Clinical Features, Antibody Profiles, and Prognostic Factors in Autoimmune Encephalitis: A Single-Center Study

Bedriye Karaman , Gülcan Neşem Baskan , Merve Yavuz , Ayşe Güler , Özgül Ekmekci , Nur Yüceyar , Rasim Tunçel *

Posted Date: 31 July 2025

doi: 10.20944/preprints202507.2641.v1

Keywords: autoimmune encephalitis; limbic encephalitis; prognostic factors

 doi:10.20944/preprints202507.2641.v1

Article

Clinical Features, Antibody Profiles, and Prognostic Factors in Autoimmune Encephalitis: A Single-Center Study

Bedriye Karaman [1], Gülcan Neşem Baskan [2], Merve Yavuz [1], Ayşe Güler [1], Özgül Ekmekci [1], Nur Yüceyar [1] and Rasim Tunçel [1,*]

[1] Ege University Faculty of Medicine, Neurology Department

[2] Izmir Bozyaka Training and Research Hospital, Neurology Department

* Correspondence: rasimtuncel@gmail.com

Abstract

Background/Objectives: Autoimmune encephalitis (AEI) is a heterogeneous group of inflammatory central nervous system (CNS) disorders characterized by variable clinical presentations and antibody profiles. This study aimed to identify poor prognostic factors in AEI by retrospectively evaluating patients diagnosed based on clinical, radiological, and serological findings. **Methods**: Forty-four patients diagnosed with AIE between 2014 and 2024 were included. Demographic, clinical, radiological, and serological data were collected retrospectively. Patients were grouped based on antibody localization (intracellular, surface and seronegative) and classified by treatment response. Poor prognosis was defined as lack of objective clinical improvement to treatment or death. **Results**: The mean age was 57.8 ± 13.6 years, with a female-to-male ratio of approximately 1:1. Limbic encephalitis (LE) was the most common clinical presentation (43.2%). Malignancy was detected in 33.3% of patients, most frequently in those with SOX1 (83.3%), anti-Hu (60.0%), anti-Yo (50.0%) antibodies. Anti-SOX1 positivity was significantly associated with both malignancy (OR=27.5, p=0.007) and mortality (OR=13.2, p=0.009), while anti-LGI1 positivity correlated with the absence of malignancy (p=0.036). Patients with LE showed significantly better treatment responses (OR=14.0, p=0.019). Mortality was 20.1% overall, highest among anti-SOX1 positive patients (66.7%). Presence of multiple antibodies was associated with trend toward higher mortality and poorer prognosis, although not statistically significantly. **Conclusions**: Anti-SOX1 positivity is a key poor prognosis indicator in AEI, strongly associated with both malignancy and mortality. In contrast, LE presentation was linked to better treatment response. Antibody profile, clinical features, and malignancy screening are critical for risk stratification and guiding management in AEI.

Keywords: autoimmune encephalitis; limbic encephalitis; prognostic factors

1. Introduction

Autoimmune encephalitis (AIE) is an inflammatory condition resulting from the immune system attacking components of the central nervous system (CNS). The classification of AIE is various due to clinical findings, underlying antibodies, and overlapping encephalitis types [1]. AIE has been shown to have a prevalence comparable to infectious encephalitis, with an incidence of 0.2–0.8 per 100,000 person-years [2]. Although misdiagnosis persists, AIE is increasingly regarded in the differential diagnosis of individuals exhibiting subacute cognitive decline, psychiatric symptoms, movement disorders, and seizures. The rise in AIE diagnosis is attributable to the more widespread use of antibody testing and the growing awareness among clinicians [3]. In recent years, with the increase in clinical studies and the publication of international guidelines, the chance for earlier diagnosis and treatment are improving [4]. Two potential triggers of AIE include tumors and viral encephalitis. Some involved cancers contain nerve tissue or express neuronal proteins targeted by

 doi:10.20944/preprints202507.2641.v1

autoantibodies, suggesting that ectopic expression may initiate the autoimmune response. Antibodies against the NMDAR and other neuronal cell-surface proteins may cause recurrent neurologic symptoms weeks after herpes simplex encephalitis and perhaps other viral encephalitis [5]. Cerebrospinal fluid (CSF) examination, brain magnetic resonance imaging (MRI), electroencephalography, and panel-based neural antibody testing are recommended for all patients suspected of having AIE [6].

