DOI: https://doi.org/10.17816/nb633510

Anti-NMDA receptor encephalitis fatal case was mistaken as a debut of schizophrenia: the devil is in the psychopathological details

Vladimir D. Mendelevich[1], Konstantin S. Sergienko[1], Kausar K. Yakhin[1], Elvira A. Abdullina[2]

[1] Kazan State Medical University, Kazan, Russia;
[2] Republican Clinical Psychiatric Hospital named after. V.M. Bekhtereva, Kazan, Russia

ABSTRACT

The article presents the clinical case of the patient Victor, 42 years old, who was admitted to a psychiatric hospital in an acute psychotic state with schizophreniform symptoms. His illness progressed rapidly, and at different stages, epileptic paroxysms and disorders of consciousness joined the symptoms typical of schizophrenia. It was then that the question arose of the need to revise the diagnosis of schizophrenia, to carry out a targeted neurological examination and to correct of the treatment regimen. Victor's case, which ended in death, raises questions for psychiatrists about finding significant differential diagnostic criteria for distinguishing schizophrenia from schizophrenia-like psychoses. To avoid mistakes, it is proposed to rely on the so-called. "diagnostic clues" and "red flags" in identifying the psychopathology of anti-NMDA receptor encephalitis.

Keywords: anti-NMDA receptor encephalitis; schizophrenia; schizophreniform symptoms; catatonia; psychiatric diagnosis.

To cite this article:
Mendelevich VD, Sergienko KS, Yakhin KK, Abdullina EA. Anti-NMDA receptor encephalitis fatal case was mistaken as a debut of schizophrenia: the devil is in the psychopathological details. *Neurology Bulletin.* 2024;56(3):299–310. DOI: https://doi.org/10.17816/nb633510

Received: 17.06.2024 **Accepted:** 29.06.2024 **Published online:** 17.09.2024

DOI: https://doi.org/10.17816/nb633510

Случай летального исхода анти-NMDA-рецепторного энцефалита, ошибочно принятый за дебют шизофрении: дьявол кроется в психопатологических деталях

В.Д. Менделевич[1], К.С. Сергиенко[1], К.К. Яхин[1], Э.А. Абдуллина[2]

[1] Казанский государственный медицинский университет, Казань, Россия;
[2] Республиканская клиническая психиатрическая больница им. В.М. Бехтерева, Казань, Россия

АННОТАЦИЯ

В статье приведён клинический случай Виктора 42 лет, поступившего в психиатрический стационар в остром психотическом состоянии с шизофреноформной симптоматикой. Его болезнь развивалась стремительно, а на отдалённых этапах к симптомам, типичным для шизофрении, присоединились эпилептические пароксизмы и расстройства сознания. Именно тогда встал вопрос о необходимости пересмотра диагноза шизофрении, целенаправленного неврологического обследования и коррекции схемы лечения. Случай Виктора, закончившийся летальным исходом, ставит перед психиатрами вопросы о поиске значимых дифференциально-диагностических критериев для отграничения шизофрении от шизофреноподобных психозов. Для исключения ошибок предложено опираться на так называемые «диагностические подсказки» и «красные флаги» в выявлении психопатологии анти-NMDA-рецепторного энцефалита.

Ключевые слова: анти-NMDA-рецепторной энцефалит; шизофрения; шизофреноформная симптоматика; кататония; психиатрическая диагностика.

Как цитировать:
Менделевич В.Д., Сергиенко К.С., Яхин К.К., Абдуллина Э.А. Случай летального исхода анти-NMDA-рецепторного энцефалита, ошибочно принятый за дебют шизофрении: дьявол кроется в психопатологических деталях // Неврологический вестник. 2024. Т. 56, № 3. С. 299–310. DOI: https://doi.org/10.17816/nb633510

Рукопись получена: 17.06.2024 **Рукопись одобрена:** 29.06.2024 **Опубликована online:** 17.09.2024

DOI: https://doi.org/10.17816/nb633510

Анти-NMDA-рецепторлы энцефалитның үлемгә китерә торган очрагы: иблис психопатологик детальләргә яшеренә

В.Д. Менделевич[1], К.С. Сергиенко[1], К.К. Яхин[1], Э.А. Абдуллина[2]

[1] Казан дәүләт медицина университеты, Казан, Рәсәй;
[2] В.М. Бехтерев ис. республика клиник психиатрия хастаханәсе, Казан, Рәсәй

АННОТАЦИЯ

Мәкаләдә шизофрения билгеләре күзәтелгән кискен психотик халәттә психиатрия стационарына китерелгән 42 яшьлек Викторның клиник очрагы китерелә. Аның авыруы бик тиз көчәя, соңга таба шизофрениягә хас типик симптомнарга эпилепсия пароксизмнары һәм акыл ягыннан тайпылышлар да өстәлә. Нәкъ менә шул вакытта шизофрения диагнозын яңадан карау, максатчан неврологик тикшеренүләр уздыру һәм дәвалау схемасын коррекцияләү зарурлыгы туа. Викторның үлеме белән тәмамланган әлеге очрак психиатрлар алдында шизофренияне шизофрения сыман психозлардан аера белүдә гаять әһәмиятле булган дифференциаль-диагностика критерийларын булдыру-эзләү мәсьәләсен куя. Ялгыш нәтиҗәләрне булдырмас өчен, анти-NMDA-рецепторлы энцефалитның анти-nmda-рецептор энцефалиты психопатологиясен ачыклауда «диагностик киңәшләр» һәм «кызыл флаглар» га таянырга тәкъдим ителә.

Төп төшнчәләр: анти-NMDA-рецепторлы энцефалит; шизофрения; шизофрения формалы симптоматика; кататония; психиатрия диагностикасы.

Өземтәләр ясау өчен:
Менделевич В.Д., Сергиенко К.С., Яхин К.К., Абдуллина Э.А. Анти-NMDA-рецепторлы энцефалитның үлемгә китерә торган очрагы: иблис психопатологик детальләргә яшеренә // Неврология хәбәрләре. 2024. Т. 56, Чыг. 3. С. 299–310. DOI: https://doi.org/10.17816/nb633510

Кулъязма алынган: 17.06.2024 **Кулъязма хупланган:** 29.06.2024 **Бастырылган online:** 17.09.2024

In recent years, there has been a notable increase in schizophreniform syndromes in clinical practice, presenting a challenge for diagnosticians. The most frequently reported cases involve schizophreniform disorders associated with psychoactive substance use (most commonly cannabinoids), COVID-19[1], and encephalitis (e.g., limbic encephalitis) [1–4].

