International Journal of Pharmacy and Pharmaceutical Research (IJPPR)
Volume 31, Issue 4, April 2025 **ijppr.humanjournals.com** ISSN: 2349-7203

Anti-N-Methyl-D-Aspartate (NMDA) Receptor Autoimmune Encephalitis in a 15-Years- Old Female: A Case Report

Vivek A K[1], Gayathri Ajaya Prasad[1], Dr. Alnon L J[2], Dr. Shaiju S Dharan[3]

1. Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram, 695124 India.

Received: 2025-4-01 Revised: 2025-4-12 Accepted: 2025-4-20

ABSTRACT

Autoimmune encephalitis (AE) refers to a group of disorders caused by immune system dysfunction, leading to brain inflammation. It is often associated with autoantibodies targeting neuronal antigens and presents with symptoms such as cognitive impairment, seizures, psychiatric disturbances, and movement abnormalities.

Here, we report the case of a 15-year-old female who was admitted with complaints of fever for 18 days, abnormal stiffening of the limbs with right eye deviation, and multiple episodes of loss of consciousness over 15 days. She was initially evaluated and treated at MCH and diagnosed with anti-NMDA receptor autoimmune encephalitis.

The patient received five doses of intravenous immunoglobulin (IVIG). Due to persistent fever and recurrent seizures, she was administered intravenous steroids and antiepileptic medications. Intravenous antibiotics were also initiated. In view of ongoing seizure episodes, inj. Lacosamide, tab. Clobazam, and tab. Perampanel were added. Physiotherapy was started to support neurological recovery.

During the course of treatment, the patient's general condition showed slight improvement. However, due to financial constraints, her relatives requested discharge, and she was discharged against medical advice.

Keywords: Autoimmune encephalitis, Anti-NMDA receptor encephalitis, Intravenous immunoglobulin (IVIG), Neuronal autoantibodies.

INTRODUCTION

Autoimmune encephalitis (AE) is a condition where the body's immune system mistakenly attacks the brain, causing inflammation. This happens due to certain antibodies that interfere with nerve function. Common types include those affecting N-methyl-D-aspartate receptor (NMDAR), leucine-rich glioma-inactivated 1 (LGI1), and contactin-associated protein-like 2 (CASPR2)[(1)]. Symptoms can range from mood changes and memory loss to seizures and issues with body functions like heart rate and breathing. Identifying and treating AE early can greatly improve recovery.

AE happens when the immune system produces harmful antibodies that target specific proteins on nerve cells[(2)]. These proteins play a role in brain signaling, and when they are attacked, brain activity is disturbed. The most well-known types of AE are linked to antibodies against proteins such as N-methyl-D-aspartate receptor (NMDAR), leucine-rich glioma-inactivated 1 (LGI1), and contactin-associated protein-like 2 (CASPR2).

Anti-NMDAR encephalitis is the most common form and usually affects young adults. In some cases, it is linked to ovarian tumors. LGI1 and CASPR2-related encephalitis are more common in middle-aged and older adults, leading to conditions such as limbic encephalitis and Morvan syndrome.

Anti-NMDA receptor encephalitis is a rare but serious autoimmune disorder that primarily affects young individuals, especially females. The estimated incidence is approximately 1.5 cases per million annually, with a mortality rate of 5–11.5%. The condition is often misdiagnosed due to its psychiatric and neurological symptoms, including seizures, cognitive decline, movement

abnormalities, and autonomic dysfunction. Diagnosis is confirmed through cerebrospinal fluid (CSF) analysis and detection of anti-NMDA receptor antibodies[3]. MRI findings may be normal or show subtle inflammation. Early treatment with immunotherapy, including IVIG, corticosteroids, and plasmapheresis, significantly improves outcomes, though 10–30% of patients experience relapses within two years. Supportive therapies such as physiotherapy and long-term immunosuppressants help prevent recurrence and manage residual symptoms. Despite advances in treatment, around 20% of patients may develop long-term neurological deficits or succumb to the disease[4]. Early recognition and aggressive management are crucial in improving survival rates and functional recovery.

CASE REPORT:

A 15 year old female patient was admitted with complaints of fever for 18 days, abnormal stiffening of the limbs with right eye deviation, and multiple episodes of loss of consciousness over 15 days. The patient was initially evaluated and treated at MCH and diagnosed with anti-NMDA receptor autoimmune encephalitis based on clinical presentation, cerebrospinal fluid (CSF) and serological testing.

