

*Prikaz slučaja /
Case report*

PSYCHOTIC PRESENTATION AS A FIRST
CLINICAL MANIFESTATION OF
ANTI-N-METHYL-D-ASPARTATE
RECEPTOR (NMDAR) ENCEPHALITIS –
Case report

PSIHOTIČNA PREZENTACIJA KAO PRVA
KLINIČKA MANIFESTACIJA
ANTI-N-METIL-D-ASPARTAT RECEPTOR
(NMDAR) ENCEFALITISA – *Prikaz slučaja*

Correspondence to:

Teodora Inić
Univerzitetski klinički centar Vojvodine
Klinika za psihijatriju
Hajduk Veljkova 1,
21000 Novi Sad
teodora.inic@gmail.com

Teodora Inić¹, Nemanja Stanković Stevanović¹,
Boško Čturić¹, Mina Cvjetković Bošnjak^{1,2},
Vanja Meši Bosić^{1,2}

¹ Klinika za psihijatriju, Univerzitetski klinički centar Vojvodine, Novi Sad, Srbija

² Medicinski fakultet Univerziteta u Novom Sadu, Novi Sad, Srbija

Key words

Autoimmune encephalitis; anti-NMDA receptor antibodies; psychosis; neuropsychiatric symptoms

Ključne reči

Autoimuni encefalitis; anti-NMDAR antitela; psihoza; neuropsihijatrijski simptomi.

Abstract

Autoimmune encephalitis (AE) is a group of inflammatory disorders of the central nervous system in which immune-mediated inflammation, caused by specific autoantibodies, leads to damage of various brain structures. The most common and best-studied form is AE associated with NMDA receptor antibodies. It is well recognized that psychiatric symptoms often represent the initial manifestation of anti-NMDAR encephalitis. In this context, it is essential to consider whether the presentation represents a first psychotic episode in affected patients, initiate treatment according to the current clinical picture, and continue close monitoring. If psychotic symptoms persist despite appropriate psychopharmacological treatment, especially with the emergence of accompanying neurological deficits such as cognitive impairment, new-onset seizures, and autonomic dysfunction, the possibility of AE must be considered.

Particular attention should be paid to younger female patients, especially those with a history of gynecological comorbidities, primarily ovarian teratoma. To establish an accurate diagnosis, initiate timely therapy, and avoid numerous complications, diagnostic procedures should be started as early as possible. **The aim** of this report is to discuss the potential diagnosis of AE, particularly NMDA receptor encephalitis, in patients presenting with new-onset psychotic symptoms. **Case presentation:** To familiarize clinicians with immune-mediated neurological disorders that may predominantly manifest with psychiatric symptoms and facilitate further management, we present a case of NMDA receptor encephalitis viewed through the lens of its psychotic manifestations. **Conclusion:** Early recognition of autoimmune etiology in psychiatric disorders is crucial for prompt diagnosis and timely initiation of appropriate treatment, which is essential for a favorable disease course and outcome.

INTRODUCTION

Autoimmune encephalitis (AE) is an inflammatory disease of the brain caused by an immune response directed against self-antigens expressed in the central nervous system. A key pathogenetic role in the development of AE is played by specific autoantibodies directed against various neuronal surface or intracellular antigens. In addition to the

pathogenetic significance, specific autoantibodies play an important role in determining the clinical presentation of AE and the therapeutic response, indicating their association with malignancies, as well as the course and outcome of the disease. AE are characterized by a heterogeneous clinical presentation with predominant cognitive and psychiatric symptomatology, associated with new-onset epileptic

seizures, involuntary movements and dysautonomia^(1,2). In some cases, especially if the disease is not recognized in time may lead to various complications, including lethal outcomes⁽³⁾.

Psychosis is defined by both DSM-5-TR and ICD-11 as a condition marked by impaired reality testing, most commonly presenting through persistent delusions, hallucinations, and disorganized thinking or behavior. While these symptoms are central to primary psychiatric disorders such as schizophrenia and brief psychotic disorder, they are not pathognomonic and can also occur in a range of neurological and systemic conditions, including AE⁽³⁾. Among the most studied forms of AE is anti-NMDA receptor encephalitis, a disorder in which autoantibodies target NMDA-type glutamate receptors in the brain, leading to widespread synaptic dysfunction. NMDA receptors are integral to processes of cognition, memory, and emotional regulation, and their disruption produces a clinical picture that often overlaps with functional psychiatric disorders⁽⁴⁾. In the early stages of anti-NMDAR encephalitis, patients—particularly young women—may present with acute psychotic symptoms in the absence of overt neurological signs, leading to misdiagnosis and delays in appropriate treatment. Although the presence of seizures, dyskinesias, autonomic instability, or fluctuating consciousness may later suggest an organic etiology, these signs are frequently absent at initial evaluation. Ancillary investigations such as cerebrospinal fluid analysis, EEG, and MRI can support the diagnosis but are not always immediately conclusive^(5,6). Therefore, a high index of suspicion is essential, especially in first-episode psychosis.