Diagnostic challenges often arise when psychopathological symptoms develop acutely, especially in the absence of timely paraclinical verification of the condition. Such cases can be dangerous due to possible somatic complications and, in some instances, even death. Typically, these psychopathological symptoms are treated using standard psychopharmacological antipsychotic regimens. However, when the encephalitic origin of acute schizophreniform psychosis is identified, the patient needs a fundamentally different therapeutic approach. In such cases, accurate diagnosis becomes crucial for effective treatment and preventing tragic outcomes.

This is a clinical case involving Victor[2], a 42-year-old patient admitted to a psychiatric hospital in an acute psychotic state with schizophreniform symptoms. His condition developed rapidly, and in the later stages, he experienced epileptic paroxysms and disorders of consciousness, alongside the typical symptoms of schizophrenia. At this point, the need to reconsider the initial diagnosis of schizophrenia arose, prompting a targeted neurological examination and treatment adjustment.

Victor's fatal case raises questions for psychiatrists regarding the search for significant differential diagnostic criteria for distinguishing schizophrenia from schizophrenic-like psychoses. To eliminate diagnostic errors, it has been proposed to rely on the so-called "diagnostic clues" and "red flags" when assessing the psychopathology of anti-NMDA receptor[3] encephalitis [5].

Patient Victor was 42 years old. Victor was urgently admitted to the emergency department of a psychiatric hospital on January 11, 2024, on the referral of a district psychiatrist. The preliminary diagnosis was "Schizophrenia, debut?" He did not actively complain, but his behavior was purposeless and disorganized. The psychiatrist noted that "the patient's orientation is inadequate, his answers are irrelevant, he moralizes, talks to himself, and mutters something about 'voices' inside his head".

Anamnesis of life. There was no family history of mental illness. His mother's pregnancy and childbirth were without complications, and he was born on time as the younger of two children (his older brother is 48 years old). His early development was unremarkable. He attended preschool and began school at the age of 7. He completed the 11th grade and later studied at an Aviation Institute but did not complete his studies. During his academic years, he took two leaves of absence to balance his studies and work.

Victor has been married since 2003 and has a 7-year-old daughter. Family relations were described as "very warm". According to his wife, "Victor is kind, shy, afraid to harm anyone — even stepping over worms — gentle, very responsible, and careful. He is always concerned about the well-being of his family and loved ones". In recent years, Victor has been working as an individual entrepreneur, conducting business primarily from home.

There were no special stressful situations in Victor's life, though he was still grieving the death of his father from COVID-19 in 2020. Every year on the day of remembrance of his father, Victor would travel alone to another region to visit his grave. He drank alcohol only on holidays in small amounts. He smoked until his mental state changed. He denied taking psychoactive substances.

Anamnesis morbi. Victor's mental state began changing two weeks before the forced hospitalization in a psychiatric hospital. On December 29, 2023, he was actively working, preparing an accounting report, which kept him glued to his computer for long hours leaving the room only when necessary. His wife noted a change in his behavior during this time: he slept less, overworked, but at the same time seemed cheerful. She was puzzled, describing it as though "he was drinking too much coffee". On the night of December 30th to 31st, Victor slept only 2–3 hours before a planned trip with his family to visit relatives in a neighboring city. He drove a car all day (December 31) for almost 700 km. At that time, his wife observed that his mental state was unusual: he was overly tense and worried about potential accidents, a stark contrast to his typical calmness behind the wheel, given his extensive driving experience.

At his relatives' house, Victor remained overexcited and anxious. He drank alcohol moderately on New Year's holidays, but by the morning of January 1, he complained of "general soreness in the head". The surrounding people attributed this to a hangover and recommended drinking alcohol, which Victor refused, and by the evening, he "felt better". The next day in the bathhouse, he complained of "pain in the forehead area on the left and unpleasant sensations in the left eye", which resolved its own.

During the drive back home on January 3, his anxiety intensified. He became extremely tense and anxious and clutched the steering wheel. Noticing Victor's poor mental state and fearing an even greater deterioration, his wife suggested they stop in the city halfway to rest. While walking around the city, he continued to behave strangely — holding his wife's hands tightly, looking around in fear, and becoming anxious when crossing the road. Upon returning home, Victor breathed a sigh of relief and became overly joyful. He expressed pleasure that they "returned alive", did not stop

[1] COVID-19 (from Coronavirus Disease 2019) is a 2019 coronavirus infection.

[2] The name has been changed.

[3] NMDA is an N-methyl-D-aspartic acid.

hugging his wife and daughter, and even expressed his love for his mother-in-law, despite their rather cold relationship.

On the night of January 6–7, Victor barely slept, sitting in front of the TV with a vacant stare, unresponsive to conversations or remarks. His mental state fluctuated between periods of detachment and brief moments of engagement. During his detached episodes, there was no muscle tension or stiffness. Over time, the light intervals shortened, and his speech became more disjointed. For example, he said that he could not drive a car. He became unavailable for productive contact, was immersed in his thoughts, and sat in a monotonous pose.

On January 7, while visiting his mother-in-law, he stopped talking to anyone, answered questions in monosyllables, and passively agreed if asked about something. His mother-in-law suspected he was having a stroke, and on the same day, his wife called an ambulance. Before the ambulance arrived, Victor behaved even more strangely — he decided to wipe the floors and took a rag, but immediately forgot what he was doing and wandered the apartment in confusion.

At the emergency department of the somatic hospital, imaging studies (X-ray computed tomography) showed no evidence of an acute cerebrovascular accident or subarachnoid hemorrhage. He was diagnosed with "G90. Syndrome of autonomic dysfunction". However, admission to a neurological hospital was refused.

After returning from the hospital, Victor suddenly began expressing suicidal thoughts, stating that he would die soon. He continued to experience "bouts of stupor", after which he could not remember what was happening to him, did not understand what was happening, and was confused. When his wife inquired about the probable causes of his anxiety, he replied "this is a trigger", without explaining in any way what it meant. On January 9, Victor reported having a panic attack in the morning. He was visibly tense again and shackled, with cold, white hands — a stark contrast to his typical anxious state, where he would usually sweat excessively.

Escalation of Symptoms and Psychiatric Evaluation. Following his wife's insistence, Victor was seen by the district psychiatrist. During the consultation, he presented as inhibited, with pronounced stiffness and tension. There were no indications for hospitalization, and the psychiatrist recommended diazepam at a dosage of 5 mg/day).

According to his wife, Victor initially appeared to return to his "usual state", seeming more like himself on January 10 after following the prescribed medication. However, by evening, the condition changed again — he showed excessive motor activity and was fussy, with accelerated speech, an unusual "gluttony" ("he ate buckwheat like a horse"), and an inability to sit still, performing several actions at once. The diazepam taken in the morning had no effect.