Her peripheral smear study revealed normocytic and normochromic red blood cells (RBCs), with a few polychromatophilic cells observed. No nucleated RBCs, hemoparasites, or inclusions were noted. The white blood cell (WBC) count was within normal limits, but differential counts indicated relative neutrophilia. No immature cells or blasts were detected. The platelet count was also within normal limits, appearing scattered singly and in clumps, with normal morphology. The smear findings suggested normocytic normochromic anemia with moderate relative neutrophilia.

The patient was started on intravenous immunoglobulin (IVIG) therapy (five doses). Due to persistent fever and recurrent seizures, she was administered intravenous steroids and antiepileptic medications. Intravenous antibiotics were also initiated. Given the ongoing seizure episodes, inj. Lacosamide and tab. Clobazam were added to control seizures. Supportive physiotherapy was introduced to aid in neurological recovery.

OUTCOME AND FOLLOW UP:

During the course of treatment, the patient showed slight improvement. However, due to financial constraints, her family requested discharge, and she was discharged against medical advice (DAMA). She was advised to continue regular follow-ups for neurological and psychiatric evaluations, along with ongoing antiepileptic treatment and physiotherapy. The long-term outcome remains uncertain, as patients with anti-NMDA receptor encephalitis often require prolonged immunosuppressive therapy and rehabilitation for recovery.

MRI scan of the brain showed a small bright area in the right thalamus without enhancement (fig.1).

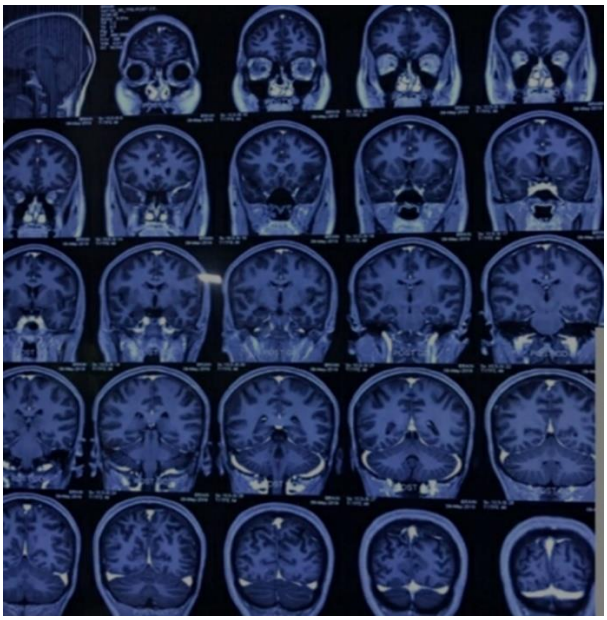

Fig:1

DISCUSSION

Autoimmune encephalitis occurs when the immune system produces antibodies that attack proteins in nerve cells, leading to brain inflammation and impaired nerve signaling[5]. In anti-NMDAR encephalitis, these antibodies reduce the number of NMDA receptors, disrupting brain communication and causing psychiatric and neurological symptoms [6]. Other types, like LGI1-associated encephalitis, affect potassium channels in the brain, leading to seizures and memory problems.

Pathophysiology

Autoimmune encephalitis is mediated by autoantibodies targeting neuronal proteins, leading to synaptic dysfunction and neuroinflammation. In anti-NMDAR encephalitis, antibodies internalize NMDA receptors, reducing glutamatergic signaling and causing neuropsychiatric symptoms. Other forms, such as LGI1-associated encephalitis, involve disruption of voltage-gated potassium channels, contributing to seizures and memory impairment.

Diagnosis

AE diagnosis relies on a combination of clinical criteria, MRI, electroencephalography (EEG), and antibody testing. Graus et al. (2016) proposed diagnostic criteria emphasizing early symptom recognition and antibody confirmation [7].

Treatment Approaches

The treatment of AE includes:

- **First-line therapy**: High-dose corticosteroids, intravenous immunoglobulin (IVIG), or plasmapheresis.
- **Second-line therapy**: Rituximab or cyclophosphamide for refractory cases.
- **Supportive care**: Management of seizures, autonomic instability, and psychiatric symptoms.
- A meta-analysis by Titulaer et al. (2013) demonstrated that early immunotherapy improves functional outcomes in anti-NMDAR encephalitis, with nearly 80% of patients achieving substantial recovery [8].