This case report presents a young woman with anti-NMDAR encephalitis initially misdiagnosed as manic psychosis. The aim of this paper is to emphasize the clinical and diagnostic overlap between autoimmune and primary psychiatric disorders, to explain the role of NMDA receptor dysfunction in the pathophysiology of AE, and to highlight the importance of early recognition and multidisciplinary assessment in achieving a timely and accurate diagnosis.

CASE REPORT

A 28-year-old woman, employed, single, and without children, was admitted to the psychiatric clinic due to acute behavioral disturbances and rapidly evolving psychiatric symptoms. Her medical history included endometriosis and rheumatoid arthritis. She had no prior psychiatric treatment or hospitalizations. Several months before the admission, she underwent a single psychiatric consultation for persistent anxiety, emotional instability, and sleep disturbances. She was diagnosed with a mixed anxiety and depressive disorder and was prescribed escitalopram and a sedative. Approximately five days prior to hospitalization, the patient developed abrupt behavioral changes: incoherent speech, psychomotor agitation, disinhibited actions (such as disrobing and throwing objects), decreased need for sleep, and racing, grandiose ideation. She expressed an intense need for activity and reported feeling misunderstood and mistrusted by those around her. These symptoms were interpreted as a first manic episode, possibly iatrogenic, and antipsychotic medication was initiated. However, her condition continued to deteriorate in the days following hospitalization. The ini-

tially polymorphic psychotic presentation evolved further, with the emergence of confusion, disorientation, and intuitive delusional ideas—specifically, the belief that someone or something was coming to get her—accompanied by intense fear for her life and a significant impact on her behavior. She persistently repeated that she had to leave because „something was coming for her.”

Due to the severity of her agitation, parenteral pharmacological treatment was initiated, including diazepam and haloperidol. Episodic muscle spasms were initially interpreted as extrapyramidal symptoms, particularly acute dystonia, possibly related to antipsychotic therapy. Consequently, biperiden was administered, but it led to minimal clinical improvement.

Soon thereafter, the patient's condition further worsened. Cognitive disorganization became apparent, along with the emergence of speech disturbances. Her spontaneous speech became progressively reduced, disorganized, and at times incoherent, with intermittent perseveration and paraphasic errors. She exhibited echolalia, occasionally repeating words or phrases spoken by others without context. Periods of dysarthria, characterized by slurred and effortful speech, were observed intermittently, along with transient episodes of mutism, during which the patient was unresponsive despite appearing alert. This was followed by the development of orofacial (twitching, grimacing, and repetitive movements of the mouth and tongue) and limb dyskinesias, muscular rigidity, and unexplainable headaches, ultimately progressing to fluctuating levels of consciousness. The patient also exhibited intermittent generalized muscle spasms, which culminated in episodes of opisthotonus.

The limited response to symptomatic therapy, along with the presence of fluctuating consciousness, stereotypic movements, and resistance to standard psychiatric management, raised clinical suspicion of an underlying organic etiology—prompting further evaluation.

Initial diagnostic workup, including standard laboratory serum analyses (complete blood count, renal and liver function tests, thyroid function tests, and inflammatory markers) were performed, chest X-ray, abdominal ultrasound, and brain CT – all showed no pathological findings. Although episodes of intermittent fever were observed after few days following the admission, serum inflammatory markers remained normal. Due to symptom progression and lack of treatment response, consultations with an infectious disease specialist and a neurologist were conducted to rule out infectious, metabolic, or immune-mediated encephalopathy. Following the neurologist's recommendations, serological and cerebrospinal fluid (CSF) analyses were performed to investigate possible autoimmune encephalitis, along with laboratory testing for systemic autoimmune diseases, electroencephalography (EEG), and cerebral MRI. Based on the infectious disease specialist's indications, targeted microbiological investigations were also carried out. Antibody screening was conducted to evaluate potential autoimmune etiologies. The panel included antibodies against neuronal cell surface antigens (NMDA-R, CASPR2, LGI1, AMPA-R, GABAB-R), intracellular neuronal antigens (Hu, Ri, Yo, CV2/CRMP5, Ma2 [Ta], amphiphysin), as well as thyroid-specific antigens (thyroglobulin [TG], thyroid peroxidase