Here, we aimed to identify prognostic factors in AIE by retrospectively evaluating forty-four patients diagnosed with AIE based on clinical, serological, and radiological features.

2. Materials and Methods

Fifty patients who were investigated for possible AIE between 2014 and 2024 were included in the study. The patients' demographic data, as well as their clinical, radiological, electrophysiological and serological characteristics, were obtained retrospectively from electronic medical records. All patients underwent brain MRI, and the findings were classified as limbic pattern, striatal pattern, perivascular enhancement, diencephalic/brainstem involvement, cortical pattern and MRI-negative, according to the involvement patterns described by Sanvito et al [7]. Diagnostic criteria for possible AIE were described as fullfillment of following three critera: 1. subacute (rapid progression of less than three months) onset of working memory deficits, altered mental status, or psychiatric symptoms 2. New focal CNS findings, or seizures not explained by previously known seizure disorder, or CSF pleocytosis (white blood cell count of more than five cells per mm^3), or MRI features suggestive of encephalitis 3. Reasonable exclusion of alternative causes [8] Antibody-negative but probable AIE in our cohort met all these criteria: a) Subacute working memory deficits, altered mental status, or psychiatric symptoms; b) Exclusion of well-defined autoimmune encephalitis syndromes; c) Absence of well-characterized autoantibodies in serum and CSF, with at least two features: MRI abnormalities, CSF pleocytosis or oligoclonal bands/elevated IgG index, or inflammatory infiltrates in brain biopsy [9]. Six patients were excluded for failing to meet the diagnostic criteria.

Patients were classified into four groups according to their treatment response: death, no objective response, partial objective response (defined as improvement in less than 50% of clinical findings), and good clinical response (defined as improvement in more than 50% of clinical findings). Poor prognosis was defined as the absence of an objective clinical response to treatment or death.

The data were analyzed using SPSS version 25. Descriptive statistics and frequency distributions were calculated initially. For categorized data, Chi-square test or *Likelihood Ratio* (LR) test was applied as appropriate. Non-parametric correlation analysis was performed, and based on the results, logistic regression analysis was conducted. For the group of 29 patients with intracellular antibodies, the same analyses—Chi-square or *LR* test, non-parametric correlation, and logistic regression modeling—were repeated. Statistical analysis could not be conducted separately for the extracellular antibody group and the seronegative group due to insufficient sample sizes. $P \leq 0.05$ was considered statistically significant.

3. Results

The mean age of the 44 patients included in the study was 57.8 ± 13.60 years (range: 21–83), and the proportion of female patients was 47.7% (n=21). The average duration from symptom onset to the first medical consultation was 38.5 ± 66.83 weeks (range: 1–312). The time from first medical consultation to definitive diagnosis was found to be 8.7 ± 38.86 weeks (range: 1–260). The patients were divided into three groups based on the localization of the detected antibody: intracellular, surface, and seronegative. Descriptive data of the groups are presented in Table 1.

 doi:10.20944/preprints202507.2641.v1

Table 1. Descriptives of the entire cohort and groups according to the antibody localization.

	Total (N=44)		*İntracellular Ab (N=29)*		*Surface Ab (N=11)*		*Seronegative (N=4)*		*K-W*
	Mean/Median	*±SD/IQR*	*Mean/Median*	*±SD/IQR*	*Mean/Median*	*±SD/IQR*	*Mean/Median*	*±SD/IQR*	*p*
Onset Age	57.82	±13.60	60.14	±9.60	49.36	±20.64	64.25	±1.50	0.27
Symptom to Diagnosis in weeks	8.00	16.00	8.00	52.00	4.00	23.00	7.00	40.00	0.62
CSF Cell	4.00	5.00	4.00	4.00	15.00	19.00	1.50	7.00	0.40
CSF Proteine	45.81	±23.28	41.90	±17.63	55.86	±34.91	48.75	±27.17	0.96
	N	*%*	*N*	*%*	*N*	*%*	*N*	*%*	X^2
Female	21	47.7	14	48.3	5	45.5	2	50	0.983
Malignancy	14	31.8	12	41.4	1	9.1	1	25	0.156
Mortality	9	20.5	8	27.6	1	9.1	0	0	0.246
Good response to treatment	8	18.2	3	10.3	4	36.4	1	25	0.152
LE	19	43.2	7	24.1	10	90.9	2	50	**0.001**
PSS	18	40.9	17	58.6	1	9.1	1	25	**0.003**
PP	3	6.8	3	10.3					0.435
OMS	2	4.5					1	25	0.056
SPS	2	4.5	2	6.9					0.582

