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Case Report

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Autoimmune Encephalitis. An Analysis of Three Cases

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ABSTRACT The diagnosis of diseases from the group of autoimmune encephalitis (AE) requires a detailed analysis of clinical data and correlation of results from a wide range of laboratory and instrumental research methods. This article presents three clinical cases of patients with AE. In one patient, AE was associated with a malignant neoplasm, in the second one – with the novel coronavirus infection, and in the third patient, no comorbid pathology was detected. The specific type of encephalitis was determined for each of the three patients. The diagnosis of "autoimmune limbic encephalitis" was established in two patients based on visualization of changes in the medial temporal structures in the magnetic resonance imaging of the brain, detection of pathological bioelectrical activity in the temporal lobe cortex in electroencephalography, and pleocytosis in cerebrospinal fluid. The diagnosis of "anti-NMDA receptor encephalitis" was made based on the detection of specific antibodies to the subunit of the corresponding receptor. Neurological deficit regression was observed in two patients after the application of combined immune therapy methods and in one patient without the use of immune therapy.

Keywords: autoimmune, anti-NMDA receptor, limbic encephalitis, autoantibodies, magnetic resonance imaging, electroencephalography

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anti-NMDARE — anti-NMDA receptor encephalitis
AE — autoimmune encephalitis
MV — mechanical ventilation
CE — contrast enhancement
LE — limbic encephalitis
MRI — magnetic resonance imaging
CSF — cerebrospinal fluid
GCS — Glasgow Coma Scale
EEG — electroencephalography
NMDA — N-methyl-D-aspartate (NMDA) receptor

Autoimmune encephalitis (AE) is a group of diseases of different etiology, which pathogenesis is based on autoimmune inflammation of the structures of the central nervous system. This category includes autoimmune limbic encephalitis (LE), anti-NMDA receptor encephalitis (anti-NMDARE), acute disseminated encephalomyelitis, Bickerstaff encephalitis and Hashimoto encephalopathy [1]. Etiological factors for the development of AE can be infections, endocrine and oncological diseases. Autoimmune encephalitis associated with malignant neoplasms is a form of paraneoplastic syndrome. These include LE and anti-NMDARE [2].

Three symptom complexes with acute or subacute development are common to AE: mnestic and intellectual disorders, mental disorders, as well as changes in the level of wakefulness. This set of signs forms the basis of the diagnostic criteria for AE [1].

Diagnosis of AE can be difficult for a number of reasons. Diseases with similar clinical symptoms constitute a wide differential range, including stroke, toxic and metabolic encephalopathies, infectious encephalitis, psychiatric diseases, neurodegenerative diseases, neoplasms and epilepsy [2].

Objectification of AE can be a difficult task. Thus, characteristic radiological manifestations on magnetic resonance imaging (MRI) of the brain at the onset of the disease are observed only in 31–33% of patients with anti-NMDARE and in 57–69% of patients with LE, and specific autoantibodies may not be detected in the early stages of the disease. In addition, the availability of laboratory test systems for the detection of antineuronal antibodies is not widespread [1–5].

Malignant neoplasms are found in 38% of patients with anti-NMDARE and in 60–70% of patients with LE [6, 7]. The manifestation of AE can precede the detection of cancer by months and sometimes years. Thus, in 60% of patients with LE, a malignant neoplasm was first detected within 3.5 months [5].

Treatment for AE includes the following components: immunotherapy, symptomatic therapy of AE manifestations (motor and psychiatric disorders, seizures, etc.), prevention and treatment of complications and rehabilitation [8–10].

The aim of this publication is to analyze and discuss clinical, laboratory and instrumental data in patients with AE hospitalized at the N.V. Sklifosovsky Research Institute for Emergency Medicine (the Institute).

