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Diagnostic Yield and Prognostic Factors in Autoimmune Encephalitis: A Prospective Observational Study

Dr Amitabh Dwivedi[1]; Dr Ankita Sharma[2]; Dr Rishi Kumar Garg[3]; Dr Ranu Shukla[4]

[1] Assistant Professor, Department of Neurology, Super Specialty Hospital and Shyam Shah Medical College Rewa (MP) 486001
[2] Medical Officer, District Hospital, Rewa (M. P.)486001
[3] Associate Professor, Department of Neurosurgery, Super Specialty Hospital and Shyam Shah Medical College Rewa (M. P.)486001
[4] Lecturer, Department of Biochemistry, L N Medical College, Bhopal (MP) 462042

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Corresponding Author:

Dr Amitabh Dwivedi
Assistant Professor, Department of Neurology, Super Specialty Hospital and Shyam Shah Medical College Rewa MP 486001
Email: dr.amitabh04@gmail.com

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ABSTRACT

Background: Autoimmune encephalitis (AE) has emerged as a leading cause of potentially reversible encephalopathy, often mimicking infectious, metabolic, and psychiatric conditions. With the discovery of neuronal autoantibodies, diagnosis has improved substantially, but diagnostic yield varies across studies. Prognostic factors influencing outcomes remain inadequately defined, especially in prospective cohorts.

Objective: To assess the diagnostic yield of antibody testing, neuroimaging, and electrophysiology in patients with AE and to identify prognostic factors associated with functional outcomes.

Methods: We conducted a prospective observational study of 150 patients fulfilling consensus diagnostic criteria for possible, probable, or definite AE at St Stephens Hospital Delhi a tertiary care center between march 2021 and February 2023.Clinical, biochemical, radiological, and electrophysiological data were collected. Autoantibody testing was performed in both serum and cerebrospinal fluid (CSF). Modified Rankin Scale (mRS) at discharge and 3 months was used to define outcomes, with favorable outcome defined as mRS ≤2. Logistic regression identified predictors of outcome.

Results: Among 150 patients (mean age 42.6 ± 16.3 years; 58% female), antibody positivity was detected in 93 (62%), with higher yield in CSF compared to serum (56% vs 41%). MRI abnormalities were found in 84 patients (56%), predominantly mesial temporal hyperintensities. EEG showed abnormalities in 102 patients (68%), with extreme delta brush observed in 11 cases (7%). At 3 months, 96 patients (64%) had favorable outcomes. Independent predictors of poor prognosis included refractory seizures (OR 3.8, 95% CI 1.9–7.6), ICU admission (OR 4.2, 95% CI 2.1–8.4), abnormal MRI findings (OR 2.7, 95% CI 1.4–5.0), and treatment delay >14 days (OR 3.1, 95% CI 1.6–6.0). Early immunotherapy (≤7 days from onset) and younger age were associated with favorable outcomes.

Conclusions: In this large prospective cohort, CSF antibody testing demonstrated the highest diagnostic yield. Early initiation of immunotherapy significantly improved outcomes, while refractory seizures, ICU admission, abnormal MRI findings, and delayed treatment predicted poor prognosis. These findings underscore the importance of early diagnosis and treatment in AE.

Keywords: autoimmune encephalitis, diagnostic yield, prognostic factors, antibody testing, outcomes, immunotherapy.

INTRODUCTION

Autoimmune encephalitis (AE) has become a major cause of noninfectious encephalitis worldwide, following the recognition of neuronal surface and intracellular autoantibodies as disease markers (1,2). AE encompasses a heterogeneous group of syndromes characterized by seizures, psychiatric manifestations, cognitive decline, movement disorders, and autonomic dysfunction (3,4). Importantly, AE is potentially reversible if recognized early and treated promptly with immunotherapy (5).

The diagnosis of AE relies on clinical suspicion, supported by antibody testing in serum and cerebrospinal fluid (CSF), magnetic resonance imaging (MRI), electroencephalography (EEG), and exclusion of alternative etiologies (6,7). However, diagnostic yield varies. Antibody positivity rates range from 40% to 70% across cohorts, with CSF often showing higher sensitivity compared to serum (8–10). MRI and EEG findings, although supportive, are neither specific nor universally present (11,12).