Ab: antibody, K-W: Kruskal-Wallis, X^2: Chi-square, SD: Standard deviation, IQR: Interquartile range, CSF: Cerebrospinal fluid, LE: Limbic encephalitis, PSS: Paraneoplastic cerebellar syndrome, SPS: Stiff person syndrome.

Among the specific antibodies, the most frequently detected were anti-Yo (n=10, 22.7%), anti-SOX1 (n=6, 13.6%), anti-Hu (n=5, 11.4%), anti-NMDAR (n=5, 11.4%), anti-LGI1 (n=5, 11.4%), anti-amphiphysin (n=3, 6.8%), anti-GAD (n=3, 6.8%), and anti-titin (n=3, 6.8%). No specific antibodies were identified in four patients. Other antibodies were detected in only one patient each. The most common co-occurring antibody was anti-titin, observed in 4.5% of patients.

Nineteen patients (43.2%) were diagnosed with limbic encephalitis (LE), 18 (40.9%) with paraneoplastic cerebellar syndrome (PCS), 3 (6.8%) with paraneoplastic polyneuropathy (PP), 2 (4.5%) with opsoclonus-myoclonus syndrome (OMS), and 2 (4.5%) with stiff-person syndrome (SPS) as clinical presentations.

Radiological evaluation revealed that patients with a limbic pattern on MRI did not exhibit malignancy, and this finding was statistically significant (p=0.036). Moreover, a correlation was observed between limbic pattern on MRI a good treatment response ($p = 0.030$, $r = 0.328$; Spearman correlation). In contrast, no significant association was found between prognosis and either a normal MRI or other involvement patterns.

Malignancy screening was performed in 95.5% (n=42) of the patients. Among these 42 patients, the malignancy rate was 33.3% (n=14). According to the clinical presentation, the frequency of malignancy was found to be 33.3% (n=6) in patients with PCS, 27.8% (n=5) in those with LE, 66.6% (n=2) in patients with PP, and 50% (n=1) in patients with OMS. No statistically significant difference was found between the groups. When evaluated according to antibody positivity, the frequency of malignancy was 83.3% (n=5) in anti-SOX1 positive patients, 50.0% (n=5) in anti-Yo positive patients, 60.0% (n=3) in anti-Hu positive patients. Among seronegative patients the malignancy rate was 25.0% (n=1). No malignancy was detected in patients positive for anti-LGI1, anti-GAD, or anti-amphiphysin

 doi:10.20944/preprints202507.2641.v1

antibodies. When compared with other patients, the absence of malignancy in anti-LGI1 positive cases was statistically significant (LR, p=0.036).

Among the 44 patients, 9 deaths were recorded, corresponding to a mortality rate of 20.1%. The highest mortality was observed in patients with anti-SOX1 antibodies (66.7%), followed by anti-Hu (40%), anti-amphiphysin (33%), and both anti-Yo and anti-NMDAR (20%). Of the 9 deaths, 4 occurred in anti-SOX1 positive patients, 2 in anti-Yo, 2 in anti-Hu, 1 in anti-amphiphysin, and 1 in anti-NMDAR positive patients. Patients with multiple specific antibody positivities (n=6) had a higher mortality rate (50.0%) compared to those with a single antibody positivity (15.8%, n=38), but this difference was not statistically significant (p=0.077). However, anti-SOX1 positivity was significantly associated with higher mortality (p=0.007). No deaths occurred in patients with anti-LGI1, anti-GAD, or in seronegative individuals, although these findings were not statistically significant when compared to the rest of the cohort (p=0.118, p=0.232, and p=0.165, respectively). By clinical diagnosis, mortality rates were 33.3% in PP, 22.2% in PCS, and 21.1% in LE patients, with no significant differences among the groups.