Victor began to express suicidal thoughts again, prompting a second visit to the district psychiatrist. During the January 11 consultation, his behavior was inappropriate — he withdrew and stopped responding to questions. An electroencephalography (EEG) was performed, which revealed slight cerebral changes in cortical rhythmics, including alpha rhythm disorganization and polymorphic delta deceleration in the frontal lobe, predominantly on the left. Magnetic resonance imaging (MRI) of the brain was recommended.

Hospital Admission and Initial Assessment. Considering the detected mental disorders, the patient was admitted to the hospital. Upon admission, his body temperature was 37.4°C. Tests for COVID-19 and type A flu were negative, and he was diagnosed with acute respiratory viral infections, for which antiviral treatment was prescribed.

Mental Status on Admission. Viktor appeared outwardly neat but agreed to the conversation only passively. His facial expressions were extremely limited, and he maintained a calm demeanor until the discussion turned to the reasons for his hospitalization, at which point he strained and began to frown. His emotional state at the time of the examination appeared neutral. He maintained brief eye contact but mostly looked at the floor. He exhibited minimal, repetitive gestures such as rubbing a corn on his finger.

He reacted to the addressed speech only after long pauses and required external stimulation in the form of repeated questions. It seemed as if he was attempting to respond (his mouth would move and facial expressions changed), but ultimately, most of the questions went unanswered. He nodded when asked to continue the conversation at a later time.

After a while, Viktor approached the doctor in the corridor, requesting another conversation. During this follow-up, he struggled to organize his thoughts: "It's hard to get together like this right away". He apologized to the doctor, without elaborating further. His speech was fragmented and cryptic: "I've decided directly and clearly... Apologize to the family... I promise not a finger, no way... I'm not harming... I'm not a threat". When pressed to clarify these statements, he was unable to provide any explanation; he replied: "It's not the doctor who thinks — I think". His speech was of moderate volume, slow, and agrammatic.

His attention was unstable and exhausted. His thinking is inconsistent and paralogical, such as claiming that his 48-year-old older brother was 7 years old. When questioned about this inconsistency, he simply confirmed his earlier statement without offering any rationale.

The psychopathological condition of the patient upon admission to the hospital was regarded as catatonic with severe anxiety and marked by hallucinatory experiences. His symptoms included mutism and negativism, suggestive of catatonic manifestations. Given the presence of thinking disorders, along with delusional ideas of harming family members and hallucinations he had seemingly reported to the district psychiatrist, the following treatment plan was initiated: a solution of bromodihydrochlorophenylbenzodiaz epine (0.1%; 3 ml/day) to alleviate the catatonic symptoms and a haloperidol solution (0.5%; 2 ml/day) to address the psychotic symptoms, particularly the hallucinations and paranoid ideation.

Viktor was treated for almost two weeks, from January 11 to January 24, but his mental state remained unstable. Although some improvement was noted, his symptoms fluctuated significantly. He showed persistent polymorphic symptoms. Confusion, disorganized thinking, answering questions at random, anxiety, and mutism (taking a long time to choose words) were noted. His handwriting became erratic, and the grammatical and semantic structures were impaired. He struggled to read, often syllabifying words and losing the meaning of what he read. By the end of the 10th day of the hospital stay, there were signs of improvement — his thinking became more consistent, and his behavior was more appropriate. The reason for this change was again called "a trigger in the form of an evening with friends". However, this progress was followed by a relapse when Victor unexpectedly mentioned drug use, though no one confirmed this.

On the 12th day (January 23) of hospitalization, Viktor's condition changed dramatically. He developed an increase in body temperature to 38.7°C, tonic-clonic seizures, vomiting, and urinary incontinence. He became completely unresponsive and showed severe disorientation — moving between beds, interacting with invisible objects, and showing erratic, catatonic behavior.

His condition changed constantly throughout the day. Despite the persistence of mutism, perseverations were noted, along with catatonic, apathetic, and hebephrenic features. He also exhibited pronounced negativism and hallucinatory experiences. His consciousness fluctuated, ranging from mild stun to amentia. Due to the change in the condition, neuroleptic therapy was canceled, and infusion therapy was started (0.9% sodium chloride solution up to 750 ml/day).

The psychiatrists raised doubts about the initial diagnosis of schizophrenia, given the polymorphic nature of Victor's symptoms, including the mix of catatonic, hebephrenic, and aphasic elements. They hypothesized an exogenous-organic genesis. To clarify the diagnosis, excluding the volumetric process, COVID-19, and the current neurological disease, MRI of the brain and transfer to the intensive care unit were recommended.

On January 26, Victor regained consciousness during the transfer, got up on his own, refused to be put on a "carrier", and with the support of others, he reached the ambulance. He recognized his wife but did not answer any questions. In the department, the condition was assessed as extremely severe, due to respiratory failure of the second degree and cardiovascular insufficiency. Preliminary diagnosis of the intensive care unit was as follows: new coronavirus infection smear positive for COVID-19 with severe encephalopathy in the form of microfocal symptoms and pyramidal insufficiency; epi-paroxysm; schizophrenia (debut); possible febrile catatonia.

Treatment with methylprednisolone and human immunoglobulin was initiated. However, despite therapy, his condition continued to worsen, and his fever persisted (around 38°C). His blood pressure remained elevated at 150/100 mm Hg. His level of consciousness deteriorated to sopor-stunned. When his consciousness cleared, he exhibited pronounced anxiety, fear, incoherent cries, psychomotor agitation, and insomnia.

To manage these symptoms, diazepam (0.5%; 2 ml) was administered. However, pronounced rigidity of the occipital muscles, hyperthermia, greasy skin, and tonic-clonic twitching were noted. His condition was so severe that productive contact with him became impossible, requiring nutrition through a nasogastric tube and urinary management via a catheter. A drop in oxygen saturation to 80% prompted his transfer to artificial lung ventilation.

An MRI of the brain, conducted on January 29, showed no signs of a volumetric process, focal pathology of the brain, or acute cerebrovascular accidents. However, it revealed small atrophic changes in the frontal-parietal regions of the cerebral hemispheres.

A cerebrospinal fluid analysis, performed on January 30, revealed weak opalescence (fibrinogen/high molecular weight proteins; class G immunoglobulins), lymphocytic pleocytosis, hyperproteinarchy (inflammation), and hyperglycoarchy (cerebral ischemia/meningoencephalitis). Signs of cellular protein dissociation strongly suggested meningoencephalitis.