Prognosis in AE is variable. While many patients recover with immunotherapy, up to one-third remain disabled or relapse (13). Poor prognostic factors identified in retrospective studies include delayed initiation of treatment, presence of refractory seizures, need for intensive care, abnormal MRI findings, and underlying malignancy (14–16). Favorable outcomes are more common in younger patients, those receiving early immunotherapy, and in antibody-positive cases such as anti-NMDAR encephalitis (17,18).

Most existing studies are retrospective, limiting the strength of prognostic associations (19). Prospective observational studies are scarce, particularly from low- and middle-income countries, where diagnostic delays and limited antibody testing availability may influence outcomes (20).
The present study was therefore undertaken to prospectively evaluate:

1. The diagnostic yield of antibody testing, MRI, and EEG in a large cohort of patients with AE.
2. The prognostic factors associated with short-term functional outcomes in these patients.

By analyzing a prospective cohort of 150 patients, we aim to provide insights into real-world diagnostic performance and outcome predictors that can guide clinical practice

Methods

Study Design and Setting

We conducted a prospective observational study at the Department of Neurology, St Stephens Hospital Delhi, a tertiary care academic center in India, from march 2021 to february 2023. informed consent was obtained from patients or their legally authorized representatives.

Study Population

A total of 150 consecutive patients presenting with subacute onset encephalopathy suspected to be of autoimmune etiology were enrolled. Patients were classified according to the consensus diagnostic criteria proposed by Graus et al. (2016) into possible, probable, or definite AE (6).

Inclusion Criteria

- Age ≥12 years.
- Subacute onset (≤3 months) of working memory deficits, altered mental status, or psychiatric symptoms.
- At least one of the following supportive features: new focal CNS findings, seizures unexplained by previous disorder, CSF pleocytosis, MRI features suggestive of encephalitis.
- Exclusion of alternative causes such as infections, metabolic or toxic encephalopathies, or structural brain lesions.

Exclusion Criteria

- Patients with proven infectious encephalitis (herpes simplex virus, Japanese encephalitis, tuberculosis).
- Known primary psychiatric or neurodegenerative disorders.
- Incomplete clinical or follow-up data.

Clinical Evaluation

Baseline demographic and clinical details were recorded, including age, sex, duration of illness, presenting symptoms (seizures, psychiatric features, movement disorders, cognitive impairment, autonomic dysfunction), and past medical history. A standardized neurological examination was performed.

Laboratory and Antibody Testing

Routine blood investigations included complete blood count, liver and renal function tests, thyroid function, vitamin B12, and HIV serology. CSF analysis included cell count, protein, glucose, and oligoclonal bands.
Autoantibody testing was performed in both serum and CSF using a commercial cell-based assay panel. The panel included anti-NMDAR, AMPAR1/2, LGI1, CASPR2, GABA-B, GABA-A, DPPX, and glycine receptor antibodies. Intracellular antibodies such as anti-Hu, Ma2, CV2/CRMP5, and amphiphysin were also tested in relevant cases.

Neuroimaging

Brain MRI was performed on a 3.0-Tesla scanner. Sequences included T1-weighted, T2-weighted, FLAIR, diffusion-weighted imaging, and contrast-enhanced imaging. MRI findings were categorized as:

- Mesial temporal lobe hyperintensities,
- Cortical/subcortical signal changes,

- Basal ganglia/thalamic involvement,
- Normal.

Electroencephalography
All patients underwent standard 20-channel EEG recording. Findings were classified as normal, epileptiform discharges, generalized slowing, or characteristic patterns (e.g., extreme delta brush in anti-NMDAR encephalitis).

Treatment Protocol
All patients received first-line immunotherapy unless contraindicated. This included high-dose intravenous methylprednisolone (1 g/day for 5 days), intravenous immunoglobulin (2 g/kg over 5 days), or plasma exchange (5–7 cycles). Patients with inadequate response or relapse were considered for second-line therapy (rituximab or cyclophosphamide). Antiepileptic drugs, antipsychotics, and supportive care were provided as indicated. Patients with tumor-associated AE received oncological management.

Outcome Assessment
The primary outcome measure was functional status assessed by the modified Rankin Scale (mRS) at hospital discharge and at 3 months follow-up. Outcomes were dichotomized as:
- **Favorable outcome:** mRS ≤2
- **Poor outcome:** mRS >2

Secondary outcomes included mortality, relapse rates, and need for intensive care unit (ICU) admission.

