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A CASE REPORT OF ANTI-NMDA RECEPTOR ENCEPHALITIS

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ABSTRACT

Background: Anti-NMDA receptor encephalitis is a form of autoimmune encephalitis which is characterized by the presence of IgG antibodies against the NR1 subunit of NMDA receptors. While often associated with paraneoplastic origins, the exact triggers of autoantibody production remain unclear in many cases. It predominantly affects children and young adults, with a female preponderance. Cerebrospinal fluid (CSF) analysis is used to confirm the diagnosis. **Case Report:** A 26 year old female with no comorbidities presented with headache and projectile vomiting followed by altered behaviour. She was taken to a local hospital and was given antipsychotics. After 2 days, she developed recurrent episodes of generalized seizures. On examination, she was disoriented and vitals were unstable. MRI brain was normal. CSF autoimmune panel came as strongly positive for anti-NMDA receptor antibody. She was managed with immunosuppressive medications, multiple antiepileptics and supportive care. Her clinical condition gradually improved and seizures subsided. Whole-body PET–CT performed in search of a tumoral origin was negative. **Conclusion:** Anti-NMDA receptor encephalitis is a rare and often underdiagnosed condition which requires prompt medical attention and multidisciplinary management. Clinicians should consider anti-NMDA receptor encephalitis in the differential diagnosis for patients presenting with encephalitis, refractory seizures, or acute psychosis, before attributing symptoms to a psychiatric illness. Delayed immunotherapy is associated with poor prognosis.

INTRODUCTION

Anti-NMDA receptor encephalitis is a complex autoimmune disorder that affects the central nervous system, characterized by the presence of IgG antibodies targeting the NR1 subunit of NMDA receptors. While often associated with paraneoplastic origins, the exact triggers of autoantibody production remain unclear in many cases. It predominantly affects children and young adults, with a significantly higher incidence in females compared to males (4:1 ratio)[1]. The clinical presentation typically begins with nonspecific symptoms mimicking viral infections, followed by rapid progression to complex neuropsychiatric manifestations and autonomic instability. Due to its variable initial presentation, the condition is frequently underdiagnosed or misdiagnosed as other pathologies, including viral encephalitis. When suspected, diagnostic evaluation often involves EEG, brain MRI, and cerebrospinal fluid analysis to confirm the presence of specific antibodies. Therapeutic approaches focus on immunosuppression and tumor resection when indicated.

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CASE REPORT

A 26 year old married female, with no significant comorbidities, presented with history of headache and non-projectile vomiting 2 weeks back. On the subsequent days, she developed altered behaviour in the form of irrelevant talk, reduced interest in doing daily activities and caring children, reduced sleep and aggressive behaviour towards relatives. She was taken to a local hospital and was started on antipsychotics. After 2 days, she developed 2 episodes of generalized tonic clonic seizures lasting for 10minutes. No history of fever, cough, breathlessness, weakness, facial deviation, diplopia, oral ulcers, arthralgia or photosensitivity. No history of miscarriages or neuropsychiatric illness among the family members. On examination, she was conscious but disoriented. General examination was unremarkable. No thyroid or breast swelling. Vitals showed pulse rate of 150/min, systolic BP of 90mmhg, normal temperature and oxygen saturation. Pupils were bilaterally equal and reactive to light. Cranial nerves, motor and sensory system examinations were unremarkable. There was no neck stiffness, cerebellar signs or papilledema.

Plantar was bilaterally equivocal. Other systemic examinations were unremarkable.

Blood investigations are given in the table1. Serum CPK was markedly elevated. Anti- TPO, viral markers, ANA profile and toxicology screening were negative. No evidence of hemorrhage in CT head. Lumbar puncture was done to rule out CNS infection. The results are given in table 2. CSF samples were sent for testing viral and autoimmune panel. MRI brain had no evidence of infarct, inflammation or cortical vein thrombosis. EEG was abnormal with background slowing and bifrontal spikes. Chest x-ray and USG abdomen with pelvis were normal.

Table 1. Blood investigation results of the patient	
Hemoglobin	13.5 g/dl
Total count	24100 cells/mm^3
Differential count	N76 L14
ESR	45
Platelet	2.86 Lakh
Urea/Creatinine	60/4.5
Sodium	135
Potassium	4.5
Random blood sugar	185
Calcium/Phosphorous	8.1/4.4
Magnesium	1.9
Uric acid	9.1
Total bilirubin	0.5
Direct bilirubin	0.2
SGOT/SGPT	290/88
ALP	123
CPK	16480

Table 2. CSF study findings of the patient	
CSF parameters	
Total count	5 cells
Differential count	Lymphocyte 100
Sugar	83
Protein	6
ADA	Negative

She developed recurrent episodes of seizures and was shifted to ICU. A working diagnosis of encephalitis with acute kidney injury and rhabdomyolysis was made. She was started on with IV antibiotics, IV acyclovir, antiepileptics, IV fluids and other supportive measures. Meanwhile CSF viral panel results came as negative. She had persistent seizures and hence antiepileptics were hiked. Her renal and liver parameters worsened. Acyclovir was stopped and hemodialysis was initiated. Serum creatinine and transaminases improved but seizures and tachycardia persisted. Meanwhile she was intubated for saturation fall and midazolam infusion was started. CSF autoimmune panel came as strongly positive for anti- NMDA receptor antibody.

