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Management of Autoimmune Encephalitis Patients with Refractory Status Epilepticus

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ABSTRACT

Introduction: Autoimmune encephalitis (AE) is a leading cause of non-infectious encephalitis. Its diagnosis remains challenging due to the often non-specific clinical presentation and difficulties in confirming antibody-negative cases. Comprehensive evaluation is essential to establish the diagnosis of AE.

Case Report: We report a 25-year-old male presenting with decreased consciousness and seizures, accompanied by behavioral changes over the preceding 10 days. Electroencephalography (EEG) showed normal waveforms, and cerebrospinal fluid (CSF) analysis did not suggest infection. Brain CT scans were unremarkable, and serologic tests for herpes simplex virus (HSV) IgG and IgM were non-reactive. Notably, anti-NMDAR antibodies were positive. The patient was managed in the ICU with mechanical ventilation, sedated with propofol, and administered phenytoin for seizure control. Empirical treatment with acyclovir was given for 10 days, with no clinical improvement. First-line immunotherapy with methylprednisolone (1g/day for 5 days) was initiated but failed to produce neurological recovery. On day 14, CSF analysis indicated autoimmune etiology; plasma exchange was performed over three days, resulting in clinical improvement.

Conclusion: Diagnosing and managing antibody-negative AE remains challenging. Clinical judgment, supported by the exclusion of differential diagnoses and the absence of characteristic radiological and immunological findings, can justify the initiation of immunosuppressive therapy or plasma exchange, which may lead to significant clinical improvement.

Keywords

Autoimmune encephalitis, Immunosuppressants, NMDAR, Plasma exchange

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INTRODUCTION

Autoimmune encephalitis is one of the most common forms of non-infectious encephalitis, caused by autoantibodies targeting neuronal epitopes, such as synaptic surface structures (e.g. receptors, ion channels, or supporting proteins) or intracellular antigens, including onconeural antigens. The spectrum of autoimmune encephalitis has expanded significantly over the past 15 years, primarily because of ongoing research on various neuronal autoantibodies. Most patients present with altered levels of consciousness and may experience symptoms, such as fever, seizures, movement disorders, or focal neurological deficits [1,2].

Diagnosis relies heavily on lumbar puncture and cerebrospinal fluid (CSF) analysis; however, imaging and electroencephalography (EEG) can also provide valuable diagnostic support. Although acyclovir treatment has been shown to dramatically improve outcomes in herpes simplex virus (HSV) encephalitis, the optimal management of autoimmune encephalitis remains uncertain. Many patients with autoimmune encephalitis have residual physical or neuropsychological deficits that necessitate long-term multidisciplinary care.

Identifying the exact aetiology of the condition is crucial for tailoring appropriate therapeutic regimens, which is the key to successful treatment [3,4].

This case report discusses the diagnosis and management of a patient with autoimmune encephalitis presenting with refractory status epilepticus in the intensive care unit (ICU). Management includes emergency stabilisation (airway, breathing, and circulation), seizure control, and its aetiology, as well as essential supportive therapies, including fluid management, nutrition, analgesia, sedation, thromboprophylaxis, stress ulcer prophylaxis, blood glucose control, antibiotic therapy, and prevention of ventilator-associated pneumonia (VAP) in critically ill ICU patients [1].

CASE REPORT

This case report outlines the clinical management of a 25-year-old male, Mr. M.S., who was diagnosed with refractory status epilepticus and suspected autoimmune encephalitis. The patient initially presented with a 10-day history of a gradual decline in consciousness. The early signs included difficulty in communication, inability to follow commands, and subsequent agitation and restlessness. Over the last three days prior to hospitalisation, the patient became increasingly lethargic and non-responsive. The patient exhibited abnormal hand movement, frequent headaches, and seizures. The seizures were characterised by upward eye deviation and generalised stiffness of all limbs, lasting approximately three minutes. The patient also had recurrent vomiting but without any fever or limb weakness.

On arrival at the emergency department of RSHS on 12 October 2024 the patient was assessed using the Glasgow Coma Scale (GCS) score of E4 M4 V1 and vital signs indicating slight haemodynamic instability. Initial CT tomography and electroencephalography (EEG) performed at an external facility were normal. Given the patient's clinical history and presentation, he was diagnosed with non-convulsive status epilepticus, with a differential diagnosis of viral or bacterial encephalitis and autoimmune encephalitis.