When treatment responses were evaluated according to antibody positivity, patients with multiple antibody positivities and those positive for anti-SOX1 showed significantly poor prognosis (p=0.014 and p=0.003, respectively). No significant associations were found with other antibody positivities.

In the logistic regression analysis of the entire patient cohort (n=44), malignancy presence, good clinical response to treatment, and mortality were evaluated (Table 2). Mortality was found to be on average 13.2 times higher in patients positive for SOX1 compared to those without (B=2.58, p=0.009, OR=13.2, 95% CI=1.89–91.90). Good clinical response to treatment was observed to be 14 times higher in patients diagnosed with LE compared to those without (B=2.64, p=0.019, OR=14.0, 95% CI=1.54–127.23). The presence of malignancy was on average 27.5 times higher in patients positive for anti-SOX1 antibodies (B=3.31, p=0.007, OR=27.5, 95% CI=2.50–302.17) and 5.5 times higher in those positive for anti-Yo antibodies (B=1.71, p=0.041, OR=5.5, 95% CI=1.07–28.20).

Table 2. Logistic regression models.

		B	S.E.	p	Exp(B)	95% C.I.for EXP(B)	
	Model for Mortality					Lower	Upper
All cases	SOX1	2,58	0,99	0,009	13,2	1,896	91,907
	Constant	-1,887	0,48	0	0,152		
	Model for Good Response to treatment						
	LE	2,639	1,126	0,019	14	1,541	127,225
	Constant	-3,178	1,021	0,002	0,042		
	Model for Malignity						
	SOX1	3,314	1,223	0,007	27,5	2,503	302,174
	Yo	1,705	0,834	0,041	5,5	1,073	28,198
	Constant	-1,705	0,544	0,002	0,182		
İntracellular Ab positive cases	**Model for Mortality**						
	SOX1	2,251	1,026	0,028	9,5	1,272	70,964
	Constant	-1,558	0,55	0,005	0,211		
	Model for Malignity						
	SOX1	2,372	1,187	0,046	10,714	1,046	109,784
	Constant	-0,762	0,458	0,096	0,467		

S.E.: Standard error, C.I: Confidence interval, LE: Limbic encephalitis.

Among patients with antibodies targeting intracellular proteins (n=29), mortality was found to be on average 9.5 times higher in those positive for anti-SOX1 compared to those without (B=2.25, p=0.028, OR=9.5, 95% CI=1.27–70.96). Similarly, the presence of malignancy was on average 10.7 times

 doi:10.20944/preprints202507.2641.v1

higher in anti-SOX1 positive patients compared to those who were negative (B=2.37, p=0.046, OR=10.71, 95% CI=1.05–109.78).

4. Discussion

This retrospective study included 44 patients, with a mean age of 57.8 ± 13.6 years and a female-to-male ratio of approximately 1:1. Patients with AIE show distinct patterns in age and sex distribution, which vary by antibody subtype. For example, anti-NMDAR positive AIE is more common in the pediatric population or young adult females, whereas anti-LGI1 positive cases are more frequently observed in older males [10,11]. Many patients experience significant delays between symptom onset and medical evaluation or diagnosis, often due to the psychiatric or non-specific nature of early symptoms. The mean time from symptom onset to first medical consultation was 38.5 weeks, and from consultation to diagnosis was 8.7 weeks in our cohort. In a study evaluating the duration from symptom onset to antibody testing in patients with AIE, the interval was reported to be approximately 67 weeks in patients from 2007 to 2012, whereas it decreased to 10.5 weeks in those diagnosed between 2013 and 2016 [12]. Increased awareness and use of antibody testing widely have reduced diagnostic delays in recent years.