Clinical case 1

A 40-year-old patient was transferred to the Institute from a hospital, where he was staying due to subacutely developed and progressive cognitive and motor impairments, and a decrease in the level of wakefulness. From the anamnesis it was known that one month before admission to the Institute, the patient was diagnosed with coronavirus infection COVID-19, complicated by pneumonia, and therefore the patient was hospitalized in the infectious diseases department. On the second day of the disease, the patient told the doctors about a feeling of "déjà vu," that he could "anticipate" the actions of the medical personnel, and in addition, he saw "pictures in his head". There were no signs of respiratory failure. On the 11th day of the disease, a decrease in the level of wakefulness to coma was noted, and mechanical ventilation (MV) was started. Electroencephalography (EEG) revealed a focus of pathological slow-wave activity in the temporal region on the right. MRI of the

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brain with contrast enhancement (CE) showed no pathological changes. Clinical analysis, molecular genetic, microbiological examination of cerebrospinal fluid (CSF) did not reveal any abnormalities. After excluding stroke, neoplasms and infections of the central nervous system, status epilepticus and metabolic disorders, the diagnosis of Autoimmune Encephalitis was made. Analysis for antibodies to receptors and synaptic proteins (Hu, Yo-1, CV2, PNMa2, Ri, AMPH, NMDA-S, CASPR-S, LGI-S, AMPA1-S, AMPA-2, GABAR-S) was taken into account (later - negative). Anti-inflammatory therapy with 24 mg of dexamethasone per day was carried out for 3 days without significant effect. Afterwards, 5 sessions of plasmapheresis were performed, and then a 3-day course of immunoglobulin therapy at a dose of 0.4 g/kg/day. Positive dynamics were noted in the form of restoration of clear consciousness, the patient was weaned from mechanical ventilation. The course of the disease was complicated by nosocomial pneumonia and deep vein thrombosis. The patient was transferred to the Institute. Neurological status upon admission to the Institute: clear consciousness, Glasgow Coma Scale (GCS) score – 15. The patient was oriented to place, time and self. A decrease in short-term memory was detected, MMSE (Mini-Mental State Examination) score of 28. Complaints of difficulty to fall asleep. Tetraparesis: 4 points in the upper, 3 points in the lower extremities. Muscle tone was increased and has a plastic quality in all extremities. Bradykinesia, hypokinesia. Focal upper extremity dystonia. At night, psychomotor agitation was noted, and the patient described visual and auditory hallucinations.

Given the positive effect of previously administered immune therapy, further management included symptomatic therapy and treatment for complications. In order to correct extrapyramidal disorders, 500 mg of levodopa per day in 3 doses was prescribed; in connection with episodes of psychomotor agitation - 45 mg of chlorprothixene per day. Etiotropic antibacterial therapy and treatment of deep vein thrombosis, as well as individual physical therapy sessions, were continued.

Subsequently, on the 56th day of the disease, restoration of muscle strength of the limbs, regression of rigidity and dystonia were noted. Repeated MRI of the brain revealed an increase in the MR signal in the T2 FLAIR (Fluid Attenuated Inversion Recovery) mode from the mesotemporal parts of the right and left hemispheres of the brain (amygdala, hippocampus), and partially from the cortex of the right insula (Fig. 1). A diagnosis of Autoimmune Limbic Encephalitis was made. The patient was discharged with good clinical and functional outcomes (Modified Rankin score 0, Rivermead Mobility Index 14 points).

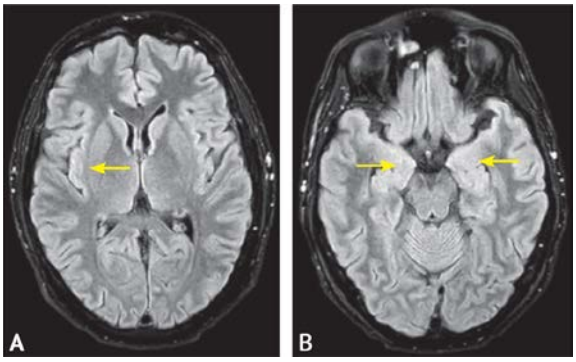

Fig. 1. Magnetic resonance imaging of the brain in T2 FLAIR mode of the patient from clinical case 1. Increased MR signal from the insular cortex of the right hemisphere (A) and the structures of the mesial temporal parts of the brain on the right and left (B) (indicated by arrows)