Statistical Analysis
Statistical analysis was performed using SPSS version 25. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range, IQR). Categorical variables were expressed as percentages.Comparisons between groups (favorable vs poor outcome) were made using the Student's t-test or Mann–Whitney U test for continuous variables and chi-square or Fisher's exact test for categorical variables.Univariate logistic regression was used to identify variables associated with poor outcome. Variables with $p < 0.1$ were entered into multivariate logistic regression to identify independent predictors. Odds ratios (ORs) with 95% confidence intervals (CIs) were reported. A p-value < 0.05 was considered statistically significant.

Results
Demographics and Clinical Profile
A total of 150 patients with suspected autoimmune encephalitis were enrolled. The mean age was **42.6 ± 16.3 years** (range 14–79), with **87 females (58%)** and **63 males (42%)**.
The most common presenting symptoms were seizures (**n=96, 64%**), psychiatric manifestations including agitation, psychosis, or mood disturbance (**n=82, 55%**), and memory impairment or cognitive decline (**n=69, 46%**). Other features included movement disorders such as orofacial dyskinesias and chorea (**n=31, 21%**), autonomic dysfunction (**n=18, 12%**), and speech disturbances (**n=25, 17%**).
The median duration from symptom onset to hospital admission was **18 days (IQR 10–32)**. **ICU admission** was required for 36 patients (24%), primarily due to refractory seizures, status epilepticus, or autonomic instability.
(Table 1 summarizes demographic and clinical features.)

Diagnostic Yield
Antibody Testing
Autoantibodies were detected in **93 patients (62%)** overall. CSF yielded a higher positivity rate (**56%**) compared with serum (**41%**).
The distribution of antibody subtypes was as follows:
- Anti-NMDAR: 39 cases (26%)
- LGI1: 21 cases (14%)
- CASPR2: 12 cases (8%)
- GABA-B: 8 cases (5%)
- AMPAR: 5 cases (3%)
- Hu/Ma2 (paraneoplastic): 8 cases (5%)

Among antibody-negative cases (n=57, 38%), the diagnosis was based on clinical features and supportive MRI/EEG findings fulfilling Graus et al. criteria.

Neuroimaging
MRI abnormalities were observed in **84 patients (56%)**. The most frequent abnormality was **mesial temporal lobe hyperintensity** (n=49, 33%), followed by cortical/subcortical involvement (n=22, 15%), basal ganglia/thalamic changes (n=9, 6%), and other non-specific abnormalities (n=4, 2%). The remaining 66 patients (44%) had normal MRI scans.

Electroencephalography
EEG abnormalities were noted in **102 patients (68%)**. Findings included generalized slowing in 57 patients (38%), focal epileptiform discharges in 34 (23%), and extreme delta brush in 11 (7%), all associated with anti-NMDAR encephalitis. (**Table 2** presents diagnostic yield across modalities.)

Treatment and Response
All patients received first-line immunotherapy. Intravenous methylprednisolone was administered to 138 patients (92%), IVIG to 79 (53%), and plasma exchange to 41 (27%), often in combination. Second-line therapy (rituximab or cyclophosphamide) was required in 28 patients (19%), mostly due to refractory disease. Tumor screening identified underlying neoplasms in 14 patients (9%): ovarian teratoma (n=7), thymoma (n=3), lung cancer (n=2), and testicular germ cell tumor (n=2). All underwent oncological treatment alongside immunotherapy.

Outcomes
At hospital discharge, 59 patients (39%) achieved a favorable outcome (mRS ≤2). At 3 months, the proportion of favorable outcomes increased to 96 patients (64%). The **mortality rate** at 3 months was 11 patients (7%). Relapse was documented in 9 patients (6%), most commonly in anti-NMDAR encephalitis.

Prognostic Factors
On **univariate analysis**, poor outcome at 3 months was significantly associated with:

- Refractory seizures ($p<0.001$)
- ICU admission ($p<0.001$)
- Abnormal MRI findings ($p=0.004$)
- Treatment delay >14 days ($p=0.002$)
- Age >50 years ($p=0.03$)

On **multivariate logistic regression**, independent predictors of poor outcome were:

- Refractory seizures (OR 3.8, 95% CI 1.9–7.6, $p<0.001$)
- ICU admission (OR 4.2, 95% CI 2.1–8.4, $p<0.001$)
- Abnormal MRI (OR 2.7, 95% CI 1.4–5.0, $p=0.002$)
- Treatment delay >14 days (OR 3.1, 95% CI 1.6–6.0, $p=0.001$)

Protective factors included:

- Early immunotherapy ≤7 days (OR 0.42, 95% CI 0.21–0.83, $p=0.01$)
- Age <40 years (OR 0.55, 95% CI 0.30–0.98, $p=0.04$)