The patient was immediately intubated and placed on mechanical ventilation. He was started on sedation, anticonvulsant therapy with acyclovir and phenytoin, and supportive measures, including fluid resuscitation and nutritional support. After five days of treatment, his clinical condition showed no improvement, and the seizures continued when sedation was interrupted. Immunological testing for anti-NMDAR antibodies was performed, and plasma exchange therapy was considered part of the treatment protocol.

Table 1. Hematology and Electrolytes

Parameter	Day 0	Day 7	Day 10	Day 16
Hemoglobin (g/dL)	12	10.5	10.2	10.3
Hematocrit (%)	34.9	31.5	31.7	30.7
Leukocyte Count (cells/µL)	9060	8210	14180	14180
Platelet Count (cells/µL)	203000	306000	270000	227000
Electrolytes (Na/K)	137/3.9	137/4.1	140/4.0	136/3.6
Lactate (mmol/L)	1.8	-	-	-
Procalcitonin (ng/mL)	-	-	0.27	-

Laboratory investigations revealed normal haemoglobin levels, platelet count, and kidney function. Cerebrospinal fluid (CSF) analysis revealed a glucose level of 66 mg/dL, total protein level of 42 mg/dL, and 71 cells/µL with 85.9% polymorphonuclear leukocytes (PMNs), indicative of an inflammatory process. The CSF microbiological culture result was negative, supporting the hypothesis of autoimmune encephalitis. Immunological testing for anti-NMDAR antibodies confirmed the diagnosis (Table 1).

Over the following days, the patient's treatment regimen was modified to include methylprednisolone and five sessions of plasma exchange. By day 16 post-intubation, after two sessions of plasma exchange, the patient's GCS improved to E4M6Vt, allowing for extubation and transfer to a high care unit (HCU) on day 21. The patient's fluid balance and urine output were closely monitored, and enteral nutrition was well tolerated.

Table 2. Gas Analysis and Microbial Results

Test	Day 0	Day 10	Day 16
AGDA (pH/pCO2/pO2)	7.392/38.7/154.7	7.430/43.1/157.3	7.490/34.9/177.5
Sputum Culture	Negative	Acinetobacter baumanii, Klebsiella pneumonia	Acinetobacter baumanii, Klebsiella pneumonia
Antibiotic Sensitivity	N/A	Gentamycin, Co-trimoxazole, Levofloxacin, Meropenem, Amikacin, Ampicillin-Sulbactam	Gentamycin, Co-trimoxazole, Levofloxacin, Meropenem, Amikacin, Ampicillin-Sulbactam

This case highlights the complexity of managing refractory status epilepticus in a patient with suspected autoimmune encephalitis. Confirmation of the diagnosis through anti-NMDAR antibody testing and a positive response to plasma exchange therapy demonstrate the importance of early intervention. This report underscores the need for ongoing research to optimise treatment protocols for autoimmune encephalitis, particularly in cases of refractory seizures and complex presentations.

Table 3. Conclusion of Laboratory and Antibiotic Treatment Results

Examination	Results
Hemoglobin	12 g/dL initially, decreased to 8.2 g/dL on day 16
Leukocyte Count	Elevated initially at 9,060/μL, peaked at 14,180/μL
Platelet Count	Normal range (203,000/μL to 371,000/μL)
CSF Analysis	Glucose 66 mg/dL, Protein 42 mg/dL, WBC 71/μL (85.9% PMNs)
Anti-NMDAR Antibodies	Positive
CT Scan	No ischemic lesions, hemorrhage, or SOL
Sputum Culture	Acinetobacter baumanii, Klebsiella pneumonia
Antibiotic Sensitivity	Gentamycin, Co-trimoxazole, Levofloxacin, Meropenem, Amikacin, Ampicillin-sulbactam

The laboratory results indicated significant findings, including a decrease in haemoglobin levels from 12 g/dL to 8.2 g/dL over the course of 16 days, suggesting ongoing blood loss or haemolysis. The leukocyte count initially elevated at 9,060/μL, peaked at 14,180/μL, reflecting an inflammatory or infectious process. The platelet count remained within the normal limits throughout the observation period. CSF analysis showed glucose at 66 mg/dL, protein at 42 mg/dL, and a mild increase in white blood cells (71/μL) with a predominance of neutrophils (85.9%), indicative of possible infection or inflammation (Table 3). The presence of anti-NMDAR antibodies suggested autoimmune encephalitis. Imaging via CT scan revealed no ischemic lesions, hemorrhage, or space-occupying lesions (SOL), ruling out major structural brain abnormalities. Sputum culture identified Acinetobacter baumanii and Klebsiella pneumoniae, with antibiotic sensitivity to multiple agents, including gentamycin and meropenem, though Klebsiella exhibited resistance to all antibiotics tested, indicating potential treatment challenges. This comprehensive laboratory work highlights a complex case of autoimmune encephalitis with secondary bacterial infection, necessitating a tailored approach to treatment.

