diffuse changes in the liver.

One major and one minor Modified Duke Criteria (2015) were used to make a diagnosis for the patient:

Primary disease: Subacute primary infectious endocarditis of the anterior leaflet of the intact mitral valve.

Concurrent disease: Guillain-Barre syndrome.

Background disease: Mucoid degeneration of mitral leaflets.

Primary disease complications: Detached anterior mitral leaflet chords. Stage 3 mitral insufficiency. Chronic cardiac insufficiency with preserved ejection fraction, stage IIA, NYHA functional class II.

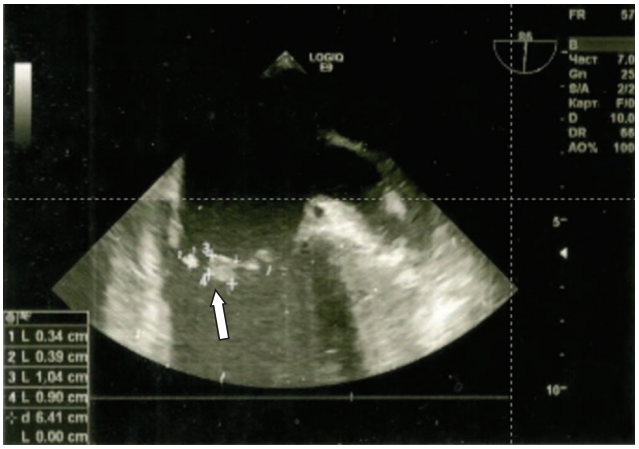

***Fig.** 2. Transesophageal echocardiography. Two vegetations on the anterior leaflet of the mitral valve are 0.34*0.39 cm and 1.04*0.9 cm in diameter (indicated by an arrow)*

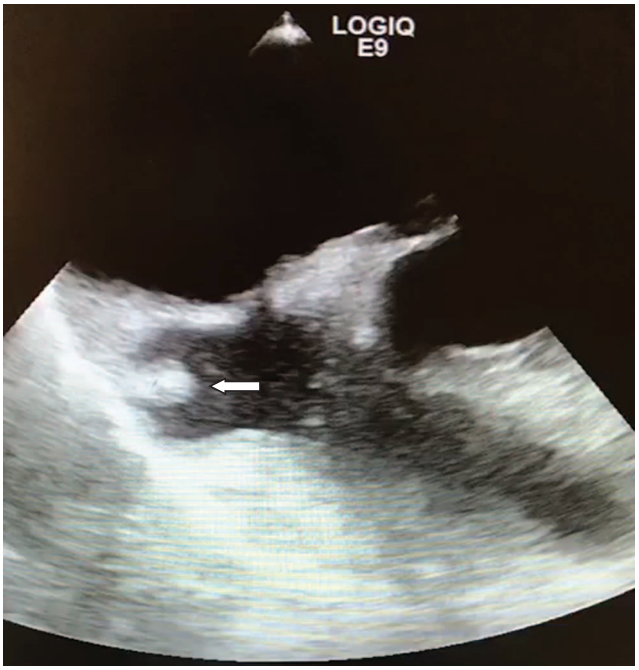

***Fig.** 3. Transesophageal echocardiography. Thrombus in the cavity of the right atrium (indicated by an arrow), fixed to the wall*

Comorbidities: Stage II hypertensive disease, uncontrolled. Target BP values: 120–130/70–79 mm Hg. A very high risk of cardiovascular complications. Right atrium thrombosis. Gallstone disease: chronic calculous cholecystitis, remission. Acute maxillary sinusitis (left side). Acute ethmoiditis.

Multiple *blood culture for sterility:* no flora growth was observed. Laboratory test results are presented in Table 1.

Antibacterial therapy was initiated (meropenem trihydrate 1 g IV three times daily; linezolid 600 mg IV two times daily). Several thromboconcentrate transfusions were performed because of persistent low platelet count. Once low platelet count was corrected, anticoagulant therapy was initiated. Because of aggravated respiratory insufficiency, the patient was connected to ventilatory support. On day 3, tracheostomy was performed.