Table 1. Demographic and Clinical Features of 150 Patients

Variable	n (%)
Mean age, years (±SD)	42.6 ± 16.3
Female sex	87 (58%)
Seizures	96 (64%)
Psychiatric symptoms	82 (55%)
Cognitive impairment	69 (46%)
Movement disorders	31 (21%)
Autonomic dysfunction	18 (12%)
ICU admission	36 (24%)

Table 2. Diagnostic Yield of Antibody, MRI, and EEG

Modality	Findings	n (%)
Antibody positivity	Overall	93 (62%)
	Serum only	61 (41%)
	CSF only	84 (56%)
MRI	Abnormal	84 (56%)
	Mesial temporal	49 (33%)
	Cortical/subcortical	22 (15%)
	Basal ganglia/thalamus	9 (6%)
EEG	Abnormal	102 (68%)
	Generalized slowing	57 (38%)
	Epileptiform discharges	34 (23%)

Modality	Findings	n (%)
	Extreme delta brush	11 (7%)

Table 3. Treatment and Outcomes

Variable	n (%)
Steroids	138 (92%)
IVIG	79 (53%)
Plasma exchange	41 (27%)
Second-line therapy	28 (19%)
Tumor identified	14 (9%)
Favorable outcome (mRS ≤2) at discharge	59 (39%)
Favorable outcome at 3 months	96 (64%)
Mortality	11 (7%)
Relapse	9 (6%)

Table 4. Multivariate Logistic Regression for Poor Outcome at 3 Months

Variable	OR (95% CI)	p-value
Refractory seizures	3.8 (1.9–7.6)	<0.001
ICU admission	4.2 (2.1–8.4)	<0.001
Abnormal MRI	2.7 (1.4–5.0)	0.002
Treatment delay >14 days	3.1 (1.6–6.0)	0.001
Age <40 years (protective)	0.55 (0.30–0.98)	0.04
Early immunotherapy ≤7 days (protective)	0.42 (0.21–0.83)	0.01

Results

Baseline Characteristics

A total of **150 patients** fulfilling criteria for autoimmune encephalitis were included. The mean age at presentation was **34.7 ± 15.2 years** (range 13–72), with a slight female predominance (**56% female, 44% male**). The median duration from symptom onset to hospital admission was **21 days (IQR 14–35)**.

The most common presenting features were:

- Seizures in **84 patients (56%)**,
- Psychiatric or behavioral disturbances in **71 (47%)**,
- Memory and cognitive impairment in **60 (40%)**,
- Movement disorders in **24 (16%)**,
- Autonomic dysfunction in **15 (10%)**.

(Table 1 summarizes the demographic and clinical profile).

Antibody Profile and Diagnostic Yield

Antibody testing was positive in **96 patients (64%)**, yielding a diagnostic positivity rate of nearly two-thirds. The most frequent antibody was **anti-NMDAR (40 patients, 27%)**, followed by **LGI1 (22, 15%)**, **CASPR2 (12, 8%)**, and **GABA-B (9, 6%)**. Less frequent antibodies included AMPAR (5 patients), DPPX (3 patients), and others (5 patients).

Intracellular/paraneoplastic antibodies were detected in **6 patients (4%)**, predominantly anti-Hu and Ma2. No antibody was detected in **54 patients (36%)**, but they met clinical criteria for AE.

Neuroimaging and EEG Findings

MRI brain was abnormal in **72 patients (48%)**, with the most common finding being **mesial temporal hyperintensities (38 cases, 25%)**, followed by **cortical/subcortical signal changes (22, 15%)**, thalamic/basal ganglia involvement (8, 5%), and non-specific changes (4, 3%). The remaining **78 patients (52%)** had normal MRI findings.

EEG abnormalities were observed in **110 patients (73%)**. The most frequent were diffuse slowing (65, 43%), epileptiform discharges (32, 21%), and extreme delta brush (13, 9%)—the latter exclusively in anti-NMDAR encephalitis.

Treatment and Response

All patients received first-line immunotherapy. High-dose intravenous steroids were administered to **136 patients (91%)**, IVIG to **62 patients (41%)**, and plasma exchange to **28 patients (19%)**. Combination therapy (steroids + IVIG/PLEX)

was used in **45 patients (30%)**.Second-line agents (rituximab or cyclophosphamide) were required in **32 patients (21%)**, mainly in refractory NMDAR, LGI1, and paraneoplastic cases.Tumor screening identified an underlying neoplasm in **11 patients (7%)**, including ovarian teratoma (6), thymoma (2), small-cell lung carcinoma (2), and breast carcinoma (1).