DISCUSSION

Encephalitis is defined as inflammation of the brain parenchyma associated with neurological dysfunction [1]. While pathological examination and brain tissue biopsy are considered the "gold standard" for diagnosis, these procedures are rarely performed premortem because of the associated morbidity from invasive neurosurgical procedures. The proposed definitions for encephalitis and encephalopathy of suspected infectious etiology are based on expert consensus and available literature. Validation through cohort studies and additional prospective studies will be essential to refine and improve case definitions of encephalitis [2-4].

The majority of the pathogens reported to cause encephalitis are viruses. However, despite extensive testing, the aetiology of encephalitis remains unknown in most patients. One of the challenges in encephalitis is identifying the relevance of infectious agents detected outside the central nervous system (CNS), which may

play a role in neurological manifestations without directly attacking the CNS. It is also crucial to distinguish between infectious encephalitis and autoimmune encephalitis [1]. In this case, the diagnosis of encephalitis was established based on major criteria, including a decrease in consciousness lasting more than 24 h, with minor criteria such as fever, seizures, and EEG abnormalities. Although the symptoms overlap with encephalopathy caused by refractory hypoglycaemia, normal blood glucose levels, along with the absence of consciousness improvement, help rule out this differential diagnosis.

Cerebrospinal fluid (CSF) analysis plays a vital role in confirming the aetiology of encephalitis, except in cases with contraindications. Detection of IgM antibodies for specific viruses in CSF samples from patients with encephalitis caused by various viruses is considered diagnostic for neuroinvasive diseases. PCR amplification has significantly enhanced the ability to diagnose CNS infections, particularly viral infections, such as those caused by herpes simplex virus (HSV) [1].

In this case, CSF analysis showed a glucose level of 66 mg/dL, total protein level of 42 mg/dL, clear appearance, and 71 cells/μL (85.9% PMN and 14.1% MN). No growth of Gram-positive or Gram-negative bacteria, fungi, or Mycobacterium tuberculosis was observed. Immunological testing for HSV IgG and IgM was nonreactive. However, the presence of anti-NMDAR antibodies in the CSF confirmed an autoimmune aetiology, ruling out bacterial and tuberculosis causes of encephalitis. The clinical history of behavioural changes prior to hospitalisation further supports the likelihood of autoimmune encephalitis [3].

Acute management of encephalitis should address the airway, breathing, and circulation as primary steps. Reduced consciousness caused by metabolic encephalopathy, seizures, or cerebral oedema may impair airway reflexes and require endotracheal intubation. Systemic conditions, such as pneumonia, may also complicate oxygenation, necessitating mechanical ventilation support after intubation [5-7].

In this patient, intubation and mechanical ventilation were performed because of loss of consciousness and recurrent seizures. The patient had recurrent seizures, and initial anticonvulsant therapy was ineffective. A diagnosis of refractory status epilepticus was established, and treatment with propofol and phenytoin successfully controlled the seizures. Propofol was used for sedation and seizure control, while phenytoin modulated synaptic neurotransmitter release by stabilising sodium channels, reducing neuronal action potentials, and halting seizures [6].

Seizures and status epilepticus (SE) are common complications in patients with encephalitis. Seizures should be treated immediately with first-line agents, such as lorazepam or midazolam, followed by second-line antiepileptic drugs, such as fosphenytoin, levetiracetam, or valproic acid in persistent cases. In refractory status epilepticus, third-line agents, including barbiturates, propofol, or ketamine, are used [5].

Non-convulsive status epilepticus (NCSE) is characterised by persistent behavioural or consciousness changes without overt seizures but can still result in neurological injury if left untreated [8]. In this case, no further seizures were observed after administration of propofol and phenytoin. This management was successful in halting the seizures, confirming the diagnosis of refractory status epilepticus.

The Infectious Diseases Society of America (IDSA) recommends starting acyclovir in all suspected cases of encephalitis while awaiting diagnostic results. Acyclovir should be administered at 10 mg/kg every 8 hours and continued for 14 days in HSV infections and 21 days in VZV infections [1,8]. In cases of autoimmune encephalitis, immunotherapy with corticosteroids, intravenous immunoglobulin (IVIG), and plasma exchange (PLEX) is recommended [9-12].