Clinical case 2

A 60-year-old female patient was taken to the emergency department of the Institute due to amnesia of the events of the day of hospitalization. From the life history it was known about an oncological disease – the patient suffered from small cell cancer of the upper lobe of the left lung, IVA stage cT2bN2M1a (pe, pu), undergoing immune therapy with the antitumor drug atezolizumab. The day before admission was the last administration of this drug.

On admission: clear consciousness, GCS score 15. Neither motor nor sensory disorders were detected. Decreased short-term memory, amnesia for the events that accompanied hospitalization. The patient was disoriented in time and place. MMSE score 22. Criticism of one's own condition was reduced.

MRI of the brain with CE, performed on the 6th day of the disease, revealed an increase in the MR signal in T2-weighted and T2 FLAIR images from the mesotemporal parts of the right and left hemispheres of the brain (amygdala, hippocampus). Intravenous CE revealed active accumulation of the contrast agent in these structures, mainly on the left (Fig. 2).

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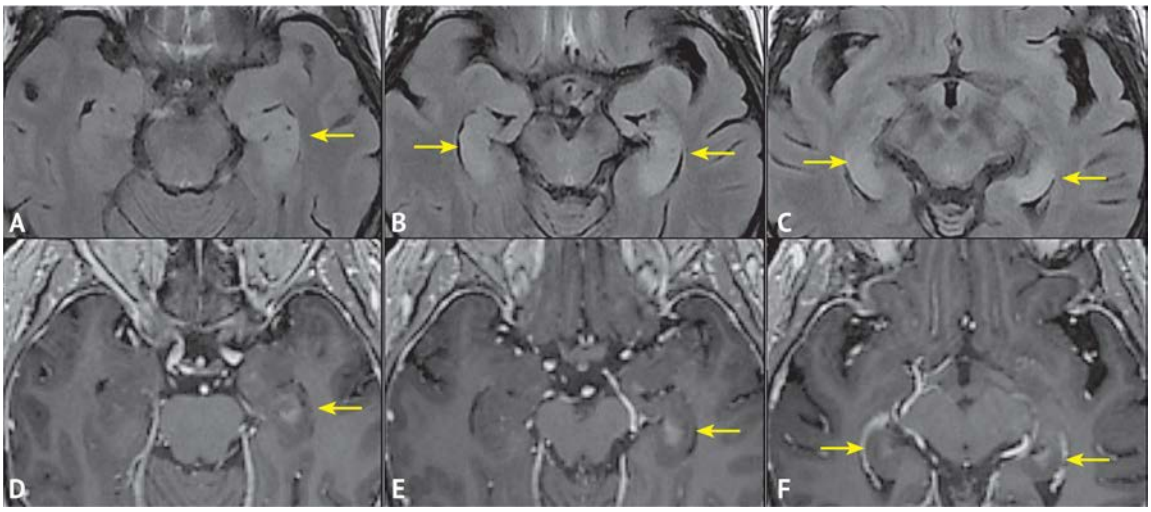

Fig. 2. Axial magnetic resonance imaging of the brain of the patient from clinical case 2. Increased signal from mesial temporal structures in T2 FLAIR mode (A, B, C) and accumulation of the contrast agent in these structures on T1 weighted images (D, E, F) (indicated arrows)