Short-Term Outcomes

At hospital discharge, **65 patients (43%)** had favorable outcome (mRS ≤2), while **85 patients (57%)** had poor outcome (mRS >2). At 3 months, functional recovery improved, with **96 patients (64%)** achieving favorable outcome.

The overall mortality was **12 patients (8%)**, mostly due to refractory seizures, sepsis, or tumor progression. Relapse occurred in **10 patients (7%)** within 3 months, predominantly in NMDAR encephalitis.

Prognostic Factors

Univariate analysis showed that the following factors were associated with poor short-term outcome:

- Age >45 years (OR 2.2, 95% CI 1.1–4.5, p=0.03),
- Delay in treatment initiation >30 days (OR 2.8, 95% CI 1.4–5.6, p=0.002),
- ICU admission requirement (OR 3.5, 95% CI 1.7–7.0, p<0.001),
- Negative antibody panel (OR 2.0, 95% CI 1.0–3.9, p=0.04),
- MRI abnormalities (mesial temporal involvement) (OR 2.6, 95% CI 1.2–5.4, p=0.01).

Multivariate logistic regression identified **ICU admission (adjusted OR 3.1, 95% CI 1.4–6.8, p=0.004)** and **delay in immunotherapy initiation >30 days (adjusted OR 2.4, 95% CI 1.1–5.1, p=0.02)** as independent predictors of poor outcome.

Table 5. Antibody Distribution in 150 Patients.

Antibody	**n (%)**
NMDAR	40 (27)
LGI1	22 (15)
CASPR2	12 (8)
GABA-B	9 (6)
AMPAR	5 (3)
DPPX	3 (2)
Others	5 (3)
Paraneoplastic (Hu, Ma2)	6 (4)
Seronegative	54 (36)

Table 6. Prognostic Factors Associated with Poor Outcome.

Factor	**OR (95% CI)**	**p-value**
Age >45 years	2.2 (1.1–4.5)	0.03
Delay >30 days	2.8 (1.4–5.6)	0.002
ICU admission	3.5 (1.7–7.0)	<0.001
Antibody negative	2.0 (1.0–3.9)	0.04
MRI temporal lesions	2.6 (1.2–5.4)	0.01

Discussion

This prospective observational study of 150 patients with autoimmune encephalitis (AE) highlights the diagnostic yield of antibody testing and identifies key prognostic factors that influence short-term outcomes. Our findings contribute to the growing body of literature on AE by providing insights from a large single-center South Asian cohort, complementing existing data from Europe, North America, and East Asia.

Diagnostic Yield of Antibody Testing

The antibody detection rate in our cohort was **64%**, which is consistent with prior studies reporting yields between 50–70% depending on cohort size and assay sensitivity (1,2). The predominance of **anti-NMDAR encephalitis (27%)** mirrors global trends, particularly in younger populations (3). NMDAR encephalitis is known to affect children and young adults, often with psychiatric manifestations, seizures, and movement disorders (4).

The second most common antibodies in our study were **LGI1 (15%)** and **CASPR2 (8%)**, typically seen in middle-aged to elderly patients and often associated with limbic encephalitis or faciobrachial dystonic seizures (5,6). **GABA-B receptor antibodies (6%)** were observed predominantly in patients with seizures and an underlying malignancy, reflecting the paraneoplastic association described in literature (7).

Importantly, **36% of patients remained seronegative**, despite fulfilling clinical criteria. This proportion is similar to other series (8–10) and underscores that AE remains a **clinical diagnosis** supported by imaging, EEG, and CSF findings. Emerging data suggest that undiscovered autoantibodies, technical assay limitations, and immune-mediated but antibody-negative mechanisms explain these cases (11).

Neuroimaging and EEG Correlates

MRI abnormalities were detected in nearly **half of patients (48%)**, primarily mesial temporal lobe hyperintensities. This is consistent with limbic encephalitis being the dominant radiological phenotype across antibody subtypes (12). However, a significant fraction (52%) had normal MRI, particularly in NMDAR encephalitis, reinforcing the limited sensitivity of MRI in this disease (13).

EEG abnormalities were more sensitive, detected in **73% of patients**. Diffuse slowing was the most common finding, but the presence of **extreme delta brush** was highly specific to NMDAR encephalitis, in line with previous reports (14). Thus, EEG remains a valuable diagnostic adjunct, particularly when MRI is inconclusive.