On day 3 after hospital admission, quadriparesis was one point; no tendon reflexes were observed. Immunomodulating therapy was initiated (human plasma proteins with immunoglobulin G content of no less than 98%, 400 mg/kg/day), and the neurological status improved: muscle strength in the upper and lower limbs rose from one to three points in proximal sections and from one to two points in distal sections. The patient was transferred to unassisted breathing with oxygen insufflation of 5 L/min.

***Table 1.** Results of laboratory research methods.*

Indicators \ Day of hospitalization	Reference values	1	3	7	10	12	14
Hemoglobin, g/l	130-160	134	119	112	87	91	90
Тромбоциты, x10⁹/l	150-400	6	82	226	275	438	454
Platelets, x10⁹/l	4-9	9,2	10,1	17,6	11,67	21,79	25,7
Band neutrophils, %	1-6	-	-	7	-	4	8
Segmented neutrophils, %	47-72	80,9	81,2	78	-	91	85
Creatinine, mmol/l	70-120	75,5	73	90,1	83,6	109,3	284,8
Urea, mmol/l	2,5-8,3	6,2	5,55	13,4	16,69	20,54	49,93
Total protein, g/l	65-85	61,1	68	72,6	63,5	62,7	60,4
Albumin, g/l	35-52	39,7	33,7	31,1	27,5	27,6	25,7
Alanine aminotransferase (ALT), U/l	10-41	19,7	25,4	38,5	362	341	1060,8
Aspartate aminotransferase (AST), U/l	11-41	37,9	27,9	27,2	292	178	1699,6
Alkaline phosphatase, U/l	30-120	82,8	86,7	72,4	88,1	108,8	124,8
Potassium, mmol/l	3,5-5,1	3,89	4,6	3,8	3,9	4,2	7,7
Procalcitonin, ng/ml	< 0,05	-	0,1	0,47	-	6,3	30,15

Despite the therapy, on day 12 after hospital admission, the patient's condition deteriorated: body temperature rose to 38.0 °C, respiratory distress worsened, the patient was again connected to a lung ventilator.

Inoculation of intratracheal tube discharge showed the presence of Pseudomonas aeruginosa (10^5 CFU/mL), Acinetobacter baumannii (10^7 CFU/mL).

Chest CT revealed signs of bilateral multisegmental pneumonia; *when IV bolus contrast medium was used* — signs of thromboembolism of the left lower lobe artery.