Further biochemical blood analysis revealed a moderate cytolysis syndrome, cholestasis (direct bilirubin 15.84 mmol/l), protein-synthetic insufficiency, and moderate hypokalemia (risk of hypoventilation). A coagulogram showed possible signs of disseminated intravascular coagulation (DIC) syndrome[4], liver failure, and heparin side therapy.

Following a consultation with a clinical pharmacologist on January 30, the diagnosis pointed to signs of meningoencephalitis of unknown origin. Given the presence of meningeal signs, previous disorders, and convulsive seizures, the pharmacogenic nature of the condition cannot be excluded (malignant neuroleptic syndrome?).

On January 31, neuroimaging suggested that the patient had a viral brain lesion. The side effects of antipsychotic therapy could have been exacerbated by decreased drug tolerability due to the underlying infection. By February 1, laboratory indicators showed no signs of rhabdomyolysis, leading to the assumption that the patient had a febrile attack of catatonic schizophrenia. However, this condition needed to be differentiated from autoimmune encephalitis.

The neurologist's conclusion on February 1 noted the following: "Considering the acute onset of the disease with psychotic symptoms, personality changes, the appearance of convulsive seizures (generalized tonic-clonic), poor tolerability and ineffectiveness of neuroleptic therapy

[4] DIC is disseminated intravascular coagulation.

(suggesting malignant neurolepsy), previous flu-like symptoms (headache, elevated body temperature), the development of vegetative disorders (requiring oxygen therapy, sweating), cerebrospinal fluid results, and EEG data, a diagnosis of limbic encephalitis (anti-NMDA) can be considered".

A cerebrospinal fluid sample was taken to test for autoimmune antibodies, and the patient was recommended for transfer to the neurological department, with continued care in the intensive care unit of a multidisciplinary hospital. In the intensive care unit, the patient received a range of treatments, including prednisone, dexamethasone, heparin, furosemide, meropenem, amikacin, potassium chloride, mannitol, favipiravir, immunoglobulin, and oxycarbamazepine.

On February 13, the cerebrospinal fluid test confirmed the presence of (8) anti-NMDA receptor antibodies. Despite intensive therapy, the patient's condition worsened, with pronounced respiratory disorders, impaired consciousness leading to coma, and vegetative dysautonomia. The patient suffered respiratory arrest on February 6, followed by cardiac arrest on February 14 and 16. He died on February 16, 2024.

Victor's clinical case, which led to death from unrecognized anti-NMDA receptor encephalitis, is unfortunately a fairly common diagnostic error [6–18]. This case raises several important practical and theoretical questions.

The main question is whether psychiatrists, when presented with schizophreniform symptoms, could initially identify the typical psychopathological signs of autoimmune encephalitis. It is important to understand exactly which psychopathological features make it possible to differentiate between schizophreniform symptoms related to anti-NMDA receptor encephalitis and those signaling the onset of schizophrenia. The most significant theoretical problem is finding evidence of the existence of so-called febrile (hypertoxic) schizophrenia in the form of "fatal catatonia" and whether this concept still holds legitimacy in psychiatry.

From the analysis of Victor's clinical case, his illness had an acute onset (within a few days), progressing rapidly for one and a half months. What began as symptoms of anxiety, insomnia, "flickering" orientation disorders, and qualitative disorders in thinking (disconnectedness, incoherence, and verbigerations) eventually developed into a severe condition. Initially, there were isolated motor disorders, particularly involving facial muscles. Over time, his symptoms evolve into states described as catatonic, hallucinatory, and hebephrenic. Later, epileptic paroxysms and disorders of consciousness (amenia) were also observed, marking the progression of his illness.

The progression of psychopathological symptoms occurred alongside a low-grade fever that later escalated to febrile temperatures but without any obvious neurological symptoms. Neurologists had already excluded acute cerebral circulatory disorders. Diagnostic difficulties were apparent at the onset of the disease, whereas at the advanced stage, with the appearance of new ("obvious") symptoms, making a diagnosis became easier.

The directional diagnosis ("schizophrenia, debut?"), which later proved incorrect, was based on observations of pronounced thinking disorders in the patient, including rupture, inadequacy of affect, apathy, abulia, and a condition that was regarded as "mutism". No other symptoms typical of schizophrenia were found. The only instance that hinted at potential psychotic symptoms was when the patient briefly mentioned hearing a "voice inside his head", which he never mentioned again, and no behavioral evidence corroborated this.

Attention should be paid to the fact that upon admission to the hospital, outside of neuroleptic therapy, the patient had violent "mouth movements and facial expression changes", which the doctors regarded as mutism (i.e., appearing as if trying to respond but unable to do so). As the illness progressed, the attending physicians began to note catatonic symptoms such as negativism and impulsivity, while some psychiatric consultants pointed out the presence of "waxy flexibility".

In retrospect, the clinical picture was dominated by polymorphic psychopathological symptoms at a psychotic level, without any critical insight from the patient about his condition. Many of the observed symptoms can now be interpreted differently from what was indicated in the patient's medical history. For example, what was seen as disconnected, incoherent thinking, speech disorders (such as mumbling and agrammatism), episodes of detachment and orientation disorders, and motor disorders affecting facial muscles could suggest a different pathological process than schizophrenia, such as an underlying organic or autoimmune encephalitic disorder.

The treating psychiatrists regarded the thought disturbances as typical of schizophrenia, interpreting them as signs of disconnectedness. However, based on medical history, these disturbances reflected incoherence rather than disconnectedness. Moreover, they were episodic rather than permanent, which is atypical for schizophrenia. It is essential to differentiate between thinking disorders and speech disorders; it is possible that the observed incoherence was indicative of a "flicker of consciousness" and was a symptom of motor aphasia. It can be assumed that the same neurological phenomenon was reflected in the motor disorders of the facial muscles, known as orofacial dyskinesia.

Attention is drawn to the atypical nature of the symptoms described as catatonic by the attending physicians. For example, the mutism observed within the context of catatonia should not include the resumption of periodic verbal contact with others or "fruitless striving" to answer the questions asked. In addition, the patient had no obvious psychopathological symptoms typically deemed significant for diagnosing schizophrenia; there were no Schneider's First Rank Symptoms , no pseudohallucinations, delusions of exposure, or other forms of delirium. The patient also lacked the diagnostic criteria for the catatonic form of schizophrenia;

there was no stupor, arousal, or distinct congestion noted. "Waxy flexibility" was observed briefly on January 25-26, where the patient displayed an "air cushion" effect for several minutes.

According to D.I. Malin et al. [19], catatonic symptoms in anti NMDA-receptor encephalitis tend to present more expansively, including features such as negativism, autonomic dysfunction, lethargy, stupor, mutism, and, less often, impulsivity, stereotypies, catalepsy, echolalia, and echopraxia. Therefore, Victor's catatonic symptoms were not only atypical for schizophrenia but also differed from those typically seen in anti-NMDA-receptor encephalitis. This disparity may have contributed to the delayed recognition of his underlying condition.