[TPO]) and antinuclear antibodies (ANA). The results revealed positive anti-NMDA receptor antibodies and ANA in serum. Screening for neurotropic viruses such as Cytomegalovirus (CMV), Epstein-Barr virus (EBV), Herpes simplex virus (HSV), varicella-zoster virus (VZV), influenza virus as well as other pathogens including *Borrelia burgdorferi* and *Toxoplasma gondii* was conducted too. All microbiological tests returned negative, ruling out the infectious etiology. Electroencephalography (EEG) showed dominant beta activity, a nonspecific but frequently observed pattern in anti-NMDAR encephalitis. Brain MRI revealed bilateral pachymeningeal enhancement with suspicion of accompanying leptomeningitis, but no parenchymal lesions. Cerebrospinal fluid (CSF) lymphocytic pleocytosis, normal glucose levels, and mildly elevated protein were found.

Given the progression of symptoms—marked by persistent neuropsychiatric disturbances, agitation refractory to treatment, and episodic muscle spasms unresponsive to biperiden—the patient was transferred to a specialized neurology department. There, further diagnostic workup and comprehensive management were conducted. Subsequently, the patient developed epileptic seizures, which were successfully controlled with the introduction of antiepileptic therapy. Given the well-established association between anti-NMDAR encephalitis and ovarian teratomas, particularly in young female patients, a comprehensive oncological evaluation was undertaken. Tumor markers and whole-body contrast-enhanced CT showed no evidence of malignancy. However, pelvic MRI revealed a right-sided endometriotic ovarian cyst. Although no teratoma was radiologically identified, the lesion was surgically removed due to high clinical suspicion. Histopathological analysis confirmed a benign endometriotic cyst, with no neoplastic or teratomatous elements detected.

Given the significant clinical improvement following its initiation, the focus of treatment in this case will be discussed primarily in the context of immunotherapy. Immunotherapy included high-dose intravenous corticosteroids (pulse therapy), therapeutic plasma exchange, and intravenous immunoglobulins (IVIG). Psychiatric consultations were performed repeatedly, leading to the use of diazepam to manage agitation and the cautious reintroduction of antipsychotic therapy.

Following nearly three months of inpatient care, the patient achieved significant clinical improvement and was discharged in a satisfactory remission.

This case emphasizes the importance of identifying „warning signs” that may point toward an underlying organic etiology—particularly autoimmune encephalitis—in patients presenting with acute psychiatric symptoms. While the complete neurological diagnostic, complications and therapeutic procedures were essential for the final outcome, they are not described in detail here, as they fall beyond the scope and focus of this case report, which centers on early recognition and differential diagnostic considerations in the psychiatric setting.

DISCUSSION

As it is known the NMDA receptors have a significant role in many diseases, including Alzheimer’s disease, Huntington’s disease, epilepsy, stroke, major depressive disorder, heavy metal poisoning, tinnitus, schizophrenia, and autoimmune encephalitis⁽⁴⁾. AE, including anti-NMDAR encephalitis, often present initially with prominent psychiatric symptoms that closely mimic primary psychiatric disorders, posing a significant diagnostic challenge⁽⁵⁾. Psychotic features such as delusions, hallucinations, disorganized thinking, and mood disturbances are common in both conditions, yet their underlying mechanisms and clinical course differ fundamentally. In primary psychiatric illnesses like schizophrenia or bipolar disorder, these symptoms generally arise from intrinsic neurochemical and structural brain alterations without an identifiable external trigger. In contrast, anti-NMDAR encephalitis results from autoimmune-mediated disruption of synaptic NMDA receptor function, which can lead to rapid onset and progression of symptoms^(5,6).