When evaluating responses in the EEG to intermittent photic stimulation with a frequency of 1 Hz, the patient reported "internal restlessness with a vague feeling of discomfort" followed by disorientation in space and time, and then by behavioral cessation. At this moment, the electrographic picture was represented by a pattern of a focal epileptic seizure with the beginning in the posterior parts of the left frontal lobe and spreading to the temporo-parietal parts of the left hemisphere. During the entire attack, unchanged background activity was recorded over the right hemisphere, which indicated its strict focality, despite the generalized semiology (Fig. 3). Analysis of the CSF revealed an increase in protein levels to 1 g/l and a cytosis of 10 cells in 1 μl. No antineuronal autoantibodies were detected. Polymerase chain reaction based analysis did not reveal the genetic material of herpes viruses types 1, 2 and 6, Epstein-Barr virus and cytomegalovirus; and microbiological examination showed no growth of microorganisms. Based on the clinical picture, MRI, EEG and CSF pleocytosis data, the patient with small cell lung cancer was diagnosed with Autoimmune Limbic Encephalitis. Antiepileptic therapy with valproic acid was prescribed at a dose of 1500 mg per day. Pulse therapy with methylprednisolone was performed at a dose of 1000 mg per day for 5 days. No significant clinical effect was noted. 5 plasmapheresis procedures were performed with a plasma replacement volume of up to 1500 ml per session, with a moderate positive effect in the form of an increase in the volume of auditory-verbal memory (MMSE score 25).

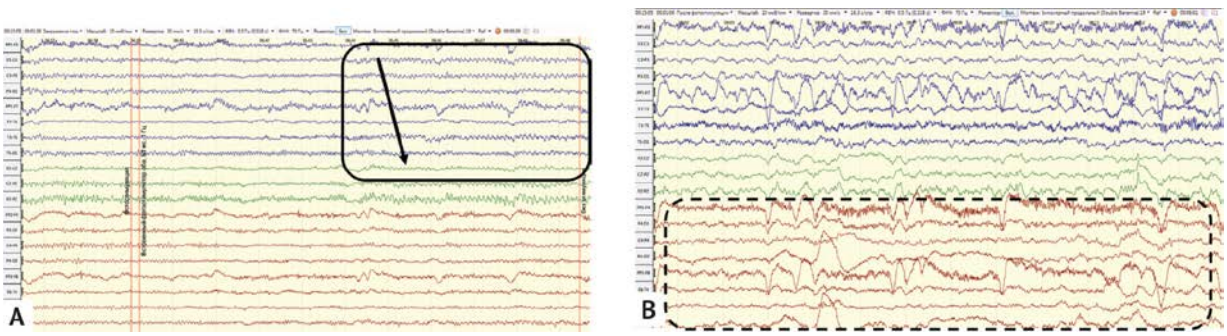

Fig. 3. Electroencephalogram of the patient from clinical case 2. Longitudinal bipolar montage (double banana). The electrographic onset of the attack from the posterior frontal parts of the left hemisphere (circled with a solid frame) with spread to the temporoparietal region (indicated by the arrow), which caused aphasia and behavioral cessation (A). Recording segment 90 seconds after the onset of the attack. The dotted frame outlines leads from the right hemisphere, along which normal background activity is maintained (B)

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On the 31st day of the disease, complete regression of symptoms was noted. The patient was discharged for further cancer treatment.

Clinical case 3

A 50-year-old female patient was admitted to the Center for the Treatment of Acute Poisonings of the Institute with suspected poisoning by an unknown substance. She was found not making contact by relatives at home. Upon admission, the level of wakefulness was depressed to the point of surface stupor (GCS score 14), the patient is disoriented in place and time, her thinking is inconsistent, she hears "voices in her head", "sees spirits". Laboratory studies revealed leukocytosis 15.26 x 109/L, increased AST (aspartate aminotransferase) of 343 U/L, and ALT (alanine aminotransferase) of 82 U/L, hypoproteinemia up to 48 g/L, and hyponatremia 129 mmol/L. A chemical toxicity testing did not reveal any toxic agents. Non-contrast MRI of the brain did not visualize pathological changes. On the 1st day of hospitalization, restoration of the level of wakefulness to clear consciousness was observed. On the 4th day of the disease, mnestic-intellectual disorders and productive symptoms regressed completely.