Comparison with Other Case Reports

A case report by Yadav et al. (2022) described a 22-year-old female presenting with fever, altered sensorium, and abnormal body movements, initially misdiagnosed as tuberculous meningoencephalitis. Unlike our patient, she showed worsening symptoms over time, eventually leading to a correct diagnosis of anti-NMDAR encephalitis after further testing. Both cases were treated with IVIG and steroids; however, the Yadav et al. case included long-term immunosuppressive therapy (Azathioprine) and tumor screening, which led to the discovery of an ovarian dermoid cyst requiring surgical removal[9].

Another case by Bhat et al. (2018) detailed a 26-year-old female who initially presented with nausea, vomiting, memory loss, and personality changes. She rapidly developed severe neurological symptoms, including aphasia and decreased consciousness. Imaging was unremarkable, but CSF analysis showed lymphocytic pleocytosis. Unlike our patient, she was found to have an 8 cm ovarian teratoma, which was surgically removed. Her neurological symptoms resolved rapidly post-surgery, highlighting the critical role of tumor removal in treatment. This case emphasizes the importance of early malignancy screening in suspected anti-NMDAR encephalitis cases. [10]

These comparisons show that screening for tumors is important, as patients with ovarian teratomas often recover better after surgery. In our case, the lack of tumor screening and financial difficulties that led to DAMA made it harder to ensure a good long-term outcome.

Prognosis

The prognosis of AE varies based on the specific antibody involved and treatment response. Patients with anti-NMDAR encephalitis tend to have a better prognosis with early immunotherapy, while LGI1-related encephalitis generally shows favorable outcomes with steroid therapy alone. Long-term follow-up is essential to monitor for relapses, which occur in approximately 10-25% of cases.

Conclusion

Autoimmune encephalitis is an important differential diagnosis in patients with acute neuropsychiatric symptoms. Timely recognition, antibody testing, and aggressive immunotherapy are key to improving outcomes. Further research is needed to optimize treatment strategies and understand long-term implications.

Patient consent

The complete written informed consent was obtained from patient and by stander for the publication in this study.

REFERENCES

1. Kunchok A, McKeon A, Zekeridou A, Flanagan EP, Dubey D, Lennon VA, et al. . Autoimmune/Paraneoplastic encephalitis antibody biomarkers: frequency, age, and sex associations. Mayo Clin Proc (2022) 97(3)
2. Van Sonderen A, Schreurs MW, Wirtz PW, Sillevis Smitt PA, Titulaer MJ. From VGKC to LGI1 and Caspr2 encephalitis: The evolution of a disease entity over time. Autoimmun Rev (2016) 15(10):970–4.
3. Yeo T, Chen Z, Yong KP, Wong PYW, Chai JYH, Tan K. Distinction between anti-VGKC-complex seropositive patients with and without anti-LGI1/CASPR2 antibodies. J Neurol Sci (2018) 391:64–71.
4. Monti G, Giovannini G, Marudi A, Bedin R, Melegari A, Simone AM, et al. Anti-NMDA receptor encephalitis presenting as new onset refractory status epilepticus in COVID-19. Seizure (2020) 81:18–20.
5. Subramanyam M, Gupta SB, Chamraj S. Caspr2 autoimmune encephalitis with COVID-19 infection. J Assoc Physicians India (2022) 70(6):11–2.
6. Ayatollahi P, Tarazi A, Wennberg R. Possible autoimmune encephalitis with claustrum sign in case of acute SARS-CoV-2 infection. Can J Neurol Sci (2021) 48(3):430–2.
7. Graus, F., Titulaer, M. J., Balu, R., et al. (2016). A clinical approach to diagnosis of autoimmune encephalitis. *The Lancet Neurology*, 15(4), 391-404.
8. Titulaer, M. J., McCracken, L., Gabilondo, I., et al. (2013). Treatment and prognostic factors for long-term outcome in patients with anti-NMDA receptor encephalitis: an observational cohort study. *The Lancet Neurology*, 12(2), 157-165.
9. Bikash Yadav et.al. Anti-N-methyl-D-aspartate Receptor Autoimmune Encephalitis: A Case report J Nepal Med Assoc 2022;60(255):962-4.
10. Pavan Bhat et.al, A case report: anti-NMDA receptor encephalitis Journal of community hospital internal medicine perceptives, 2018, vol 8, NO. 3, 158–160.

How to cite this article:
Vivek A K et al. Ijppr.Human, 2025; Vol. 31 (4): 255-258.

Conflict of Interest Statement: All authors have nothing else to disclose.