A key distinguishing factor is the evolution and constellation of symptoms. In anti-NMDAR encephalitis, psychiatric manifestations are frequently accompanied or rapidly followed by neurological signs—such as seizures, movement disorders (e.g., dyskinesias), autonomic instability, and fluctuating levels of consciousness—that are rare in primary psychiatric conditions^(1,2). These neurological features are critical “red flags” that should raise suspicion for an organic cause behind psychosis, especially when psychopharmacological treatment yields limited or paradoxical responses⁽⁶⁾. In the presented case, the disease initially manifested with symptoms resembling a manic episode. However, early signs of an underlying organic etiology soon emerged, including disorganized speech, fluctuating levels of consciousness, and the development of movement disorders, later followed by seizures. According to both heteroanamnestic and autoanamnestic reports, the patient had no prior history of epileptic seizures. Seizures are common symptoms of anti-NMDA receptor encephalitis and occur in almost 80% of cases⁽⁷⁾. This type of encephalitis is a significant cause of otherwise unexplained new-onset epilepsy. Data from the literature revealed that more than 25% of female patients aged 18–45 with new-onset epilepsy, often accompanied by neuropsychiatric symptoms, tested positive for anti-NMDA receptor antibodies, with no other cause for the seizures identified⁽⁸⁾. Seizures usually occur in the early stages of the disease, but they can resurface during all stages of the NMDA encephalitis, either as seizures or status epilepticus. The course of the disease can show significant variability. It usually exhibits five phases: the prodromal phase, the psychotic phase, the unresponsive phase, the hyperkinetic phase, and the gradual recovery phase, but not all patients experience all of the stages mentioned⁽⁹⁾.

Diagnostic tools play a vital role in differentiation. Detection of anti-NMDAR antibodies in cerebrospinal fluid and serum is the diagnostic cornerstone, offering high specificity. CSF examination typically shows elevated CSF protein levels, anti-NMDAR antibodies, pleocytosis, and oligoclonal bands in 60% of cases, while routine blood laboratory tests generally remain normal^(10,11). Most

patients have intrathecal synthesis of NMDAR antibodies that's why laboratory confirmation of the diagnosis is made by detecting antibodies in the cerebrospinal fluid, which increases the specificity of a positive finding and reduces the possibility of obtaining false positive or negative results^(1,2). Detection of antibodies in the CSF increases specificity and reduces the likelihood of false-positive or false-negative results^(1,2). In the present case, both serum and CSF were tested for a panel of neuronal antibodies, however, only the serum was positive for anti-NMDAR antibodies. The analysis was conducted using indirect immunofluorescence, a method that is now widely implemented as a routine diagnostic tool in many laboratories worldwide. Such finding may occur for several reasons, including the presence of undetected autoantibodies, omission from the assay panel, or insufficient assay sensitivity. Additionally, the brain's strong immunoadsorbent capacity may sequester autoantibodies, limiting their detectability in serum and CSF^(5,6). According to the criteria proposed by Graus et al. (2016), a diagnosis of anti-NMDAR encephalitis can be made even in the absence of detectable antibodies if all four of the following are met: (1) subacute onset (<3 months) of psychiatric symptoms, altered mental status, or memory deficits;⁽²⁾ exclusion of well-defined autoimmune encephalitis syndromes; (3) absence of well-characterized antibodies in serum and CSF; and (4) MRI features suggestive of encephalitis, CSF pleocytosis, CSF-specific oligoclonal bands or elevated IgG index, and reasonable exclusion of alternative diagnoses^(5,6). The presented case fulfilled all four criteria.

Brain MRI is normal in approximately 50% of anti-NMDAR encephalitis cases; when abnormal, it most commonly reveals medial temporal lobe hyperintensity⁽⁵⁾. However, MRI findings can also reveal less typical features, such as pachymeningitis, which was observed in the present case and further supports an inflammatory etiology distinct from primary psychiatric disorders. Jia et al. (2019) described a 68 year old patient with anti-NMDAR encephalitis who also exhibited pachymeningeal enhancement on MRI⁽¹²⁾. In line with this observation, a case report from 2021 also documented a 17-year-old patient with confirmed anti-NMDAR encephalitis and imaging findings of pachymeningitis⁽¹³⁾.

In female patients, evaluation for an underlying ovarian teratoma is important, as it is a known paraneoplastic trigger of anti-NMDAR encephalitis. Imaging methods such as pelvic ultrasound, MRI, or CT scan are commonly used for this purpose⁽¹¹⁾. In this case, pelvic MRI revealed an ovarian lesion that was surgically removed. Histopathological examination confirmed it to be benign, however, resection was still indicated, since even benign teratomas can contribute to the development or persistence of the autoimmune process in anti-NMDAR encephalitis.