Repeat MRI of the brain, supplemented with T1-weighted contrast-enhanced images, revealed evidence of bilateral hippocampal contrast uptake consistent with LE. Electroencephalography did not reveal pathological bioelectrical activity (Fig. 4).

Clinical analysis of CSF – without abnormalities. Molecular genetic and microbiological studies of the CSF did not detect pathogens of neuroinfections, however, immunological analysis showed the presence of antibodies to the NR2 peptide of NMDA receptors — 14.2 ng/ml (0.0–1.7). A diagnosis of Anti-NMDA Receptor Encephalitis was made. Due to the absence of symptoms of the disease at the time of verification of the diagnosis, a decision was made to refrain from immunotherapy. An oncological search was performed including computed tomography of the chest organs, ultrasound examination of the abdomen, kidneys and pelvis, and sciontigraphy of skeletal bones: no neoplasms were identified. The patient was discharged in satisfactory condition. Over the next 90 days, the patient had no reason to seek medical help.

DISCUSSION

Autoimmune limbic and anti-NMDA receptor encephalitis are two diseases with similar risk factors, clinical and radiological manifestations. Some researchers call encephalitis with antibodies to the subunits of the N-methyl-D- aspartate receptor a type of LE, whereas in the conceptual work by Graus F. et al. (2016), summarizing diagnostic approaches to AE, LE and anti-NMDARE are identified as independent nosologies. This scientific article formulates diagnostic criteria for LE, anti-NMDARE, and AE as a general category [1, 7].

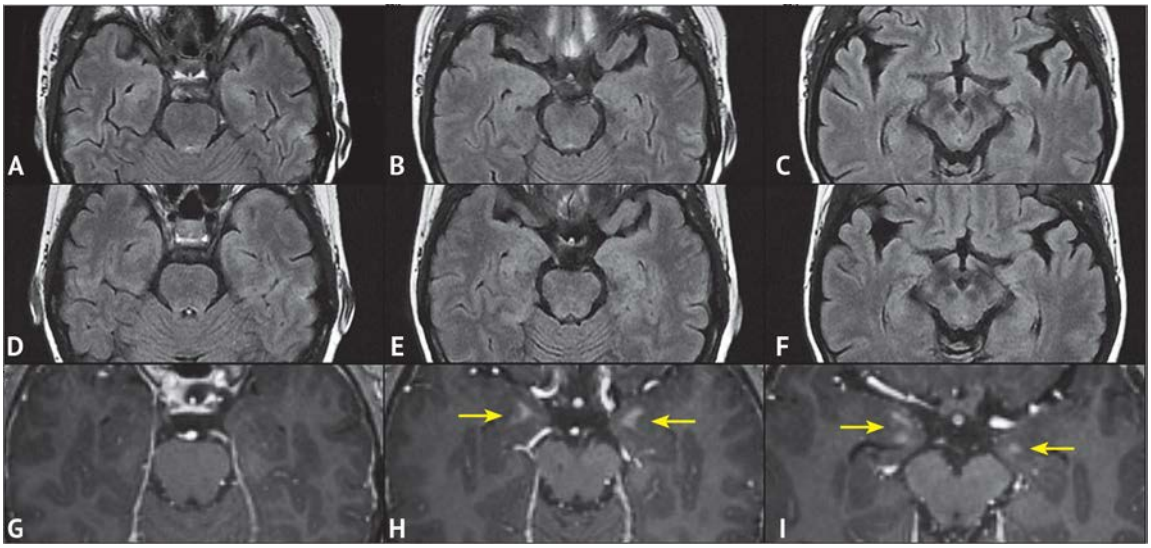

Fig. 4. Axial magnetic resonance imaging of the patient's brain from clinical case 3. Tomograms at the level of the mesial temporal parts of the cerebral hemispheres in the T2 FLAIR mode on the first day of the disease do not reveal changes in the signal from these structures (A, B, C). The study performed on the 6th day of the disease does not reveal changes in the signal in the T2 FLAIR mode (D, E, F) either; however, T1-weighted tomograms after contrast enhancement reveal the accumulation of the contrast agent in the hippocampus (indicated by arrows)