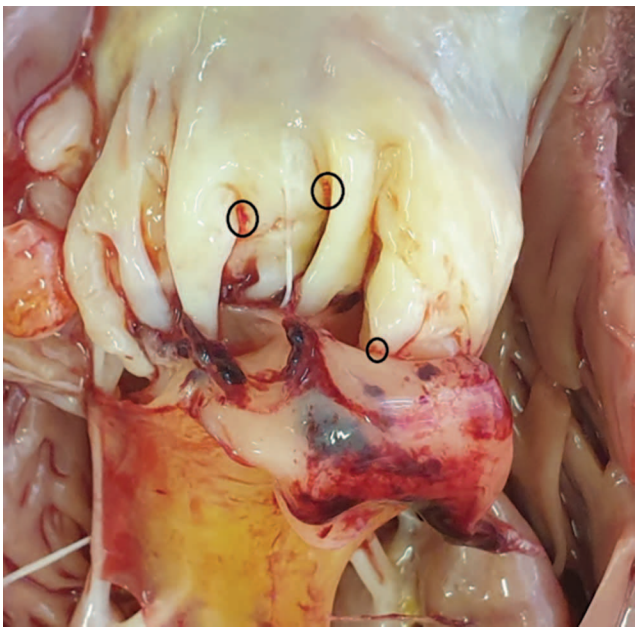

***Fig.** 4. Vegetations (marked with a circle) on the endocardium of the mitral valve*

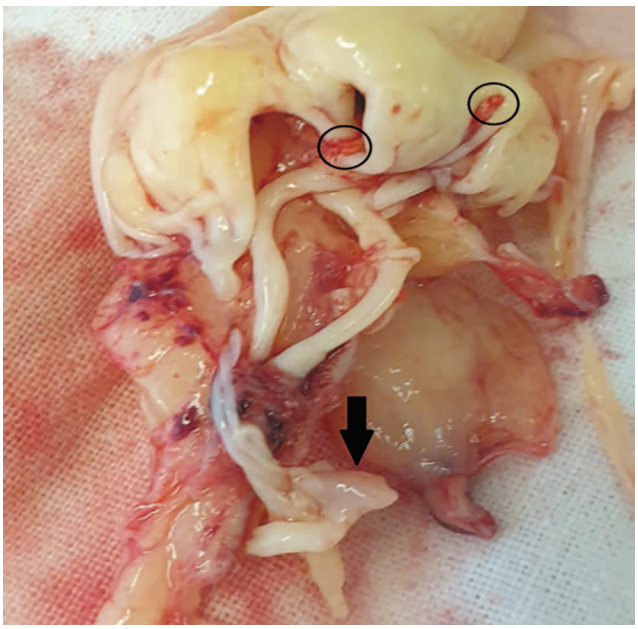

***Fig.** 5. Vegetations (marked with a circle) on the endocardium of the mitral valve. Mitral valve leaflet rupture (arrow)*

Antibacterial therapy was adjusted: meropenem trihydrate was cancelled; polymyxin B 100 mg IV two times daily was initiated; linezolid 600 mg IV two times daily.

Despite the therapy, the patient's condition kept deteriorating: on day 13 after hospital admission, multiple organ failure progressed (acute kidney injury, hyperkalemia) due to sepsis, cytolytic syndrome; ECG results showed atrial fibrillation paroxysm with tendency to hypotonia, which required inotropic support initiation.

On day 14, the monitor showed asystole. Resuscitation was unsuccessful. Natural death was recorded.

Postmortem examination results confirmed infectious endocarditis (pink vegetations on MV endocardium (Figures 4, 6), detached MV chords (Figure 5), excessive growth of Pseudomonas aeruginosa, poor growth of Klebsiella pneumoniae and Acinetobacter baumannii); blood clot was found in the right atrial cavity. No pathological changes were found in the brain substance.

Final postmortem diagnosis:

Primary diagnosis: Infectious endocarditis of the mitral valve (microbiological examination of the heart valve No. 46518: excessive growth of Pseudomonas aeruginosa, poor growth of Klebsiella pneumoniae and Acinetobacter baumannii).

Primary diagnosis complications: Sepsis: bone marrow hyperplasis, enlarged spleen (190 g), bilateral multisegmental pneumonia (microbiological examination of the heart valve No. 46518: excessive growth of Pseudomonas aeruginosa, poor growth of Klebsiella pneumoniae and Acinetobacter baumannii). Critical illness polyneuropathy. Detached mitral leaflets. Blood clot in the right atrial appendage. Thromboembolism of segmental branches of the left pulmonary artery. Pulmonary oedema. Brain swelling.

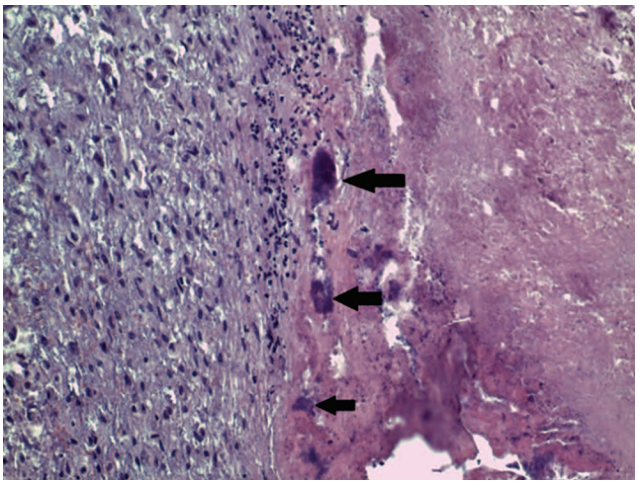

***Fig.** 6. Acute endocarditis with bacterial colonies (indicated by arrows). Staining with hematoxylin and eosin, magnification x200*