Malignancy was detected in 33.3% of patients in our cohort. The rate of malignancy varies depending on the specific antibody involved, diagnostic methods, and patients demographics, but overall, malignancy is reported in 6-25% of cases [13,14]. The highest malignancy rates were observed in patients with anti-SOX1 (83.3%), anti-Hu (60.0%), and anti-Yo (50.0%) antibodies. Multiple case reports and studies show that the presence of SOX1 antibodies in AEI is a strong predictor of underlying malignancy, especially small cell lung cancer (SCLC) [15–17]. Anti-Hu and anti-Yo antibodies are well-known associated onconeural antibodies with significant malignancy risk [18,19]. In logistic regression analyses, it was shown that anti-SOX1 positivity increased the odds of malignancy by 27.5 times (p=0.007), and anti-Yo positivity by 5.5 times (p=0.041) in all cohort. In the group of intracellular antibody positive patients, the odds of malignancy were 10.7 times higher in anti-SOX1 positive patients as well (p=0.046). There was a statistically significant association between anti-LGI1 positivity and the absence of malignancy, suggesting that patients with anti-LGI1 encephalitis were less likely to have an underlying cancer (p=0.036). This finding aligns with previous studies, which report an association with malignancy in less than 10% of anti-LGI1 encephalitis cases [20,21].

In our cohort, nine patients died during follow-up, corresponding to a mortality rate of 20.1%. Overall, the mortality rate for AIE ranges was reported from 6% to 19%, but can be much higher for certain subtypes [22]. Mortality was highest among anti-SOX1 positive patients (66.7%), followed by those with anti-Hu (40.0%) and anti-amphiphysin (33.0%) antibodies. In contrast, no deaths were observed in patients with anti-LGI1 or anti-GAD antibodies or in seronegative individuals. Mortality is reported to be highest anti-GABABR positive AIEs, whereas it is lowest in cases associated with anti-NMDAR and anti-LGI1 antibodies in previous studies [23,24]. Mortality in AEI is influenced not only by the type of antibody but also by several other factors, including age at onset, clinical presentation, presence of malignancy, and early treatment [24,25]. Anti-SOX1 positivity was significantly associated with increased mortality in this study. In univariate analysis, the association between anti-SOX1 positivity and mortality was statistically significant (p=0.007). Logistic regression analyses further supported this relationship: anti-SOX1 positivity increased the odds of mortality by 13.2 times in the entire cohort (p=0.009) and by 9.5 times among patients with intracellular antibody positivity (p=0.028). These findings suggest that anti-SOX1 antibody may serve as an important prognostic marker in AEI, potentially due to its strong association with underlying malignancy. The high mortality observed in this subgroup aligns with recent literature indicating that intracellular (onconeural) antibodies-such as anti-Hu and anti-Yo- are frequently linked to cancer and associated with poor prognosis [26]. Patients with multiple antibody positivities had higher mortality (50.0%) compared to those with a single antibody (15.8%), though the difference was not statistically significant (p=0.077). Treatment response analysis showed that patients with anti-SOX1 antibody or

 doi:10.20944/preprints202507.2641.v1

multiple antibody positivities had significantly poorer prognosis (p=0.003 and p=0.014, respectively). The presence of multiple autoantibodies is associated with more complex disease and may increase the risk of poor outcomes, but large-scale outcome data are limited [27].

In this study, limbic involvement on MRI was significantly associated with the absence of malignancy, suggesting a predominance of non-paraneoplastic AIE in these cases. Additionally, limbic patterns were positively correlated with better treatment response. In logistic regression analysis, patients with LE were 14 times more likely to show an good clinical response to treatment (p=0.019). This finding supports previous reports suggesting that AIE with predominant limbic involvement often shows a favorable response to immunotherapy. Limbic involvement may reflect a pathophysiological mechanism more amenable to immunomodulation compared to other encephalitic subtypes or paraneoplastic features [8].

5. Conclusions

This study highlights the prognostic value of antibody profile and clinical features in AIE. Anti-SOX1 positivity was strongly associated with both malignancy and mortality, while patients with LE showed better treatment responses. Early diagnosis and antibody-based risk stratification may guide clinical management and improve outcomes.