Psychiatrists who observed Victor recommended a differential diagnosis between the onset of schizophrenia, febrile schizophrenia ("fatal catatonia"), malignant neuroleptic syndrome, and, only later, autoimmune encephalitis. However, it is noteworthy that none of the listed mental disorders showed a typical clinical picture that would allow for a definitive diagnosis. While polymorphic symptoms can manifest at the onset of schizophrenia, they should include a set of affective-paranoid symptoms with specific dynamics and the addition of oneiroid symptoms, rather than an agentive disorder of consciousness. In cases of catatonic schizophrenia, the dominant disorder of consciousness is also oneiroid and is combined with motor disorders, which were absent in Victor's clinical presentation.

Psychiatrists ignored the fact that before his hospitalization and during his stay, the patient had distinct periods of undulation characterized by sharp fluctuations in psychopathological symptoms and, most likely, consciousness. The patient's wife referred to these fluctuations as periods of "detachment" alternating with times of complete return to normalcy. Such patterns are not typical for schizophrenia, especially since they were not accompanied by the diagnostic criteria significant for schizophrenia. Thus, the atypical clinical picture of the patient's disease could already, at the initial stage become a "diagnostic clue" and the basis for searching for exogenous organic causes of his condition, in particular those associated with autoimmune encephalitis.

It is known that anti-NMDA-receptor encephalitis, as a rule, manifests with psychopathological symptoms similar to those of schizophrenia. At the initial stages, patients are usually hospitalized in a psychiatric hospital, and specific dynamics of the condition are identified, as was the case with Victor (Fig. 1) [20].

Most often, at the initial stage, such patients are diagnosed with "psychotic disorder" (37.9%), "bipolar affective disorder" (25.9%), or "schizophrenia/schizoaffective/delusional disorder" (10.3%) [21].

Victor's neuropsychiatric state coincided with the typical course of anti-NMDA-receptor encephalitis, starting with flu-like precursory symptoms and subfebrile body temperature, followed by the emergence of mental disorders such as anxiety, fluctuations in consciousness, disorientation, and psychomotor agitation, along with epileptic seizures. Researchers highlighted the significance of "paroxysmal speech disorders" as specific symptoms of autoimmune encephalitis [22, 23], a phenomenon our patient also exhibited. According to C.C. Hu et al. [22], this phenomenon should be closely monitored in the initial stages of anti-NDMA-receptor encephalitis to exclude tragic diagnostic errors.

It is believed that the characteristic symptoms that raise suspicion for anti-NMDA-receptor encephalitis in the early stages include not only speech disorders but also cognitive dysfunction, seizures, motor disorders, dyskinesia or rigidity/

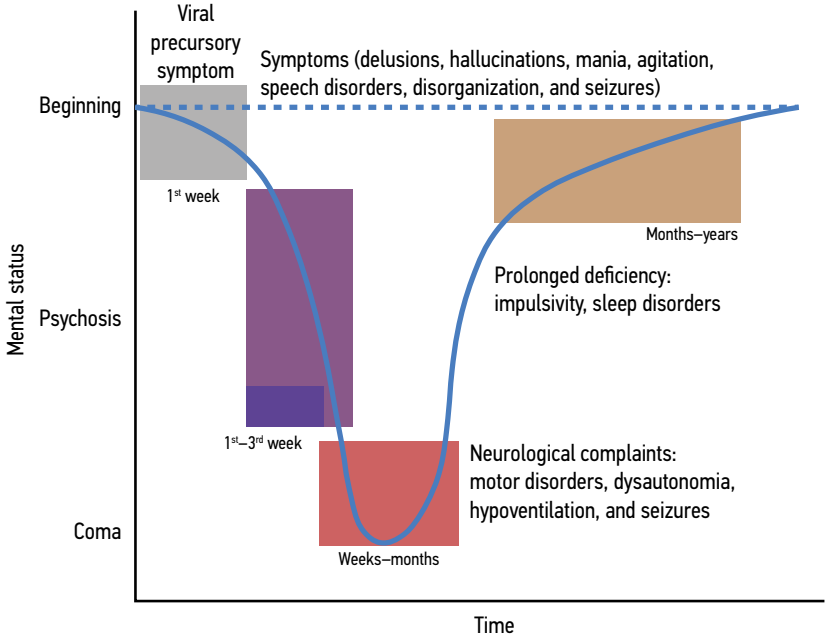

Fig. 1. Dynamics of psychoneurological symptoms in anti-NMDA receptor encephalitis [20].

abnormal postures, and a decrease in consciousness. Among the psychopathological syndromes, thinking disorders, delusions, hallucinations, psychomotor agitation, and "hostility" develop more often than others [19].

As the analysis of Victor's clinical case shows, most of the listed symptoms were present at the onset of the disease. However, some symptoms were not properly evaluated, and others were ignored. In particular, there were insufficient grounds to establish the presence of catatonic or hallucinatory symptoms, and the undulating nature of his consciousness (referred to by his wife as "detachment") was not considered. In addition, a symptom characterized by violent movements of the facial muscles was misinterpreted; the attending physicians regarded this phenomenon as a manifestation of mutism, although the patient showed all the signs of orofacial dyskinesia.

The diagnosis of "schizophrenia, debut?" in Victor was largely justified by the discovery of qualitative thinking disorders characterized by discontinuity. While this clinical phenomenon is included in the list of diagnostically significant symptoms for schizophrenia, it appeared more like incoherence in Viktor's case. This could suggest not a genuine disturbance in thinking but rather speech disorders, which, as mentioned earlier, align with the diagnostic significance associated with anti-NDMA-receptor encephalitis.

Numerous scientific papers are devoted to the topic of discontinuity and incoherence of thinking, suggesting that these symptoms can manifest as non-schizophrenic when no other diagnostically significant symptoms of schizophrenia are present. In addition, for an agentive disorder of consciousness, such thinking disorders are typically required. Therefore, it can be assumed that the discontinuity of thinking and incoherence observed in Victor were signs of a paroxysmally occurring agentive disorder of consciousness.

It is believed that anti-NMDA-receptor encephalitis can mimic various mental disorders, but individual "specific" symptoms are critically important for its detection [21]. A combination of characteristics such as acute disease onset, catatonic symptoms, memory disorders, an undulating course of symptoms, and a lack of response to psychopharmacotherapy is recognized as diagnostically significant. Approximately 91% of patients have at least two of these features, while 44% of patients have three or more. From our point of view, the most critical diagnostic criterion is the undulating nature of the psychopathological symptoms, which is extremely rare in other mental disorders, such as schizophrenia.