According to international expert consensus, the first-line treatment for autoimmune encephalitis, aimed at removing pathogenic autoantibodies, includes immunosuppressive therapy with corticosteroids, intravenous immunoglobulins (IVIg), or immunoadsorption/plasmapheresis. If no clinical improvement is observed, rituximab is recommended as a second-line agent. In refractory cases, further immunosup-

pression with cyclophosphamide, mycophenolate mofetil, or methotrexate may be required to achieve disease control⁽⁶⁾. The treatment is stopped when the recovery is achieved, usually marked by the decrease in concentration of CSF and serum antibodies⁽¹¹⁾.

Adjunctive psychopharmacological management is often necessary to stabilize psychiatric symptoms. Short-acting benzodiazepines are commonly used for agitation, and second-generation antipsychotics with a low risk of extrapyramidal side effects are preferred. Electroconvulsive therapy (ECT) may be considered as a last-resort option in severe, treatment-resistant psychiatric manifestations⁽⁶⁾.

In the case of the presented patient, treatment included high-dose corticosteroids, plasma exchange, and IVIg. Due to a favorable clinical response, second-line immunotherapy was not required. Additionally, based on clinical suspicion, a gynecological mass was surgically removed and subsequently confirmed by histopathology to be a benign endometriotic ovarian cyst. In cases of paraneoplastic anti-NMDAR encephalitis, timely tumor resection is considered essential, as it is directly linked to improved outcomes. While immunosuppressive therapy, plasma exchange, or IVIg may lead to partial neurological improvement or stabilization, thereby facilitating surgical intervention, it is important to note that delays in tumor resection may allow for tumor progression⁽¹⁴⁾. Therefore, clinicians should carefully weigh the risks and benefits of postponing surgery in suspected or confirmed paraneoplastic cases. The overall therapeutic strategy led to a marked improvement in symptoms and sustained recovery.

CONCLUSION

In this case, the patient initially presented with psychotic symptoms and was diagnosed with manic psychosis. Subsequent evaluation revealed autoimmune encephalitis as the underlying cause. This overlap is explained by NMDA receptor dysfunction due to anti-NMDA receptor antibodies, which, when cleared, lead to symptom improvement. This case highlights the importance of considering anti-NMDAR encephalitis in first-episode psychosis, especially when symptoms do not respond to psychotropic treatment. Early recognition of the autoimmune origin of psychiatric disorders is crucial for timely intervention and improved prognosis.

CONFLICT OF INTEREST

All authors declare that there was no conflict of interest.

Sažetak

Uvod: Autoimuni encefalitis (AE) spadaju u inflamatorne bolesti centralnog nervnog sistema kod kojih imunski posredovana inflamacija posredovana specifičnim autoantitelima dovodi do oštećenja različitih struktura mozga. Najčešći i do sada najbolje proučen je AE udružen sa NMDA antitelima. Poznato je da psihijatrijska simptomatologija često može biti inicijalna manifestacija anti-NMDAR encefalitisa. U tom smislu je neophodno razmotriti da li je reč o prvoj psihotičnoj epizodi kod obolelih pacijenata, ordinirati terapiju u skladu sa aktuelnom kliničkom slikom, a potom nastaviti sa pažljivim praćenjem pacijenta. Ukoliko se psihotični simptomi ne povlače, uprkos primenjenoj odgovarajućoj psihofarmakoterapiji uz pojavu pridruženih neuroloških ispada u vidu kognitivnih poremećaja, novonastalih epileptičkih napada i autonomne disfunkcije, neophodno je razmotriti mogućnost postojanja AE. Posebnu pažnju je neophodno obratiti ukoliko se radi o obolelim osobama ženskog pola mlađe životne dobi, posebno sa medicinskom istorijom ginekoloških komorbiditeta, u prvom redu ovarijalnog teratoma. Da bi se postavila adekvatna dijagnoza, blagovremeno započela terapija i izbegle mnogobrojne komplikacije, dijagnostičke procedure je neophodno započeti što pre. Cilj ovog rada je razmatranje moguće dijagnoze AE, posebno NMDA encefalitisa kod pacijenata sa novonastalom psihotičnom simptomatologijom. **Prikaz slučaja:** Kako bi se kliničarima približila oblast imunski posredovanih neuroloških oboljenja koja se mogu manifestovati predominantnom psihijatrijskom simptomatologijom i olakšao dalji rad, dat je prikaz slučaja jednog pacijenta sa NMDA AE kroz prizmu psihotičnih manifestacija bolesti. **Zaključak:** Pravovremeno prepoznavanje autoimune etiologije psihijatrijskih poremećaja je od velike važnosti u cilju što ranijeg postavljanja dijagnoze AE i pravovremenog započinjanja odgovarajućeg lečenja što je preduslov za povoljan tok i ishod ovih oboljenja.