She was given methylprednisolone pulse therapy for 3 days. Neuromedicine consultation was sought. Since she was not responding to steroid pulse, IV Immunoglobulin therapy was administered. In view of the refractory disease, inj. Rituximab was given and plasmapheresis was done to the patient. Her clinical condition gradually improved and seizures were controlled. She was extubated and vitals became stable. Whole-body PET–CT performed in search of a tumoral origin, such as ovarian teratoma, was negative. The patient returned home after 4 months of hospitalization. At 6-month follow-up visit, she still had functional deficit and mild cognitive dysfunction.

DISCUSSION

Anti-NMDA receptor encephalitis predominantly affects children and young adults, with a notable female preponderance. The disease often originates from paraneoplastic causes, particularly in females over 18 years, where ovarian teratomas are common (approximately 50%)[2]. In contrast, men rarely present with tumors. The clinical presentation of anti-NMDAR encephalitis can be divided into five distinct phases: prodromal, psychotic, unresponsive, hyperkinetic, and recovery. Adults and children exhibit different presenting features, with adults typically showing psychiatric symptoms and children often experiencing movement disorders or seizures. The onset is often abruptwith various psychiatric symptoms emerging within days to weeks, in contrast to the gradual progression of primary psychiatric diseases. These symptoms include hallucinations, schizoaffective episodes, depression, mania, and addictive or eating disorders. Many patients get admitted to psychiatric units for symptomatic management and some may develop neurological features later. Seizures, both focal and generalized are common particularly in children and male adults, and may be resistant to antiepileptic drugs. Autonomic instability, including labile blood pressure, cardiac arrhythmias and central hypoventilation necessitate ICU admission and mechanical ventilation.

Diagnosis can be challenging due to the nonspecific clinical presentation. Research has shown that anti-NMDA antibodies are present in 50% of patients with lethargic dyskinetic encephalitis[3]. Furthermore, patients with herpes simplex virus (HSV) infection may experience seroconversion with anti-NMDA antibodies during relapse, which is not directly related to HSV reactivation[4]. Confirmation of the diagnosis relies on detecting IgG antibodies against GluN1 subunits of NMDA receptors in cerebrospinal fluid, a test that may not be readily available in emergency settings[5]. While EEG often shows abnormal results, the findings are typically nonspecific, featuring slow and disorganized epileptic activity. Brain MRI results can be normal or show increased FLAIR or T2 signal in cortical or subcortical areas in some cases[6]. The differential diagnosis for anti-NMDAR encephalitis includes acute primary psychiatric disorders, neuroleptic malignant syndrome, malignant catatonia, drug intoxication and viral encephalitis. Due to the lack of standardized treatment protocols, therapy should be tailored to each patient based on factors like age, symptom severity, and the presence or absence of a tumor.

The primary approach for treating autoimmune encephalitis involves immunosuppressive therapy to mitigate inflammation and prevent further brain damage, alongside tumor resection when indicated. Initial treatment options include intravenous methylprednisolone, immunoglobulin G, or plasmapheresis. If clinical improvement is not observed, second-line treatments such as rituximab or cyclophosphamide may

be considered,with mycophenolate mofetil serving as an alternative option due to its selective anti-proliferative activity on lymphocytes and relatively favourable side effect profile[7]. For patients with severe and treatment-resistant disease, bortezomib or tocilizumab may be used as third-line therapy[8]. While immunosuppression is the cornerstone of treatment, emergency room management is often necessary for symptoms like seizures, with benzodiazepines and antiepileptic medications. Patients with severe disease may require intensive care management with mechanical ventilation and cardiac pacing due to autonomic dysfunction. Long-term care and multidisciplinary rehabilitation are often necessary and recovery can take months to years. Factors associated with poor prognosis include delayed immunotherapy, severe symptoms,presence of underlying cancer and abnormal EEG findings. Early treatment initiation is crucial for optimal outcomes and faster recovery. Long-term sequelae may include persistent cognitive impairment, psychiatric symptoms, seizures or movement disorders.

CONCLUSION

Anti-NMDA receptor encephalitis is a rare and often underdiagnosed condition which requires prompt medical attention and multidisciplinary management. Clinicians should consider anti-NMDA receptor encephalitis in the differential diagnosis for patients presenting with encephalitis, refractory seizures, or acute psychosis, before attributing symptoms to a psychiatric illness. Delayed immunotherapy is associated with worse prognosis, emphasizing the importance of timely treatment. Long-term recovery requires continuous monitoring and rehabilitation to address any lingering complications and to support patients in achieving the best possible outcomes.

STATEMENTS AND DECLARATIONS

- CONFLICT OF INTEREST – The author declare no conflict of interest
- FINANCIAL GRANTS – There is no external funding
- ETHICS – There is no ethical violation

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