It is noteworthy that the psychiatrists involved in Victor's case raised the question of the presence of so-called febrile schizophrenia and later recommended differentiating it from manifestations of a brain tumor, malignant neuroleptic syndrome, and encephalitis. The concept of febrile (hypertoxic) schizophrenia, which arose during the Soviet period of psychiatric development, was the result of the transformation of the well-known concept of "fatal catatonia" outside the framework of schizophrenia [24–27]. In Russian literature, this disorder belonged (and still belongs) to the circle of schizophrenia, whereas modern scientific research confirms the transdiagnostic significance of the phenomenon of febrile catatonia [7].

The typical clinical picture of anti-NMDA-receptor encephalitis suggests the detection of a developed catatonic syndrome occurring alongside febrile body temperature, rather than isolated or partial catatonic symptoms. In Victor's case, the catatonic symptoms were of an undeveloped and abortive nature, and a significant increase in body temperature was not synchronized with the onset or progression of his psychopathological symptoms. Thus, the rationale for differentiating his psychosis from febrile schizophrenia seems poorly supported from a theoretical point of view, especially since this form of schizophrenia is absent in modern classifications. The belief among the treating psychiatrists in the existence of such a form of schizophrenia reduced their wariness about a different interpretation of the etiopathogenesis of the developed psychosis.

The analysis of Victor's clinical case, where schizophrenia was misdiagnosed and led to his death from anti-NMDA-receptor encephalitis, dictates the need for closer attention and caution about the possibility of encephalitic genesis of acute schizophreniform symptoms. The presented analysis of his clinical case is aimed at challenging the "schizophrenocentric" approach [28–29] to diagnosis, reconsideration of some dogmas that have survived to date in understanding schizophrenia, and drawing lessons from the tragic outcome in Victor's case.

While it is recognized that when polymorphic schizophreniform symptoms appear in patients within early psychotic episode units, several laboratory studies (such as MRI, EEG, and cerebrospinal fluid analysis) are necessary, studies have proved that there is no reason to conduct these tests in all patients with primary psychosis. Anti-NDMA-receptor encephalitis is detected in less than 1% of such patients [30], making broad application of these diagnostic measures unnecessary in routine settings.

ADDITIONAL INFORMATION

Funding source. The work was conducted and the article was published using the personal funds of the author's team.

Competing interests. The authors declare that there is no potential conflict of interest requiring disclosure in this article.

Authors' contribution. V.D. Mendelevich — clinical study, literature review, case analysis; K.S. Sergienko, K.K. Yakhin, E.A. Abdullina — clinical examination.

REFERENCES

1. Bravve LV, Zaharova NV. COVID-19-associated schizophrenia-like psychosis. *Psychiatry (Moscow).* 2022;20(4):44–53. (In Russ.) EDN: QJBBOT doi: 10.30629/2618-6667-2022-20-4-44-53

2. Veraksa AE. On the question of schizophrenic reactions. *Neurology Bulletin.* 2020;(4):75–82. EDN: SQMUPZ doi: 10.17816/nb52601

3. Mendelevich VD. Psychotic disorders due to drug use: current state of the problem. *Narkologiya.* 2014;13(7):93–100. (In Russ.) EDN: SJXQHB

4. Al-Diwani A, Handel A, Townsend L, et al. The psychopathology of NMDAR-antibody encephalitis in adults: A systematic review and phenotypic analysis of individual patient data. *Lancet Psychiatry.* 2019;6(3):235–246. doi: 10.1016/S2215-0366(19)30001-X

5. Martínez MR, Bautista GP, Espínola-Nadurille M, Bayliss L. Red flags for suspecting anti-NMDAr encephalitis in a first psychotic episode: report of two cases. *Rev Colomb Psiquiat.* 2019;48(2):127–130. doi: 10.1016/j.rcp.2017.10.002

6. Beattie M, Goodfellow J, Oto M, Krishnadas R. Anti-NMDAR encephalitis for psychiatrists: The essentials. *BJPsych Bull.* 2022;46(3):235–241. doi: 10.1192/bjb.2021.35

7. Volkov VP. Febrile catatonia. *Social and Clinical Psychiatry.* 2012;22(2):16–20. (In Russ.) EDN: PBDQNV

8. Malin DA, Malina DD. Febrile catatonia syndrome in critical conditions in psychiatry. *Social and Clinical Psychiatry.* 2019;29(1):82–89. (In Russ.) EDN: ZDSVTV

9. Malin DI, Gladyshev VN. Neuroleptic malignant syndrome or autoimmune anti-nmda receptor encephalitis? Analysis of a fatal clinical case. *Social and Clinical Psychiatry.* 2017;27(1):62–68. (In Russ.) EDN: YIZWSR

10. Kato R, Takenaka R, Matsumoto T. The phenomenology of anti-NMDA receptor encephalitis: A comparison with "primary mental confusion" in late 19th century French psychiatry. *Front Biosci* (Landmark Ed). 2022;27(4):116. doi: 10.31083/j.fbl2704116

11. Ponte A, Brito A, Nóbrega C, et al. Catatonia in anti-N-Methyl-D-Aspartate (NMDA) receptor encephalitis misdiagnosed as schizophrenia. *Acta Med Port.* 2020;33(3):208–211. doi: 10.20344/amp.11077

12. Graus F, Titulaer MJ, Balu R, et al. A clinical approach to diagnosis of autoimmune encephalitis. *Lancet Neurol.* 2016;15(4):391–404. doi: 10.1016/S1474-4422(15)00401-9

13. Barry H, Hardiman O, Healy DG, et al. Anti-NMDA receptor encephalitis: An important differential diagnosis in psychosis. *Br J Psychiatry.* 2011;199(6):508–509. doi: 10.1192/bjp.bp.111.092197

14. Endres D, Leypoldt F, Bechter K, et al. Autoimmune encephalitis as a differential diagnosis of schizophreniform psychosis: Clinical symptomatology, pathophysiology, diagnostic approach, and therapeutic considerations. *Eur Arch Psychiatry Clin Neurosci.* 2020;270(7):803–818. doi: 10.1007/s00406-020-01113-2

15. Funayama M, Koreki A, Takata T, et al. Differentiating autoimmune encephalitis from schizophrenia spectrum disorders among patients with first-episode psychosis. *J Psychiatr Res.* 2022;151:419–426. doi: 10.1016/j.jpsychires.2022.05.008