REFERENCES

1. Graus F, Titular M, Balu R, Benseler S, Bien C, Celluci T, et al. A clinical approach to diagnosis of autoimmune encephalitis. *Lancet Neurol.* 2016;15:391-404.
2. Sechi E, Flanagan E. Antibody-mediated autoimmune disease of the CNS: challenges and approaches to diagnosis and management. *Front Neurol.* 2021 7;12:673339. doi: 10.3389/fneur.2021.673339 eCollection 2021.
3. Marić N, Andrić Petrović S, Pavlović Z, Petrović I. Mozak u plamenu – da li se iza prve psihotične epizode krije anti-NMDAR encefalitis? *Engrami.* 2017;39(3-4):39–47.
4. Jewett BE, Thapa B. Physiology, NMDA Receptor. [Updated 2022 Dec 11]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK51949/5/>
5. Steiner J, Pruss H, Kohler S, Frodl T, Hasan A, Falkai P. Autoimmune encephalitis with psychosis: Warning signs, step-by-step diagnostics and treatment. *World J Biol Psychiatry.* 2020;21(4):241-54.
6. Pollak T, Lennox B, Muller S, Benros M, Pruss H, Tebartz van Elst, et al. Autoimmune psychosis: an international consensus on an approach to the diagnosis and management of psychosis of suspected autoimmune origin. *Lancet Psychiatry.* 2020;7(1):93-108.
7. Chen L, Zhu L, Lu D, Dai S, Han Y, Wu Z et al. Association between autoimmune encephalitis and epilepsy: Systematic review and meta-analysis. *Seizure.* 2021;91:346-359. doi: 10.1016/j.seizure.2021.07.005. Epub 2021 Jul 12. PMID: 34284303.
8. Niehusmann P, Dalmau J, Rudlowski C, et al. Diagnostic Value of N-methyl-D-aspartate Receptor Antibodies in Women With New-Onset Epilepsy. *Arch Neurol.* 2009;66(4):458–464. doi:10.1001/archneurol.2009.5
9. Liu Y, Tian Y, Guo R, Xu X, Zhang M, Li Z et al. Anti-NMDA Receptor Encephalitis: Retrospective Analysis of 15 Cases, Literature Review, and Implications for Gynecologists. *J Healthc Eng.* 2022;2022:4299791. doi: 10.1155/2022/4299791. PMID: 35340259; PMCID: PMC8941556.8.
10. Rickards H, Jacob S, Lennox B, Nicholson T. Autoimmune encephalitis: a potentially treatable cause of the mental disorder. *Advances in Psychiatric Treatment.* 2014;20(2):92-100. doi:10.1192/apt.bp.113.011304
11. Dalmau J, Lancaster E, Martinez-Hernandez E, Rosenfeld MR, Balice-Gordon R. Clinical experience and laboratory investigations in patients with anti-NMDAR encephalitis. *Lancet Neurol.* 2011;10(1):63-74. doi: 10.1016/S1474-4422(10)70253-2.
12. Jia H, Xie X, Qi F, Wang L, Wang L, Che F. Anti-NMDAR encephalitis with simultaneous hypertrophic pachymeningitis in a 68-year-old male: a rare case report. *BMC Neurol.* 2019;19(1):215. doi: 10.1186/s12883-019-1444-x. PMID: 31472692; PMCID: PMC6717633
13. Elkady A, Anany A, Sharawy A, Bakheet M. Abnormal presentation and imaging finding of anti-NMDA receptor encephalitis, a report of two cases. *Journal of the Neurological Sciences.* 2021;429:118830. doi:10.1016/j.jns.2021.118830
14. Sansing LH, Tüzün E, Ko MW, Baccon J, Lynch DR, Dalmau J. A patient with encephalitis associated with NMDA receptor antibodies. *Nat Clin Pract Neurol.* 2007;3(5):291-6. doi: 10.1038/ncpneuro0493. PMID: 17479076; PMCID: PMC1936221.

■ The paper was received / Rad primljen 26.08.2025.
Accepted / Rad prihvaćen: 13.09.2025.