Author Contributions: Conceptualization, B.K., N.Y. and Ö.E.; methodology, B.K., A.G., G.N.B. and R.T.; software, R.T.; validation, R.T. and B.K.; formal analysis, R.T.; investigation, G.N.B., M.Y; resources, G.N.B. and M.Y.; data curation, R.T. and B.K.; writing—original draft preparation, B.K and R.T..; writing—review and editing, B.K., A.G, G.N.B., M.Y., N.Y., Ö.E. and R.T.; supervision, N.Y., Ö.E. and A.G. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethical Committee of Ege University (protocol code: n.25-3.1T/69 and date: 20.03.2025).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The anonymized data can be accessed upon request from the corresponding author.

Acknowledgments: Quillbot AI has been used for English grammer cheking and Turkish- English translate for some parts of results section. The authors have reviewed and edited the output and take full responsibility for the content of this publication.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

AEI	Autoimmune encephalitis
CNS	central nervous system
CSF	Cerebrospinal fluid
MRI	Magnetic resonance imaging
LE	Limbic encephalitis
PCS	Paraneoplastic cerebellar syndrome
PP	Paraneoplastic polyneuropathy
OMS	Opsoclonus-myoclonus syndrome
SPS	Stiff-person syndrome

References

 doi:10.20944/preprints202507.2641.v1

1. Esposito S, Principi N, Calabresi P, Rigante D: An evolving redefinition of autoimmune encephalitis. Autoimmun Rev 2019, 18(2):155-163.
2. Dubey D, Pittock SJ, Kelly CR, McKeon A, Lopez-Chiriboga AS, Lennon VA, Gadoth A, Smith CY, Bryant SC, Klein CJ et al: Autoimmune encephalitis epidemiology and a comparison to infectious encephalitis. Ann Neurol 2018, 83(1):166-177.
3. Flanagan EP, Geschwind MD, Lopez-Chiriboga AS, Blackburn KM, Turaga S, Binks S, Zitser J, Gelfand JM, Day GS, Dunham SR et al: Autoimmune Encephalitis Misdiagnosis in Adults. JAMA Neurol 2023, 80(1):30-39.
4. Hahn C, Budhram A, Alikhani K, AlOhaly N, Beecher G, Blevins G, Brooks J, Carruthers R, Comtois J, Cowan J et al: Canadian Consensus Guidelines for the Diagnosis and Treatment of Autoimmune Encephalitis in Adults. Can J Neurol Sci 2024:1-21.
5. Dalmau J, Graus F: Antibody-Mediated Encephalitis. N Engl J Med 2018, 378(9):840-851.
6. Abboud H, Probasco JC, Irani S, Ances B, Benavides DR, Bradshaw M, Christo PP, Dale RC, Fernandez-Fournier M, Flanagan EP et al: Autoimmune encephalitis: proposed best practice recommendations for diagnosis and acute management. J Neurol Neurosurg Psychiatry 2021, 92(7):757-768.
7. Sanvito F, Pichiecchio A, Paoletti M, Rebella G, Resaz M, Benedetti L, Massa F, Morbelli S, Caverzasi E, Asteggiano C et al: Autoimmune encephalitis: what the radiologist needs to know. Neuroradiology 2024, 66(5):653-675.
8. Graus F, Titulaer MJ, Balu R, Benseler S, Bien CG, Cellucci T, Cortese I, Dale RC, Gelfand JM, Geschwind M et al: A clinical approach to diagnosis of autoimmune encephalitis. Lancet Neurol 2016, 15(4):391-404.
9. Dalmau J, Graus F: Diagnostic criteria for autoimmune encephalitis: utility and pitfalls for antibody-negative disease. Lancet Neurol 2023, 22(6):529-540.
10. Kunchok A, McKeon A, Zekeridou A, Flanagan EP, Dubey D, Lennon VA, Klein CJ, Mills JR, Pittock SJ: Autoimmune/Paraneoplastic Encephalitis Antibody Biomarkers: Frequency, Age, and Sex Associations. Mayo Clin Proc 2022, 97(3):547-559.
11. Shan W, Yang H, Wang Q: Neuronal Surface Antibody-Medicated Autoimmune Encephalitis (Limbic Encephalitis) in China: A Multiple-Center, Retrospective Study. Front Immunol 2021, 12:621599.
12. Herken J, Pruss H: Red Flags: Clinical Signs for Identifying Autoimmune Encephalitis in Psychiatric Patients. Front Psychiatry 2017, 8:25.
13. Bost C, Chanson E, Picard G, Meyronet D, Mayeur ME, Ducray F, Rogemond V, Psimaras D, Antoine JC, Delattre JY et al: Malignant tumors in autoimmune encephalitis with anti-NMDA receptor antibodies. J Neurol 2018, 265(10):2190-2200.
14. Gadoth A, Segal Y, Paran Y, Aizenstein O, Alcalay Y: The importance of tissue-based assay in the diagnosis of autoimmune encephalitis. J Neurol 2022, 269(7):3588-3596.
15. Gong S, Han Y, He E, Liu M, Fu X, Deng F: Coexistence of anti-SOX1 and anti-GABAB receptor antibodies with paraneoplastic limbic encephalitis presenting with seizures and memory impairment in small cell lung cancer: A case report. Front Immunol 2022, 13:955170.
16. Ruiz-Garcia R, Martinez-Hernandez E, Garcia-Ormaechea M, Espanol-Rego M, Sabater L, Querol L, Illa I, Dalmau J, Graus F: Caveats and Pitfalls of SOX1 Autoantibody Testing With a Commercial Line Blot Assay in Paraneoplastic Neurological Investigations. Front Immunol 2019, 10:769.
17. Arnaldos-Perez C, Vilaseca A, Naranjo L, Sabater L, Dalmau J, Ruiz-Garcia R, Graus F: Algorithm to improve the diagnosis of paraneoplastic neurological syndromes associated with SOX1 antibodies. Front Immunol 2023, 14:1173484.
18. Gozzard P, Maddison P: Which antibody and which cancer in which paraneoplastic syndromes? Pract Neurol 2010, 10(5):260-270.
19. Devine MF, Kothapalli N, Elkhooly M, Dubey D: Paraneoplastic neurological syndromes: clinical presentations and management. Ther Adv Neurol Disord 2021, 14:1756286420985323.
20. Seery N, Butzkueven H, O'Brien TJ, Monif M: Contemporary advances in antibody-mediated encephalitis: anti-LGI1 and anti-Caspr2 antibody (Ab)-mediated encephalitides. Autoimmun Rev 2022, 21(5):103074.
21. Virupakshaiah A, Dalakas MC, Desai N, Mintzer S, Ratliff J: LGI1 encephalitis with squamous lung-cell carcinoma: Resolution after tumor resection. Neurol Neuroimmunol Neuroinflamm 2021, 8(1).