16. Shmukler AB. Does new knowledge allow to improve the diagnosis of mental disorders: the problem of anti-nmda receptor encephalitis. *Neurology Bulletin.* 2024;56(1):95–104. (In Russ.) EDN: RWSPHE doi: 10.17816/nb625638

17. Murashko AA. Psychiatric disorders in autoimmune encephalitis. *Social and Clinical Psychiatry.* 2021;31(1):74–79. (In Russ.) EDN: ENASCT

18. Vasenina EE, Levin OS, Gan'kina OA, et al. Autoimmune anti-NMDA-R encephalitis. *SS Korsakov Journal of Neurology and Psychiatry.* 2017;117(2):110–116. (In Russ.) EDN: YHEQKX doi: 10.17116/jnevro201711721110-116

19. Malin DI, Gladyshev VN, Ravilov RS. Clinical and psychopathological manifestations in autoimmune NMDA-receptor encephalitis. *Social and Clinical Psychiatry.* 2020;30(2):71–79. (In Russ.) EDN: VKRDJK

20. Kayser MS, Dalmau J. Anti-NMDA receptor encephalitis in psychiatry. *Curr Psychiatry Rev.* 2011;7(3):189–193. doi: 10.2174/157340011797183184

21. Murashko AA. Anti-NMDA receptor encephalitis mimic psychiatric disorders. *Social and Clinical Psychiatry.* 2022;(4):59–67. (In Russ.) EDN: LJESPJ

22. Hu CC, Pan XL, Zhang MX, Chen HF. Paroxysmal speech disorder as the initial symptom in a young adult with anti-N-methyl-D-aspartate receptor encephalitis: A case report. *World J Clin Cases.* 2022;10(24):8648–8655. doi: 10.12998/wjcc.v10.i24.8648

23. Hettiarachchi YKK, Wanigasinghe J. Speech and language abnormalities and their outcomes in autoimmune encephalitis in children: A case series. *Sri Lanka Journal of Child Health.* 2023;52(1):114–117. doi: 10.4038/sljch.v52i1.10487

24. Yudin TI. Lethal forms of schizophrenia. *Soviet Psychoneurology.* 1939. № 4–5. C. 2–23. (In Russ.)

25. Tiganov AS. *Febrile schizophrenia. Clinic, pathogenesis, treatment.* Moscow: Medicine; 1982. 128 p. (In Russ.)

26. Tsygankov BD. *Clinical and pathogenetic regularities of development of febrile attacks of schizophrenia and the system of their therapy.* Moscow: ROP; 1997. 232 p. (In Russ.)

27. Dunaevskij VV, Kuznecov AV. Catatonia-evolution of views and current understanding (literature review). *VM Bekhterev Review of Psychiatry and Medical Psychology.* 2019;4(2):29–40. (In Russ.) EDN: MGCSDQ doi: 10.31363/2313-7053-2019-4-2-29-40

28. Mendelevich VD. *Terminological bases of phenomenological diagnostics in psychiatry.* Moscow: Gorodets; 2016. 128 p. (In Russ.)

29. Mendelevich VD. Overdiagnosis of schizophrenia as a cognitive distortion of the process of knowledge of clinical reality. *Neurology Bulletin.* 2023;55(1):5–14. (In Russ.) EDN: ZWAXRT doi: 10.17816/nb160308

30. Kelleher E, McNamara P, Dunne J. Prevalence of N-Methyl-d-Aspartate Receptor antibody (NMDAR-Ab) encephalitis in patients with first episode psychosis and treatment resistant schizophrenia on clozapine, a population based study. *Schizophr Res.* 2020;222:455–461. doi: 10.1016/j.schres.2019.11.023

СПИСОК ЛИТЕРАТУРЫ

1. Бравве Л.В., Захарова Н.В. COVID-19-ассоциированные шизофреноформные психозы // Психиатрия. 2022. Т. 20, № 4. С. 44–53. EDN: QJBBOT doi: 10.30629/2618-6667-2022-20-4-44-53

2. Веракса А.Е. К вопросу о шизофренических реакциях // Неврологический вестник. 2020. № 4. С. 75–82. EDN: SQMUPZ doi: 10.17816/nb52601