Acyclovir was administered as per IDSA guidelines, but treatment was discontinued after confirming that anti-HSV IgG and IgM were non-reactive. The diagnosis of autoimmune encephalitis was confirmed by positive anti-NMDAR antibodies, and plasma exchange was recommended for treatment. Early suspicion and initiation of immunotherapy are crucial for improving a patient's condition [10]. Intravenous fluid (IVF) administration is essential in the intensive care unit (ICU) for maintaining haemodynamic stability. A proper balance in fluid administration is crucial in critically ill patients. In this case, while the patient received 500-1000 cc of intravenous fluids every 24 h, the need for this was questioned, as fluid intake from enteral nutrition and medications was sufficient, indicating that additional IVF was unnecessary [20].

The patient showed low risk of malnutrition according to the Nutritional Risk Screening (NRS 2022) and Nutric Score, yet enteral nutrition was provided at 30 kcal/kg/day, with protein intake adjusted to 1.5 g/kg/day to address potential protein deficiency [21-23]. Despite meeting the caloric needs, the patient's albumin levels remained low, indicating insufficient protein intake.

Pain management is essential in critically ill patients. As this patient could not self-report pain, alternative methods such as the Critical Care Pain Observation Tool (CPOT) and Behavioural Pain Scale (BPS) were used for pain assessment. Effective pain management reduces morbidity and improves ICU outcomes [24-28]. The patient's CPOT and Richmond Agitation Sedation Scale (RASS) scores indicated oversedation, likely due to the sedatives and analgesics used to prevent seizure recurrence. Oversedation was managed by adjusting the sedative doses and monitoring the risk of seizures.

Venous thromboembolism (VTE) and pulmonary embolism (PE) are common complications in critically ill intensive care unit (ICU) patients. Prophylaxis with low-molecular-weight heparin (LMWH) is recommended for patients at medium risk for VTE, and mechanical thromboprophylaxis is recommended for those at high risk of bleeding [29]. Given the patient's low risk for thrombosis, as assessed by the Padua and IMPROVE VTE scoring systems, thromboprophylaxis was not administered [30]. The decision to withhold thromboprophylaxis was based on the patient's low risk of VTE and the absence of complications related to mechanical ventilation.

Stress ulcer prophylaxis is essential for ICU patients at risk of upper gastrointestinal bleeding, especially those on mechanical ventilation for over 48 hours [31-33]. Proton pump inhibitors (PPIs) are often used, although their use may increase the risk of pneumonia and Clostridium difficile infection.

The patient received omeprazole for stress ulcer prophylaxis as recommended for patients on mechanical ventilation. The use of PPI helps prevent gastrointestinal bleeding, but caution is necessary regarding the potential long-term effects, such as infections [33]. Maintaining proper glycaemic control is critical in ICU patients to prevent neuroglycopenia, which can lead to irreversible brain injury if left untreated. During treatment, the patient's blood glucose levels remained stable, suggesting adequate glycaemic control. Monitoring is essential to prevent complications, such as brain dysfunction, in critically ill patients.

Antibiotic stewardship is essential for optimising antibiotic use in the ICU to prevent resistance and ensure appropriate therapy. This patient did not show any signs of infection despite positive sputum cultures for Acinetobacter baumannii and Klebsiella pneumoniae, and no additional antibiotics were administered [31]. The patient's clinical condition remained stable, and no new antibiotic treatment was initiated, despite the presence of resistant organisms in the sputum culture. This highlights the importance of careful antibiotic stewardship for critically ill patients.

CONCLUSION

Encephalitis, primarily caused by viral infections and increasingly linked to autoimmune responses, is characterised by altered consciousness, fever, seizures, and focal neurological deficits. Diagnosis relies on cerebrospinal fluid (CSF) analysis, imaging, and electroencephalography (EEG). Acute management includes initiating acyclovir while awaiting results, and considering empirical treatment based on clinical findings. For suspected autoimmune encephalitis, first-line immunotherapy involves corticosteroids, IVIG, and plasma exchange. Additionally, fluid and nutritional management, analgesia, sedation, thrombosis prophylaxis, glycaemic control, stress ulcer prevention, and appropriate antibiotic use are critical components for the successful treatment of encephalitis, particularly in ICU settings.

DECLARATIONS

None

CONSENT FOR PUBLICATION

The Authors agree to be published in the Journal of the Society of Medicine.

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AUTHORS' CONTRIBUTIONS
All authors contributed to the study, including data analysis, drafting, and review of the article. They approved the final version and were accountable for all the aspects.

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