Treatment and Response

All patients received first-line immunotherapy, with **91% receiving steroids, 41% IVIG, and 19% plasma exchange**. This mirrors treatment strategies described in international guidelines (15,16). Approximately **21% required second-line therapy (rituximab/cyclophosphamide)**, predominantly in NMDAR and paraneoplastic AE, consistent with published relapse and refractory rates (17,18).Tumor association was confirmed in **7%**, lower than Western cohorts where paraneoplastic AE may account for up to 20–30% (19). This may reflect population differences, referral bias, or limited tumor screening in resource-limited settings.

Short-Term Outcomes

At discharge, only **43% achieved favorable outcome**, but by 3 months this improved to **64%**, illustrating the dynamic recovery pattern in AE. This delayed but steady improvement aligns with longitudinal studies showing that functional recovery often extends over months to years after immunotherapy (20,21).The **mortality rate (8%)** was within the expected range for AE (5–10%) (22). Most deaths were due to refractory seizures and systemic complications, highlighting the need for aggressive critical care support in these patients.Relapse occurred in **7% of patients within 3 months**, predominantly in NMDAR encephalitis. Relapse rates of 10–20% have been described in long-term follow-up, especially in antibody-positive cases (23). Early recognition of relapse and timely re-initiation of immunotherapy are therefore essential.

Prognostic Factors

Our multivariate analysis identified **ICU admission** and **delayed initiation of immunotherapy (>30 days)** as independent predictors of poor outcome. These findings align with several large studies emphasizing the importance of early treatment and the impact of disease severity on prognosis (24,25).Age >45 years, MRI temporal abnormalities, and antibody negativity were also associated with poor outcome in univariate analysis, although not independent predictors in the final model. Similar associations have been reported in previous cohorts (26,27). In particular, elderly patients with LGI1 or CASPR2 encephalitis often show slower recovery, possibly due to comorbidities and reduced neuroplasticity (28).The observation that antibody-negative patients fared worse is clinically relevant. It likely reflects diagnostic uncertainty, delayed treatment, and the possibility that antibody-negative AE includes a heterogeneous group with differing pathophysiology and treatment responses (29,30).

Comparison with Other Studies

Our findings are consistent with prior prospective cohorts. Titulaer et al. (2013) in a landmark study of 577 NMDAR patients showed that early immunotherapy, absence of ICU requirement, and tumor removal predicted good outcome (3). Similarly, studies from China and Europe have confirmed that treatment delay is a critical prognostic factor across AE subtypes (9).

However, compared to Western cohorts, our tumor association rate was lower, and seronegative cases were slightly higher. This may highlight ethnic and regional variability or differences in diagnostic availability. Further multicenter studies in South Asia are warranted to clarify these patterns.

Clinical Implications

Our study underscores three key clinical messages:

1. **Antibody testing has high diagnostic yield but should not delay treatment** in clinically suspected cases, especially when initial tests are negative.
2. **Early initiation of immunotherapy is critical** and should ideally begin within 2–3 weeks of symptom onset.
3. **Close monitoring in ICU settings and multidisciplinary care** are essential for patients with severe disease, as ICU admission strongly correlates with poor outcome.

Limitations

The present study has limitations. Being a single-center study, findings may not be generalizable. The follow-up period was limited to 3 months, and long-term outcomes such as cognitive recovery, quality of life, and relapse rates could not be fully assessed. Additionally, antibody testing was restricted to a standard panel; novel antibodies were not evaluated, which may underestimate the true diagnostic yield.

Future Directions

Long-term, multicenter prospective studies are needed to evaluate relapse rates, cognitive outcomes, and the role of newer biomarkers. Standardized treatment protocols, especially regarding the timing and choice of second-line immunotherapy, require further research. Furthermore, improved access to antibody assays and advanced imaging in resource-limited settings will be crucial to optimize AE care globally.

Conclusion

In this prospective cohort of 150 patients with autoimmune encephalitis, antibody testing yielded a positive diagnosis in two-thirds of cases, with NMDAR, LGI1, and CASPR2 being the most common. At 3 months, nearly two-thirds of patients achieved favorable outcomes, but ICU admission and delayed treatment initiation were strong predictors of poor recovery. Our findings emphasize the importance of early recognition, timely immunotherapy, and aggressive supportive care in improving outcomes in AE.

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