Secondary diagnosis: Arterial hypertension (heart weight: 360 g, left ventricle myocardial thickness: 1.6 cm, artery-arteriolar nephrosclerosis). Atherosclerosis of aorta (stage 4, grade 3), atherosclerosis of arteries in the base of brain (stage 1, grade 1), atherosclerosis of coronary arteries (stage 3, grade 3, 25% stenosis). Nodular prostatic hyperplasia.

Clinical Case Discussion

Often GBS develops one to three weeks after a past infectious disease caused by cytomegalovirus, influenza viruses, *zika* virus, etc., or a bacterial infection, such as *Campylobacter jejuni* and *Mycoplasma pneumoniae* [12]. Since 2020, the provoking infection list includes *SARS-CoV-2* [4]. In 2023, an overview was published on the development of GBS in patients after varicella-zoster virus infection [16]. There are individual reports on GBS in patients with acute infectious endocarditis of the intact [17] or prothetic [18] valves.

According to the patient, two weeks prior to hospitalisation, he had acute respiratory virus infection, when his body temperature rose to 37.3 °C, which was complicated by acute left-sided maxillary sinusitis and ethmoiditis (seen on brain CT upon admission).

In classical GBS, symptoms progress within two to four weeks, starting from the feet and progressing to hip muscles, hands, shoulder girdle, body and, in severe cases, throat muscles [2, 6]. In this patient, the symptoms developed much faster: for the first time, he experienced weakness in his lower limbs two days prior to hospitalisation; neurologist diagnosed motor dysfunction, more on the left side, weak reflexes on both sides; 12 hours later, mild dysphagia and dysarthria developed. Bowel and bladder functions were intact.

The severity of patient's condition was promoted by infectious endocarditis, diagnosed with the help of transthoracic echocardiography and confirmed with the help of transesophageal echocardiography. It is quite difficult to identify the time when the vegetation formed, because the patient denied body temperature hikes above 38 °C, Lukin spots or Janeway lesions or other signs of the disease before hospitalisation.

Infectious endocarditis can trigger various systemic complications. Septic embolism of vital organs, including brain, can develop approximately in 25% of cases. The risk of embolic complications is higher in staphylococcal infectious endocarditis, when vegetations are located on MV and/or if vegetation size exceeds 10 mm [17]. In this patient, vegetations were found on the anterior leaflet of the mitral valve, and they were larger than 10 mm. However, tests and examinations performed upon admission (brain CT and CT angiography of the intracranial section of brachiocephalic arteries) ruled out vascular embolism in the brain. Progressive weakness in the limbs, weak and then absent tendon reflexes, development of dysphagia and dysarthria, absence of CT signs of ischaemia, negative proserine test, analysis of the clinical analysis of spinal fluid (albuminocytologic dissociation), allowed diagnosing GBS and initiating successful immunomodulating therapy. In this case, the primary disease is more likely to be infectious endocarditis, which developed after a past acute respiratory virus infection with maxillary sinusitis and ethmoiditis; GBS is a concurrent disease. However, given the complex aetiopathogenesis mechanisms, GBS cannot be ruled out as one of the complex immune complications of acute/subacute infectious endocarditis.

In the majority of patients (approximately 75% of all cases), GBS is relatively asymptomatic, and its symptoms regress even without therapy. This patient benefited from immunomodulating therapy for the management of GBS. However, despite the antibacterial therapy, the patient developed sepsis with multiple organ failure and died.

Conclusion

Therefore, this is a clinical case study of a patient with infectious endocarditis of the mitral valve after a recent acute respiratory virus infection, complicated with left-sided maxillary sinusitis and ethmoiditis and Guillain-Barre syndrome. Clinical pathogenetic correlations and management of patients with the two severe conditions require further studies.

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