 doi:10.20944/preprints202507.2641.v1

22. Kvam KA, Stahl JP, Chow FC, Soldatos A, Tattevin P, Sejvar J, Mailles A: Outcome and Sequelae of Autoimmune Encephalitis. J Clin Neurol 2024, 20(1):3-22.
23. Kang Q, Liao H, Yang L, Fang H, Hu W, Wu L: Clinical Characteristics and Short-Term Prognosis of Children With Antibody-Mediated Autoimmune Encephalitis: A Single-Center Cohort Study. Front Pediatr 2022, 10:880693.
24. Abboud H, Clardy SL, Dubey D, Wickel J, Day GS, Geis C, Gelfand JM, Irani SR, Lee ST, Titulaer MJ: The Clinical Trial Landscape in Autoimmune Encephalitis: Challenges and Opportunities. Neurology 2025, 104(8):e213487.
25. Zhong R, Chen Q, Zhang X, Zhang H, Lin W: Risk Factors for Mortality in Anti-NMDAR, Anti-LGI1, and Anti-GABABR Encephalitis. Front Immunol 2022, 13:845365.
26. Linnoila JJ: Paraneoplastic antibodies targeting intracellular antigens. Handb Clin Neurol 2024, 200:335-346.
27. Zhang J, Ji T, Chen Q, Jiang Y, Cheng H, Zheng P, Ma W, Lei T, Zhang Y, Jin Y et al: Pediatric Autoimmune Encephalitis: Case Series From Two Chinese Tertiary Pediatric Neurology Centers. Front Neurol 2019, 10:906.