3. Менделевич В.Д. Психотические расстройства в результате употребления наркотиков: современное состояние проблемы // Наркология. 2014. Т. 13, № 7. С. 93–100. EDN: SJXQHB
4. Al-Diwani A., Handel A., Townsend L., et al. The psychopathology of NMDAR-antibody encephalitis in adults: A systematic review and phenotypic analysis of individual patient data // Lancet Psychiatry. 2019. Vol. 6, N 3. P. 235–246. doi: 10.1016/S2215-0366(19)30001-X
5. Martínez M.R., Bautista G.P., Espínola-Nadurille M., Bayliss L. Red flags for suspecting anti-NMDAr encephalitis in a first psychotic episode: Report of two cases // Rev Colomb Psiquiatr (Engl Ed). 2019. Vol. 48, N 2. P. 127–130. doi: 10.1016/j.rcp.2017.10.002
6. Beattie M., Goodfellow J., Oto M., Krishnadas R. Anti-NMDAR encephalitis for psychiatrists: the essentials // BJPsych Bull. 2022. Vol. 46, N 3. P. 235–241. doi: 10.1192/bjb.2021.35
7. Волков В.П. К вопросу о фебрильной кататонии // Социальная и клиническая психиатрия. 2012. Т. 22, № 2. С. 16–20. EDN: PBDQNV
8. Малин Д.А., Малина Д.Д. Синдром фебрильной кататонии при критических состояниях в психиатрии // Социальная и клиническая психиатрия. 2019. Т. 29, № 1. С. 82–89. EDN: ZDSVTV
9. Малин Д.И., Гладышев В.Н. Злокачественный нейролептический синдром или аутоиммунный анти-NMDA-рецепторный энцефалит? (разбор клинического случая с летальным исходом) // Социальная и клиническая психиатрия. 2017. Т. 27, № 1. С. 62–68. EDN: YIZWSR
10. Kato R., Takenaka R., Matsumoto T. The phenomenology of anti-NMDA receptor encephalitis: A comparison with "primary mental confusion" in late 19th century French psychiatry // Front Biosci (Landmark Ed). 2022. Vol. 27, N 4. P. 116. doi: 10.31083/j.fbl2704116
11. Ponte A., Brito A., Nóbrega C., et al. Catatonia in anti-N-Methyl-D-Aspartate (NMDA) receptor encephalitis misdiagnosed as schizophrenia // Acta Med Port. 2020. Vol. 33, N 3. P. 208–211. doi: 10.20344/amp.11077
12. Graus F., Titulaer M.J., Balu R., et al. A clinical approach to diagnosis of autoimmune encephalitis // Lancet Neurol. 2016. Vol. 15, N 4. P. 391–404. doi: 10.1016/S1474-4422(15)00401-9
13. Barry H., Hardiman O., Healy D.G., et al. Anti-NMDA receptor encephalitis: An important differential diagnosis in psychosis // Br J Psychiatry. 2011. Vol. 199, N 6. P. 508–509. doi: 10.1192/bjp.bp.111.092197
14. Endres D., Leypoldt F., Bechter K., et al. Autoimmune encephalitis as a differential diagnosis of schizophreniform psychosis: Clinical symptomatology, pathophysiology, diagnostic approach, and therapeutic considerations // Eur Arch Psychiatry Clin Neurosci. 2020. Vol. 270, N 7. P. 803–818. doi: 10.1007/s00406-020-01113-2
15. Funayama M., Koreki A., Takata T., et al. Differentiating autoimmune encephalitis from schizophrenia spectrum disorders among patients with first-episode psychosis // J Psychiatr Res. 2022. Vol. 151. P. 419–426. doi: 10.1016/j.jpsychires.2022.05.008
16. Шмуклер А.Б. Позволяют ли новые знания улучшить диагностику психических расстройств: проблема анти-NMDA-рецепторного энцефалита // Неврологический вестник. 2024. Т. 56, № 1. С. 95–104. EDN: RWSPHE doi: 10.17816/nb625638
17. Мурашко А.А. Психические нарушения при аутоиммунных энцефалитах // Социальная и клиническая психиатрия. 2021. Т. 31, № 1. С. 74–79. EDN: ENASCT
18. Васенина Е.Е., Левин О.С., Ганькина О.А., и др. Аутоиммунный энцефалит с антителами к NMDA-рецепторам // Журнал неврологии и психиатрии им. С.С. Корсакова. 2017. Т. 117, № 2. С. 110–116. EDN: YHEQKX doi: 10.17116/jnevro201711721110-116
19. Малин Д.И., Гладышев В.Н., Равилов Р.С. Клинико-психопатологические проявления при аутоиммунном NMDA-рецепторном энцефалите // Социальная и клиническая психиатрия. 2020. Т. 30, № 2. С. 71–79. EDN: VKRDJK
20. Kayser M.S., Dalmau J. Anti-NMDA receptor encephalitis in psychiatry // Curr Psychiatry Rev. 2011. Vol. 7, N 3. P. 189–193. doi: 10.2174/157340011797183184
21. Мурашко А.А. Мимикрия: анти-NMDA-рецепторный энцефалит и психические расстройства // Социальная и клиническая психиатрия. 2022. № 4. С. 59–67. EDN: LJESPJ
22. Hu C.C., Pan X.L., Zhang M.X., Chen H.F. Paroxysmal speech disorder as the initial symptom in a young adult with anti-N-methyl-D-aspartate receptor encephalitis: A case report // World J Clin Cases. 2022. Vol. 10, N 24. P. 8648–8655. doi: 10.12998/wjcc.v10.i24.8648
23. Hettiarachchi Y.K.K., Wanigasinghe J. Speech and language abnormalities and their outcomes in autoimmune encephalitis in children: A case series // Sri Lanka Journal of Child Health. 2023. Vol. 52, N 1. P. 114–117. doi: 10.4038/sljch.v52i1.10487
24. Юдин Т.И. Смертельные формы шизофрении // Советская психоневрология. 1939. № 4–5. С. 2–23.
25. Тиганов А.С. Фебрильная шизофрения. Клиника, патогенез, лечение. Москва: Медицина, 1982. 128 с.
26. Цыганков Б.Д. Клинико-патогенетические закономерности развития фебрильных приступов шизофрении и система их терапии. Москва: РОП, 1997, 232 с.
27. Дунаевский В.В., Кузнецов А.В. Кататония — эволюция взглядов и современные представления (обзор литературы) // Обозрение психиатрии и медицинской психологии. 2019. Т. 4, № 2. С. 29–40. EDN: MGCSDQ doi: 10.31363/2313-7053-2019-4-2-29-40
28. Менделевич В.Д. Терминологические основы феноменологической диагностики в психиатрии. Москва: Городец, 2016. 128 с.
29. Менделевич В.Д. Гипердиагностика шизофрении как когнитивное искажение процесса познания клинической реальности // Неврологический вестник. 2023. Т. 55, № 1. С. 5–14. EDN: ZWAXRT doi: 10.17816/nb160308
30. Kelleher E., McNamara P., Dunne J. Prevalence of N-Methyl-d-Aspartate Receptor antibody (NMDAR-Ab) encephalitis in patients with first episode psychosis and treatment resistant schizophrenia on clozapine, a population based study // Schizophr Res. 2020. Vol. 222. P. 455–461. doi: 10.1016/j.schres.2019.11.023

AUTHORS' INFO

*** Vladimir D. Mendelevich,** MD, Dr. Sci. (Med.), Prof.,
Head of Depart.;
address: 49 Butlerova street, 420012 Kazan, Russia;
ORCID: 0000-0002-8476-6083;
eLibrary SPIN: 2302-2590;
e-mail: mendelevich_vl@mail.ru

ОБ АВТОРАХ

*** Владимир Давыдович Менделевич,** д-р мед. наук, проф.,
зав. каф.;
адрес: Россия, 420012, Казань, Бутлерова, д. 49;
ORCID: 0000-0002-8476-6083;
eLibrary SPIN: 2302-2590;
e-mail: mendelevich_vl@mail.ru

Konstantin S. Sergienko, resident of Psychiatry department;
ORCID: 0000-0002-2942-6174;
e-mail: kostya_s99@mail.ru

Kausar K. Yakhin, MD, Dr. Sci. (Med.), Prof., Psychiatry Depart.;
ORCID: 0000-0001-5958-5355;
eLibrary SPIN: 6275-6051;
e-mail: yakhin@bk.ru

Elvira A. Abdullina, MD, Head of Depart.;
ORCID: 0009-0000-3639-1806;
e-mail: abdullinaelvi@yandex.ru

Константин Станиславович Сергиенко, ординатор, каф. психиатрии и медицинской психологии;
ORCID: 0000-0002-2942-6174;
e-mail: kostya_s99@mail.ru

Каусар Камилович Яхин, д-р мед. наук, проф., каф. психиатрии и медицинской психологии;
ORCID: 0000-0001-5958-5355;
eLibrary SPIN: 6275-6051;
e-mail: yakhin@bk.ru

Эльвира Анваровна Абдуллина, зав. отд.;
ORCID: 0009-0000-3639-1806;
e-mail: abdullinaelvi@yandex.ru

* Corresponding author / Автор, ответственный за переписку