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In each of our clinical observations, the disease debuted with an acutely developed disorder of higher nervous activity: impaired memory and gnosis, thinking, productive symptoms, and depression of the level of wakefulness. Detection of symptoms of this group is the first obligate condition for establishing the diagnosis of Autoimmune Encephalitis. Also, in each of the 3 patients, clinical or instrumental signs were found that coincided with other conditions of the diagnostic criteria for AE: focal deficit in the form of dystonia (patient 1), epileptic seizure (patient 2), and characteristic changes according to MRI (all the patients).

It was possible to clarify the nosology within the AE group in each of the 3 patients. The diagnosis of Autoimmune Limbic Encephalitis was established for two patients in connection with the visualization of changes in the MR signal from the medial temporal structures on T2 FLAIR images (patients 1 and 2), EEG detection of epileptiform activity (patient 2), and slow wave activity (patient 1) of the temporal lobe cortex in addition to the above signs, pleocytosis in the CSF (patient 2). The diagnosis of Anti-NMDA Receptor Encephalitis was established based on the detection of specific antibodies to the subunit of the NMDA receptor.

Magnetic resonance imaging revealed characteristic changes in mesotemporal structures in each of the 3 cases. However, it is necessary to note that the pathological changes in the patient from clinical observation 1 were visualized with non-contrast MRI over time, whereas at the onset of the disease, this method did not detect any deviations from the norm. In clinical case 3, radiological signs of AE were revealed only during MRI with CE and were not visualized during the initial and repeated non-contrast studies.

Antineuronal antibodies were detected in only one patient (clinical case 3) - antibodies to the NR2 peptide of NMDA receptors. This example leads to the conclusion about the lower diagnostic significance of serological tests, and the dominant role of clinical, radiological and electrophysiological data in patients with AE in the early stages of the disease. In addition, false-positive test results for antineuronal antibodies in the CSF are known in patients with primary tumors, neurodegenerative, and other brain diseases [11, 12]. An emphasis on the clinical and instrumental picture was also placed in the current diagnostic criteria for AE, which distinguishes them from previously existing ones based on serological diagnosis [1, 13].

An additional and important component of the differential diagnostic search is information about comorbid pathology and past diseases. The association of coronavirus infection COVID-19 and AE, including LE and anti-NMDARE, was described in scientific papers, and the connection of LE with malignant neoplasms has been known for more than half a century [14, 15].

There is an opinion that the effectiveness of immune therapy may have diagnostic value. This view found application in diagnostic criteria for the so-called “conditions associated with neuronal surface antibodies,” a broad group of diseases that includes AE. In our first clinical example, this hypothesis supported the diagnosis of Autoimmune Encephalitis until the diagnosis was verified using brain MRI [13].

Currently, the therapeutic approach for LE and anti-NMDARE remains undifferentiated, both in terms of immune therapy and symptomatic treatment. In our observations, a positive effect was achieved in the first clinical case (LE) only after the use of all first-line immune therapy (glucocorticosteroids, plasmapheresis and immunoglobulin); whereas the spontaneous complete regression of the symptoms by the time the diagnosis was verified in the patient with anti-NMDARE in the third clinical example led us to the decision not to use immune therapy due to the prevalence of known risks over uncertain benefits.

CONCLUSION

Diagnosis of autoimmune limbic and anti-NMDA receptor encephalitis requires a detailed analysis of clinical data and comparison with a wide range of laboratory and instrumental research methods. Success in the treatment for autoimmune encephalitis may lie in the use of all first-line immune therapy, as well as in observational tactics and symptomatic therapy.

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